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 we are discussing hair.

Hair is a topic that occupies the minds of many people.

There are people that are losing their hair

and want to halt or reverse that loss of hair.

And today we will talk about all the ways

that science has taught us we can slow

or even reverse hair loss.

I confess that researching today's topic

was a particular joy for me,

not because I'm obsessed with hair,

mine or the hair of others,

but because hair turns out to be fascinating

from the perspective of cellular biology and stem cells,

which is a topic that I've long been interested in

and that for much of my career,

I focused on in the context of development.

So when your brain and your nervous system develop,

it develops from a small batch of cells

that turns into many, many trillions of cells.

And it does that by cell replication,

something that we call the cell cycle.

And we'll talk a little bit about this

and so-called mitosis today.

I promise not to get into too much detail,

but what makes hair so very interesting

from a biological standpoint is that every hair,

every single individual strand of hair

has its own little stem cell niche.

meaning its own little pocket down there in the follicle

in which specific stem cells give rise to those hairs

for different durations of time,

depending on the hair, where it is on your body, et cetera.

So for instance, the hairs on your head

will undergo ongoing growth for four to six

or even eight years.

So were you to not cut your hair?

It would continue to grow.

One single hair would continue to grow.

I guess we could say all the hairs will continue to grow for up to eight years.

That is very different from, for instance, your eyebrows,

which have a much shorter period of hair growth,

lasting on the order of months.

That's why you don't see people with eyebrows

that extend down to their waist,

but you can't see people with hair on their head

that extends down to their waist if they don't cut it.

Now that discrepancy illustrates for us

just how incredible hair follicles

and the stem cells that reside within hairs are

and their enormous potential to give rise

to these things that we call hairs,

which are simply proteins of varying length.

So today we are going to address

what determines the length of a hair

or rather what determines how long a hair continues to grow

before it ceases growing and eventually falls out.

We're going to talk about what regulates those stem cells,

what allows them to continue to produce hair

or cease producing hair.

And as we do that, you will learn all the biology

in clear, simple terms, regardless of your background,

that will really set the stage for understanding

what we'll also talk about,

which is how to slow hair loss

or halt hair loss entirely or even reverse hair loss.

So we will talk about hormone related hair loss

in both men and women.

We will talk about some of the mechanical

and stress related influences on hair loss.

And we will talk about the chemical and mechanical approaches

to halting and reversing hair loss.

Everything from minoxidil to deutasteride,

to ketoconazole to microneedling to thyroid, estrogen,

IGF-1 pathways, again, all made very clear,

regardless of whether or not you have

a background in biology or not.

I will also dispel some of the common myths

about balding and hair replacement.

If you've heard, for instance,

that you inherit your patterns of balding from your mother's father, that is not true. Although it is true that you do inherit certain genes that influence whether or not you have a predisposition to balding in particular parts of your head, and believe it or not, even in particular parts of your body. But it is not the case that you can simply find a photo of your mother's father, say at age 50 or age 60 or 75, and determine whether or not you'll have the exact same pattern of hair loss. So that's a myth that I'd like to dispel right here and now. And I will dispel some of the other myths about hair loss, hair replacement, and hair regrowth as well. 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 Helix Sleep. Helix Sleep makes mattresses and pillows that are the absolute highest quality. I've talked many times before on this podcast about the fact that sleep is the foundation of mental health, physical health, and performance. Helix understands that everybody's sleep needs are slightly different. So if you go to their website, you can take a brief quiz, and that guiz will ask you guestions such as do you sleep on your back, your side, or your stomach? Maybe you don't know, which is fine. You can simply answer, I don't know. Or do you tend to run hot or cold during the night? Helix takes those answers and then matches you to a mattress that's ideal for your sleep needs. So for me, it matched me to the Dusk at EUSK mattress,

and I've been sleeping on a Dusk mattress

for over two years now, and my sleep has been better than it ever was previously. So if you're interested in upgrading your mattress, go to helixsleep.com slash huberman, take their brief two-minute sleep quiz, and they'll match you to a customized mattress for you. You'll also get up to \$350 off any mattress order and two free pillows. They have a 10-year warranty and you get to try out their mattress for 100 nights risk-free. They'll even pick it up for you if you don't love the mattress, but I think you will, and I certainly love mine. Again, if you're interested, go to helixsleep.com slash huberman for up to \$350 off and two free pillows. Today's episode is also brought to us by HVMN ketone IQ. Ketone IO is a ketone supplement that increases blood ketones. I wanna be very clear that I, like most people, have heard of the ketogenic diet, but I, like most people, do not follow a ketogenic diet. That is, I'm not in ketosis. However, most people don't realize that you can still benefit from increasing your blood ketones, which is what HVMN ketone IO does. I take ketone IQ prior to doing really focused cognitive work. So I take it once in the afternoon, anytime I'm going to prepare for a podcast, or do a podcast, or if I'm going to do some research, or focus on a grant, anything that requires a high level of cognitive demand, and that's because ketones are the brain's preferred use of fuel, even if you're not following a ketogenic diet. I sometimes also use ketone IO prior to workouts. either resistance training workouts, or endurance workouts, such as running.

Again, that's because ketone IQ,

by raising blood ketones, is really a brain fuel.

If you're interested in trying ketone IQ, go to hvmn.com and use the code Huberman to get 20% off.

Again, that's hvmn.com and use the code Huberman to get 20% off.

Today's episode is also brought to us by Roka.

Roka makes eyeglasses and sunglasses

that were designed with the biology

of the visual system in mind.

I've spent a lifetime working on the biology $\,$

of the visual system, and I can tell you

that your visual system has to contend

with an enormous number of challenges

for you to be able to see clearly throughout the day.

So for instance, when you go from a shady area

to a well-lit area, your brain and eyes

have to make a bunch of different adjustments

that eyeglasses of the conventional type

don't always compensate for.

Roka eyeglasses and sunglasses compensate

for those sorts of changes and all the sorts of changes

that your visual system has to contend with.

So you always see things with crystal clarity.

The company was founded by two all-American swimmers

from Stanford, and initially their eyeglasses

and sunglasses were designed for sports performance.

And indeed, all of their eyeglasses and sunglasses

can be used while running, while cycling.

They won't slip off your face if you get sweaty.

They're extremely lightweight,

but they also have an enormous number of varieties

of eyeglasses and sunglasses

that are designed to be worn at work or to dinner.

So they have the sort of classic performance eyeglasses

where it makes you look like a cyborg if you like those.

They also have more conventional aesthetics

that you'd be comfortable wearing pretty much anywhere.

If you'd like to try Roka eyeglasses or sunglasses,

you can go to roka.com, that's R-O-K-A.com,

enter the code Huberman to save 20% off your first order.

Again, that's R-O-K-A.com, enter the code Huberman at checkout.

Okay, let's talk about hair.

And in researching this episode,

by talking to experts in the biology of hair and the stem cells that exist in all of us,

that give rise to our hair growth

and the pigmentation in our hair.

And in talking to experts who understand how to halt

and even reverse hair loss,

that there is a tremendously interesting biology

surrounding hair.

But there's also an incredible psychology around hair.

In fact, most people who experience even marginal hair loss undergo pretty severe anxiety.

Now, I confess this is not something I can relate to.

I am losing my hair in certain places.

I'm 47 years old, I've got a couple of patches upfront

where there's very minimal hair.

I think that as we'll later learn in this episode

reflects a higher density of DHT,

dihydrotestosterone receptors at that particular location

as opposed to elsewhere in my scalp.

But keeping my hair is not something

that I've fretted about much of my life.

And yet, as I was researching this episode,

I remembered an anecdote from my childhood

where my father told me,

and I think it was because I was stressing about something.

And I was trying to get to sleep.

And he said, don't stress, calm down.

And here's why.

If you stress too much,

it can actually make your hair fall out.

In fact, I have a cousin who lay down one night stressed

and woke up the next morning

and all of his hair was on his pillow.

And I'll never forget that story.

I think he was trying to get me to stress less.

I don't know if that story made me stress less or not.

But in any event, I don't know that that story is true.

I'm not gonna challenge the authenticity of that story.

I didn't have a chance to reach out to my father

and ask him to verify or not.

But as we'll soon learn,

it is true that our psychological wellbeing

can impact both the coloration or lack thereof

and the growth rates of our hair.

That's a real thing.

And the reverse is also true,

which is that as hair starts to thin or fall out

or change color,

many people experience intense anxiety or even depression.

This was not something I was really aware of.

And perhaps that's just because

I've always kept my hair pretty short anyway.

I was assumed that if my hair started to really fall out,

it would just shave my head.

But that's me.

And that's not most people out there.

I think most people would loathe to lose their hair.

And in fact, given the enormous number,

probably up in the high billions of dollars

and euros and other currency, of course,

that people invest in trying to halter reverse their hair loss,

it's clear that hair is very, very important to people.

What we know is that by age 50,

approximately 50% of all men and women

will have experienced significant enough hair loss

that they start to notice it.

And a large percentage, up to 85% of those people

will experience some sort of anxiety

that leads them to go out and try and either halt

or reverse that hair loss.

Now, why at age 50?

Well, an important point arises from that,

which is that the hair loss is not occurring

between the 49th and 50th birthday.

Hair loss is ongoing from about age 30 to age 50.

It's only by age 50, however,

that about 50% of people out there

start to notice that hair loss.

And this is typically because they'll be in a bathroom

or looking in a mirror and the lighting will be bright enough

that it permeates the sort of outer boundary of their hair

and they'll notice that their hair is thinning

in a particular location.

That's usually how the sort of thing happens.

So again, our psychological states can impact

our patterns of hair growth or loss.

And of course, patterns of hair growth, but more typically hair loss and hair graying can really impact psychological states. So this is a subject that people take intense interest in. And today we're going to talk about how hair normally grows, why it grows at the rate and for the duration that it happens to. And then as we talk about ways to intervene with that hair loss, those biological mechanisms will come up because they really provide a nice framework for explaining why certain treatments work more or less well or why certain treatments might have certain side effects or total lack of side effects. It will also highlight a really key theme that will come up several times in today's podcast, which is that there are both mechanical and chemical approaches to slowing and reversing hair loss. Mechanical approaches would be things as simple as massaging the scalp, but mechanical changes to the scalp can cause either hair loss or facilitate hair growth. This is why things like micro-needling are so prominent in the context of trying to reverse hair loss. But again, there are also chemical approaches to trying to halt or reverse hair loss. And this relates to the fact that the hair growth itself is strongly regulated by hormones such as estrogen, thyroid hormone, insulin-like growth factor, and that other hormones, in particular the androgens, so things like testosterone, but mainly it's derivatives like dihydrotestosterone are very much involved in setting the stage for hair growth by controlling how big or small that pool of stem cells that gives rise to hair growth is. So if any of the terms I just use are confusing to you, don't worry, I will make all of those very clear in a moment, it's actually all pretty straightforward and simple. And so I'd like to just start by talking about what hair is, how it grows, why it stops growing, and why hair normally falls out. Let's talk about the biology of hair.

And in doing so, I also want to talk about stem cells. Keep in mind that when you hear the word stem cells, you probably like most people think about the sorts of cells that people are getting injected into their face to get rid of wrinkles or give them new skin or to give them more hair if it's injected in the scalp or into a joint to repair a joint or a muscle. Those stem cells are what we call exogenous stem cells, exogenous, meaning from outside the body. The stem cells that we're gonna talk about today are so-called endogenous stem cells, cells that we all make that can give rise to other cells. And that's really the definition of a stem cell. Stem cells are present in all of us from the very beginning of life. So when sperm meets egg, that cell, which we think of as the egg, starts to duplicate, it incorporates the DNA from the sperm and the egg, of course, starts to duplicate, and then those cells give rise to more cells and more cells. And the ability of all those cells to replicate and create more cells are because those cells really are stem cells. Now, at some point, we are a completed body plan, as the biologists say. We end up with a brain and a spinal cord and limbs and fingers and livers and guts and all the things that we need in order to be a functioning human being, even though we're a baby at that time, we haven't grown up. We have all the bits that we're going to have for our entire life. At that point, many of the stem cell populations disappear. For instance, past puberty and probably earlier, you don't get many more new brain cells. You get a few, but you don't get many more new brain cells because the brain doesn't have many stem cell populations, whereas other organs in your body maintain little pockets of stem cells, or in some cases, many, many stem cells that can give rise to more and more of that tissue across the lifespan.

And hair is one such tissue. So if we take a look at hair, what we find is that indeed, there are these things that we call hairs, but there are also stem cells, and those stem cells are actually what give rise to the hairs that we see on the head of our scalp, or that we see on the surface of our body. So right off the bat, you should know that every single hair that you have is there because you have a stem cell population that is giving rise to that particular hair. So let's take a step back, or rather, I should say, let's zoom in on one hair and the stem cell population that gives rise to that hair, because in doing so, you're going to learn all the different components that you can tap into if your goal is to halt the loss of hairs or to replace hairs that have already been lost. So if we were to just zoom in at the level of one hair, what you would find is that that hair has what typically is called a hair root, so that's the portion below the skin, and when we say below the skin, it means that it dives down into a narrow trench, which is in the so-called epidermis, which is this outer layer of skin. It also has a shaft. The shaft is the part that grows out above the skin. So what you see on somebody's head or you see on their arm, or when you see an eyebrow, you're seeing the shaft of the hair. The root, of course, goes below the skin. What most people don't realize, however, is that down at the base of the root, there's actually a little cave, a little pocket. So if you were to look at this, it would look like a little bulb, a little round area with a bunch of stuff in it right below the root. And within that little cave, there are stem cells. There are populations of cells that have the ability to divide, right? We call this mitosis.

It's a process by which cells can actually divide

and take DNA with them and then give rise to other cells.

We call those cells that divide and move out.

We call those daughter cells.

We call the cells that give rise to them progenitor cells,

but they are effectively stem cells

that give rise to these, what we call daughter cells.

Those daughter cells then become the various types of cells

that make up the hair.

So when you see a hair,

you're not seeing something that grows

throughout the lifespan.

You're seeing something that's going to be born

down there in that little cave, then is going to grow.

It's actually going to stack up on top of itself.

And that's because hairs are made up of a protein

called keratin.

There are a bunch of different kinds of keratins,

depending on what kind of hair you're looking at.

But these are little proteins

that stack up on top of one another.

And they're structured in a way

that makes them pretty darn durable.

I mean, it's possible, of course, to pull a hair out.

But if you've ever tried to tear a hair,

in particular, thick hair, like one from the face,

or even one off the top of your head,

it's actually a pretty tensile, strong little thing.

And that's because keratins stack up on top of one another

and bind to one another with a really strong bond.

So what you end up with is a bunch of proteins

stacked up on top of one another,

and that's the actual hair, okay?

So we've got the hair shaft, the hair root,

and then we've got the stem cells down there

in that pocket that give rise to the various cells

that make up the actual hair.

We also have down there in that little cave,

which, by the way, is actually called the hair bulb,

if you really want to know the technical name,

because it's shaped like a bulb.

We have not just stem cells,

but we have cells that give rise to the pigment of the hair

that create what's called melanin. Now, some people have very blonde hair, very light hair. Some people have darker hair. But everybody, unless they have what's called the albino mutation, where the hairs are truly white, they lack all melanin, and it's a pretty rare condition, although it does happen. Most people have some degree of melanin in their hairs because there are little pockets of melanin-producing cells, melanin's just a protein, that essentially gets injected into the keratin, into the hair, and it gives it its darker color. Now, there are a couple other components about the hair that you need to know about, especially if you're interested in reversing hair loss or reversing graying of hair. One of those components is a little gland. So next to every hair root within the dermal layer of the skin, so this is below the epidermis, there is a gland called the sebaceous gland. And the sebaceous gland makes oily stuff, and the oily stuff is called sebum. I don't know the name sort of evokes something kind of gross, but sebum is actually really cool and really important. The sebum gets injected, or seeps rather, into the area right around the hair as the hair starts to approach the surface, where it goes from essentially root to shaft. And the sebum does two things. First of all, it forms a little bit of a seal right at the place where the hair exits the skin, and that seal is very important actually for waterproofing of your skin. So we don't often think of ourselves as waterproof because we are so accustomed to water just landing on our skin and rolling off, but that's because of some of the oily properties of our skin. Now, it's also true that our skin is pretty densely packed with cells,

but in the absence of sebum,

we would not be as waterproof as we are.

Now, as I mentioned, sebum has two important properties.

The other important property of sebum

is that it actually is a strong antibacterial

and antimicrobial.

Most people don't realize this.

The oils of your skin provide a lot of immune boundary

so that things don't get into the hair root

or the region around it and infect our skin.

So sebum, while the name is sort of unattractive,

to be honest, is actually performing some essential roles

both for waterproofing and for our immune system function,

protecting us from various kinds of infection.

We're gonna return to sebum later

because it turns out sebum is also very important

as it relates to psoriasis

and as it relates to some of the fungal components

that can cause hair loss, okay?

So just gonna file that away.

There's another important component

of the region around hairs,

which is the erector pili muscle.

The erector pili muscle is a muscle that lies diagonally

between that bulb portion of the hair

or a little bit above it

and goes up to the surface of the skin.

The erector pili muscle is a muscle that contracts

when we get cold or when we get scared.

So if you've ever had goosebumps,

that's because the erector pili muscle

contracts pulling the skin at the surface down

around the little hair follicles

or at least where the hairs meet the surface of the skin.

And so those little bumps are actually

where little micro hairs reside

and the dimples between them are the dimples

that occur when this erector pili muscle pulls down.

Now, why would this muscle exist?

Well, it has a couple of important functions.

One of the functions is that when it pulls down,

it causes, as the name suggests,

the hairs to stand up, maybe not perfectly vertical,

but when you hear, oh, I was so frightened,

my hair stood up on end

and that's because the hairs become erect, they stand up.

Now, why would this happen when we get cold?

It happens because when the hairs stand up,

air can be trapped between those hairs

and can actually warm our body.

This is not so much the case

if you have very light hair on your skin.

If you're a very hairy person,

this is going to be a more robust aspect of your physiology.

And yes, this is why dogs like Huskies

can go out in the snow and still remain warm.

When they get cold,

their hairs actually stand up a bit on end

because of the contraction of these erector pili muscles,

trapping air in there

and then their body warms the hair, trap beneath the hair.

And it's sort of like they've got a blanket on

made by the interface between the hair,

the air and their skin.

So just to recap, all the components of hair

and the different things around it

that are going to be relevant for understanding

how to replace hair that's lost,

we have the hair itself,

which has the shaft that sticks out over the skin.

It goes a little bit into the skin,

but basically sticks out over the skin.

We have the root portion, which goes down into the skin.

It goes through the epidermis and into the dermis.

Then we have this bulb-like region down at the bottom.

Down at the bottom of that bulb,

we have stem cells that actually give rise

to the actual hair

and we have pigmenting cells that pigment that hair.

In addition, and this is very important,

there are capillaries that go into that bulb region

down at the bottom of the hair

and that can serve and support the stem cells,

the melanin-producing cells,

which are called melanocytes.

So the melanin-producing cells and the stem cells

get a lot of blood flow that allows them

to keep providing new hair or the proteins that make up hair

and the pigment that goes into those hairs. And those little capillaries deliver not just nutrients and things of that sort, but they also deliver oxygen. Because it turns out that the whole process of growing more hair is a very active process. Now, as soon as you hear oxygen and you hear that the growth is an active process, that should cue you to why so many of the stories around how to keep your hair and regrow hair involve statements like, don't wear a hat. It'll make your hair fall out. Or if you want your hair to grow back, don't wear hats or massage your scalp or increase blood flow or why some people will suggest that people take peppermint oil, for instance, or menthol type oils of different kinds and massage them into the scalp. Things that make the scalp tingle or there will be light therapies designed to what? To increase blood flow to the scalp. The whole rationale there is that you're trying to increase blood flow to the stem cell and the melanocyte populations that support the hairs and that actually create the hairs. Now, whether or not those approaches work, we'll touch on a little bit later. I'll just give you a little bit of a hint right now, which is that while no single one of those approaches that I described is known to regrow hair in a very robust way, because of the requirement for oxygen and nutrients and because it's such an active process for the stem cells and melanocytes to grow and darken the hairs that grow out of your skin, it is true that manipulations or treatments that increase blood flow to those regions can at least slow the loss of hair or can even extend the duration over which hairs continue to grow. So if you've heard things like don't wear a hat if you want to maintain your hair or massage your scalp if you want your hair to grow faster,

in some sense that's true, but none of those manipulations on their own is going to robustly enhance the rate of your hair growth. Those things are designed to be done in conjunction with some other treatments that have been shown in many, many clinical studies to increase the rate and duration of hair growth. So now you have in mind a picture of what's happening at the level of individual hairs. And if you're anything like me, you're probably thinking, wow, there's a lot going on down there just below the surface of the skin. And indeed there is, but really the things to think about are that stem cell population that actually give rise to the hair proteins so that actually create the hair, the melanocytes that darken that hair, they give it pigment, that sebaceous gland and the oil sebum that provides some important antimicrobial and other properties to that general region, and that erector pili muscle. That erector pili muscle, as I mentioned earlier, is important for creating goosebumps. And it's important for keeping Huskies warm in cold environments, but it's doing some other really important things as well. And we'll talk about those as time goes on in this episode. Right now what I want to do is just talk for a moment about how hairs actually grow and why they grow the way they do. This is extremely important toward understanding hair replacement and slowing hair loss. There are three basic phases of growth of a hair. The first phase is the phase in which the stem cells down in that bulb give rise to the cells that make up the proteins of the hair. So the actual growth of the hair. And keep in mind that the hair is actually growing from the bottom up. Now you might think, of course it's growing from the bottom up.

Everyone knows that.

But a lot of people think that the hair starts growing

right at the surface of the skin.

That's not the way it works.

The hair is actually growing from deep within the root

and stacking up and then eventually

extends out across the top of the skin.

That growth phase is called the anagen phase, A-N-A-G-E-N.

And this for some people will ring a bell

because if you've ever been interested in weightlifting

or even if you're an endurance runner,

you'll hear about things that are anabolic,

that promote growth, so Anna of growth

or catabolic that promote breakdown.

So the first phase of hair growth is called the anagen phase.

And it's a period of varying duration

depending on which hair in the body we're talking about.

So the anagen or the growth phase for hairs on the head,

as I mentioned earlier, is anywhere from two to eight years.

For most people it's going to be about six years.

What this means is that if we were to just not cut our hair,

just let our hair grow for two to eight years,

that hair would eventually grow to a length

that it was at its maximum and then would stop growing.

So we can say that the anagen phase of hairs on the scalp

is two to eight years, right?

The duration of the growth phase.

Contrast that, for instance,

with the duration of the anagen phase

for hairs of the evebrows.

Hairs of the eyebrows grow about 4.2 millimeters per month.

Believe it or not, people have measured this sort of thing.

Now that's an average.

So some people are going to have eyebrows

that grow much longer per month.

I'm somebody who, for instance,

has mostly the same length eyebrow hairs,

but every once in a while I get one of those eyebrow hairs

that really seems to be heading off my head.

It really wants out of there.

And so it's much longer than the rest.

What does that mean?

Does it mean that it grew faster, maybe?

But chances are the stem cell population in that particular eyebrow follicle, for that one eyebrow hair, is longer than it is for the others.

This is really important.

I'm trying to illustrate two principles at once here.

The first principle is that different hairs on your body,

including the hairs on your scalp,

have a growth phase of different duration.

This is why the hairs on your head can grow very, very long because they have a very long antigen or growth phase.

And the hairs on your eyebrows will only grow for a few months before they actually fall out

and then have to undergo replication of the stem cells

to give you new eyebrow hair to then grow.

What's important here is not just that there are differences in the duration of the growth phase,

but rather that the rate of hair growth is not something that tends to differ within a given body region, right?

You'll hear people say,

oh, my hair grows really, really fast.

Other people will say,

oh, my hair grows really, really slowly.

That is probably not the case.

Well, there could be slight differences in the rate of growth.

That is the addition of more keratin to the actual hairs.

So creation of more hair more quickly.

Almost certainly what's happening is that the duration of the antigen phase in some people is just much longer than it is in other people.

And we don't realize this and we tend to think more in terms of how fast hair grows,

because if you were to just look at somebody's hair, you'd say, oh, you know,

they're all more or less the same length.

I mean, some people's bangs are shorter than the back because they cut them.

But if they were to just grow their hair,

you'd say, oh, it's all more or less the same length.

But if we were to zoom in with a microscope,

we would see that there are a lot of hairs down there in between the other hairs that are very, very short

or even tiny, tiny, tiny.

And those are coming in as the other ones are finishing their antigen phase, okay? So that's the antigen or growth phase. After the antigen phase comes the catagen phase. Again, this resembles the word catabolic or the breakdown phase.

During the catagen phase,

the hair is actually receding not from the top down to the skin surface and then into the root,

but the other way.

It's actually receding from that bulb region up toward the surface.

And that catagen phase is going to also be of different duration depending on which area the body you're in and it will vary a little bit depending on who you are, meaning from person to person. We'll talk about the influences on the antigen

and catagen phase in a moment.

Why is it important that it actually recedes

from the inside out?

Well, that's important because as it does that, there's actually a change in that bulb region down below because normally there's an interface, there's a conversation that's occurring between the stem cell population, the melanocytes and the hair itself

and they support each other.

And remember, there's blood vessels going into that area or rather capillaries that are feeding that area as well. After the catagen phase comes the telogen phase,

which means rest.

The telogen phase is a period in which no new hair proteins are being added by those stem cells.

And during that telogen phase, that bulb down there at the bottom, instead of being nice and oval and really having a lot of space in it with all these different things like stem cells, starts to pinch off from the little tube that comes down from the surface of the skin that normally would have a hair in it. It starts to pinch off and at some point, many hair follicles pinch off that bulb region

and it recedes and dies. And when it recedes and dies, the stem cell population and the melanocytes go with it. In other words, there is no longer a population of stem cells to give rise to more hair after that telogen phase. And there's no longer melanocytes to pigment the hair. And in fact, the hair isn't there anymore. So there's no hair even to pigment. After that telogen phase, unless it's a hair of a particular type, such as the hair on your scalp, which can then reenter the cell cycle and get back into an antigen phase and regrow more hair from stem cells. So there are three critical stages of the life cycle of a hair that are relevant to today's conversation. There's the antigen phase during which the hair grows. There's the catagen phase during which the hair actually starts to recede and die. The protein is actually disappearing from the bottom up. And then we have the telogen phase, which is the phase in which the stem cell population is what's called semi-quiescent, semi-quiet, or completely quiescent, where it's not active at all. Those three phases make up the life cycle of a hair, keeping in mind that for some hairs, they can reenter the life cycle and go back into the antigen phase. If there's stem cells there, and if there is oxygen there, and if there is sufficient blood support, and, and this is a very important, and if there are the appropriate hormonal signals to support growth, and there is a reduction or an absence in the hormone signals that actually trigger that telogen phase. I make this point now because much of the rest of today's discussion is going to focus on why particular hormones, such as dihydrotestosterone cause hair loss, and why inhibiting things

like dihydrotestosterone can support

the preservation of hair that you have and the regrowth of hair. To make a long story very short, and then we'll get into some additional details that are relevant and that I hope you'll stick around to listen to, dihydrotestosterone, which is a derivative of testosterone, causes changes in that bulb region where the stem cells reside. It shortens or halts the antigen phase of hair growth, and it extends and promotes the catagen and telogen phase. So when we hear that, oh, you know, dihydrotestosterone makes your hair fall out or estrogen makes your hair grow, there are real chemical, or we should say biochemical, legitimate reasons as to why that is, but it all comes back to this three phases of hair growth, the antigen growth phase, the catagen catabolic or hair loss phase, and the telogen phase, which is a rest period in which the hairs can either come back if it reenters the antigen phase or maybe it's over for good. Hormones are the accelerator and the break on each one of those phases. I'd like to take a guick 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 in Athletic Greens are optimal and vital for microbiotic health. In addition, Athletic Greens contains a number

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So now you have in mind the anatomy of the hair

and the area from which it grows and the stem cells, et cetera.

The fact that there's capillary innovation

delivering oxygen and blood flow to the stem cells

that give rise to the hair,

and that there are these three critical phases

of hair growth, antigen, catagen, and telogen.

Now let's talk about the accelerators on hair growth

and the breaks on hair growth.

There are many accelerators on hair growth,

but the first one that I really want to underscore

is blood flow itself, which equates to the delivery

of nutrients and oxygen.

This is very important and it explains a lot of the treatments

for halting and reversing hair loss.

For instance, one of the longest standing treatments

for halting and reversing hair loss is so-called minoxidil.

Minoxidil, sometimes also referred to

by the brand name Rogaine, was actually a drug

that was developed to treat hypertension.

So this is a cardiac drug that lowers blood pressure

and it does that by causing vasodilation.

It allows more blood flow, not just to the hairs

on your scalp, but to hairs everywhere on your body.

And indeed, most people don't realize this,

but minoxidil won't just slow the loss of hair

from your scalp, it is also effective

at slowing the loss of hair elsewhere in your body.

How does it do that?

Well, you now know one of the major ways it does that.

It does that by extending the antigen phase. So it basically makes that phase a bit longer. It doesn't make it much, much longer, which is why for most people who are losing their hair quickly or who have already lost their hair, minoxidil alone is not going to be a sufficient treatment. However, minoxidil has been shown to be effective at slowing rates of hair loss and people that are starting to experience some hair loss all get into dosages and things of that sort a little bit later. But right now I just want to really focus on the logic of why people would take this drug, which is lowering hypertension at all as it relates to hair loss, right? That might seem like kind of crazy until you understand the anatomy and the growth of hairs, which you now do. So that's what minoxidil is doing. It's creating more blood flow to the hairs, which because minoxidil does have this positive effect, at least most people would like to slow their rates of hair loss on their scalp anyway, it tells you that blood flow and delivery of oxygen and other nutrients from the blood is pretty critical, if not very critical for the support of the hair growth cycle itself. Now, again, we haven't talked at all about the sorts of chemicals or the signals within the body, such as hormones that actually direct the growth of hairs. Here we're just talking about a mechanical change, allow more blood flow to the region and thereby extend the antigen phase, which is exactly what happens with minoxidil. Now, minoxidil does have other effects and this is why dosing of minoxidil becomes a little bit complicated and can be a little bit tricky to troubleshoot. It can greatly lower blood pressure or lower blood pressure just a little bit, depending on how sensitive somebody is to that particular drug.

So oftentimes physicians will start people on minoxidil dosages that are very low, ideally that would be the case, and then ratchet it up in order to figure out where the minimal effective dose or the kind of critical threshold is, beyond which they start experiencing some pretty uncomfortable side effects, such as swelling of the ankles or headaches or dizziness. These things can happen with the use of Rogaine, aka minoxidil. Now, minoxidil has also been associated with increases in the hormone prolactin. Prolactin is a hormone that's released from the pituitary. It is a hormone that acts also as a bit of a neurotransmitter like many hormones, and it tends to be antagonistic or in opposite to dopamine. So dopamine is a neurochemical, it's actually a neuromodulator, meaning it modulates the activity of a bunch of neural circuits in the brain. It also controls the release of various hormones in the body. Dopamine is almost always associated with states of motivation, pursuit, and drive. It has a little bit of a kind of feel good element to it, which is why a lot of people think dopamine is associated with reward and pleasure, but it's really about energy, motivation, and drive. Dopamine and prolactin are, as I mentioned before, antagonistic to one another. They're in sort of a push-pull. So people who take monoxidil, especially if they're very sensitive to it, or they take dosages that are too high, will experience increases in prolactin that in turn can cause things like reductions in libido, reductions in overall feelings of well-being, apathy, and in some cases where the elevations in prolactin are more extreme, they can experience, for instance, increase in male breast tissue, gynecomastia, or even small bits of milk let down, things of that sort.

In women who take monoxidil,

the side effects are much like the ones experienced in men, so there can be swelling, edema of the tissues, because if you get too much vasodilation and too much lowering of blood pressure, that's not good, headaches, dizziness, and so on. So dosing of monoxidil is really important. If somebody's going to use monoxidil in order to try and slow or reverse hair loss, and again, it mainly is going to be used to slow rates of hair loss. not to actually reverse hair loss. The really key thing is to get that dosage right. So the ranges of monoxidil that you'll see suggested and that people use out there are vast. And I should also mention that there are two major routes by which people get monoxidil to the hair follicle. One is to take it systemically as a pill, where it goes into the general circulation. The other is to take it topically as a cream. There are prescription and non-prescription forms of monoxidil, just to further complicate things. But the ranges of oral monoxidil that you'll see out there and that people take, range anywhere from 0.25 milligrams, all the way up to five milligrams per day. So that's an enormous range, it's like a 20-fold range. The topical monoxidil is also found in various concentrations. The typical concentration is going to be a 5% concentration that people will use once per day. Topical treatment with monoxidil at 5% concentration is thought to just stay at the scalp, but we now know that it can go systemically, it can get into the general bloodstream. Why that issue make complete sense to you? Because when you put something on your scalp, I've already told you these little pits, these little tubes that go down to those bulb regions below the skin that have direct access to the blood supply. So when you massage something into your scalp, it not only has the opportunity to get into your general circulation, it often does, especially if it's something that's very water soluble and that way can get into the capillaries

and into the general bloodstream.

Although topical treatments,

of which we're going to discuss a number of them today,

don't tend to get into the general circulation

as robustly as taking something by way of pill or capsule.

Okay, so monoxidil works by way of increasing blood flow

to the stem cell niche below the hair.

The dosage ranges of the oral monoxidil are tremendous,

0.25 milligrams all the way up to five milligrams

once per day, the dosage range of the topical solutions

tends to be a little bit more confined.

Typically it's a 5% solution

and it's recommended that people use it one time daily,

maybe twice daily.

It's also important by the way,

if you're going to take this route,

that you actually leave that solution on the scalp

for three to five minutes.

This is important and should make complete sense

as to why it's important.

You can't just rub the stuff into your head

and then rinse it off and expect it to be absorbed.

It actually needs to seep down into those hair follicles

and access the niche.

How do people arrive at the correct dosage for monoxidil?

Well, for better or for worse really,

in some cases it's accomplished by finding out

that you have an unwanted side effect like dizziness

or swelling of your ankles or edema

or I would hope this wouldn't be the case,

but something that suggests there's hyperprolactinemia.

You could get a blood test to measure prolactin

or you perhaps notice a drop in libido

or some lethargy, these sorts of things

that are common to reduce levels of dopamine,

increase levels of prolactin.

I would hope that if people are working with a physician

or if they're not in taking monoxidil,

that they would start with the lowest possible dose.

So for oral monoxidil that would be $0.25\ \text{milligrams}$

and then increase it as needed rather than jumping in

right at five milligrams.

Because some of those side effects,

in particular the fluid retention and the hyperprolactinemia can be pretty uncomfortable and can disrupt a lot of aspects of life that most all of us consider desirable. Okay, so we're really focusing right now on treatments that relate to the critical requirement for hair stem cells to receive blood flow in order to receive oxygen and nutrients to get the hair to grow. And that's really what monoxidil is about. It's also what all of those anecdotes you hear are all about like massaging the scalp or putting red light on the scalp. Although red light might do some other things. In general, heating or lighting of the scalp or massaging of the scalp is really designed to increase blood flow to the scalp. Now the reason monoxidil works at all is because it is going to increase blood flow around the clock. And that's because people are taking it topically and it's seeping into the general circulation or at least as stings somewhat restricted to the hair cell niche or they're taking it orally and it makes it to that hair cell niche below the follicle. When we massage our scalp, however, that's a transient thing. You know, I can massage my scalp right now. I'm no doubt increasing blood flow to certain areas. I'm probably decreasing blood flow to the areas I'm pushing down on, but it's all temporary. I don't know many people that can massage their scalp enough during the day or long enough during the day rather that it would sufficiently increase blood flow. With that said, it is clear that increasing blood flow to the scalp by way of reducing hypertension which is effectively accomplished by broadening by expanding the blood vessels and capillaries is an effective way to at least hold on to the hair that you have. Is it going to completely halt hair loss? If you have a strong genetic bias towards hair loss, no.

Is it going to reverse hair loss?

Very unlikely that it will, but it can slow hair loss or even maintain the hair that you have. So if we were to take a step back and ask ourselves what other sorts of drug treatments are out there besides minoxidil that increase blood flow and that might increase the rates of hair growth or more likely increase maintenance of hair that one already has by increasing blood flow to the niche. And nowadays, there are more and more doctors who are familiar with this requirement for blood flow, understand the mechanisms by which minoxidil works and understand the vast desire out there for people to hold onto the hair they have and regrow hair and they're prescribing things like low dose to dalafil. So 2.5 milligram to five milligram to dalafil. To dalafil was initially discovered as a drug to treat prostate health. It was a drug that we now know can increase blood flow to the prostate and thereby offset some of the issues associated with an aging prostate. Higher doses of to dalafil, sometimes also referred to by its brand name, which is Cialis, are used to treat erectile dysfunction, but at the dosages that are used to increase blood flow to the prostate and that now a number of doctors are using to increase blood flow, not just to the prostate, but to all regions of the body, including the scalp, such as 2.5 to five milligrams to dalafil. So this is something that I think deserves attention because it falls under the umbrella of increasing blood flow to the hair stem cell niche in order to maintain hair. It is not something that most doctors are going to be familiar with as the way to reverse hair loss because it won't do that. But the use of low dose to dalafil to slow rates of hair loss is very much in a logical mechanistic sense, exactly the same as the logic of using minoxidil to slow rates of hair loss. It's all about increasing blood flow

to support the stem cell niche below the hair follicle. The critical requirement for blood flow, oxygen, and nutrients to the stem cell niche is also why you hear a lot nowadays about the use of PRP, plate-rich plasma for trying to offset hair loss or even reverse hair loss. We're going to do an entire episode about PRP. It is pretty controversial in certain circles and well-accepted in other circles. A couple of key things to understand about PRP. First of all, it is being used in multiple tissues for different purposes in different clinics. So for instance, board-certified physicians in the United States, Canada, and Europe are doing PRP injections into ovaries to try and expand the number of healthy follicles and eggs so that people can conceive later in life or even earlier in life if they don't have many follicles. People are getting PRP injections into their joints in order to try and support joint health. People are getting PRP injections into just about every tissue you can think of. However, PRP, despite what you may have heard, is not stem cells. Somebody tells you they're injecting stem cells that are either outside the US, Canada, or Northern Europe or they're injecting something else. So you want to really look into that. The safety issues there are the subject totally deserving of an entire episode. I'm not necessarily opposed to the future of stem cells as a treatment, but keep in mind that stem cells are cells that can give rise to lots of other cell types and they are cells that divide and replicate. And there's a name for that when it happens in the adult body when you don't want that and that's called cancer. Tumors are overproduction of cells from stem cells when those stem cells ordinarily should be quiescent. So keep in mind the difference between stem cells and PRP. PRP, platelet-rich plasma, again, is legal in the US

and many other places because it involves drawing somebody's blood, spinning it down at a particular speed which separates out different components within the blood, then taking the platelets and re-injecting those in a solution back into the person's body. So platelet-rich plasma, or PRP,

is platelet-enriched plasma from that person.

But the basis of PRP is really to encourage

nutrient delivery to a particular region in the body

using somebody's own platelets

because those platelets are enriched for various nutrients.

So people are getting PRP injections into their scalp.

Those are not stem cell injections.

Those are PRP injections into the scalp.

With some moderate success,

these are very expensive treatments.

They tend to be transiently successful.

I'm sure there are people out there

who are going to say PRP worked fabulously well for me.

That might be the case.

I'm not going to dispute that and I'm happy for you.

Although there are not sufficient clinical data

to suggest PRP as a treatment right now,

especially given the cost,

many thousands of dollars, many, many treatments.

It's also the case that the PRP injections,

when they work, might work for reasons independent of the platelets.

What do I mean by that?

Well, soon we're going to talk about

a different type of treatment,

which is a mechanical manipulation of the hair follicle,

typically on the scalp.

because that's typically where people want to regrow hair.

I don't know many people who are trying to maintain

or accelerate or regrow their back hair, for instance.

They might be out there,

but I don't think there are a lot of them

or their leg hair for that matter.

Almost always it's going to be scalp hair.

And one way that people are doing that

is through mechanical stimulation of the hair follicle

and the stem cell niche,

using what's called microneedling. Microneedling, as the name suggests, is taking a bunch of little needles, either in a little stamp, so a little square or nowadays typically it's a roller. So it looks like a paint roller, except it's got tons of little needles in rows all over that roller. Those needles range in length from half a millimeter to 2.5 millimeters, okay, millimeters. And one rolls that over the scalp, and if you're thinking, ouch, that probably hurts, indeed, it can hurt a little bit or a lot, depending on the thickness and the length of those needles. Microneedling has been shown to do two things. It has been shown to reactivate semi-quiescent populations of stem cells that are in that telogen phase, putting them back in antigen phase, and thereby stimulate more hair growth. It is also, and this is, I think, the best use of microneedling, it has also been shown to be a very effective augment for some of the hormone-based hair regrowth tools and pharmacology that we're going to talk about in a few minutes. So microneedling and PRP have something very critical and common, which is the needle, the actual injection into the skin. And for those of you that are hearing this and thinking, why would disrupting the skin with needles actually support hair growth or regrowth? Wouldn't that just damage the follicle? Well, this gets into some of the, I think, interesting, if not fascinating aspects of our biology, which is that all of the cells in our body really can respond to both chemical and mechanical cues. And when we hear needle injected into the skin, we think, ah, that must just be damaging everything, causing all sorts of inflammation. But it turns out that low levels of inflammation caused by things like microneedling or PRP injections, or even the introduction of any kind of fluid, right? For instance, saline fluid injected into a region

can cause changes in the cells in that region, causing, for instance, stem cell populations that were waning to reactivate again, causing tilagin phase follicles that have melanocytes and stem cells that are dying off, but not completely gone to reenter the cell cycle. So microneedling procedures, PRP injections, things like minoxidil, they all kind of center around this same general theme of increasing blood flow, increasing oxygen, delivery of nutrients, or in the case of microneedling, increasing inflammation just enough at that local site that certain cascades of biological function that relate to proliferation of stem cells or maintenance of stem cell populations are kicked off. It's sort of like reminding the cells in that area that they need to stay alive in order to replenish whatever is lost. So sometimes a wound can actually induce some healing, although I do want to point out that the micro part of microneedling is absolutely key. And this should be obvious to you when you think about scars. I don't know about you, but I've never seen a scar with hair growing out of it. Or if there was, it was probably like one hair. But if you've ever seen a scar, if someone had their appendix out, or if someone had a brain surgery, you see that scar because there is no hair growing out of it. So the micro portion of microneedling is extremely important. We are not talking about causing significant damage to a tissue in order to activate that stem cell population. We're talking about causing micro damage and micro levels of inflammation to stimulate growth. For those of you that are interested in using microneedling or microneedling in combination with chemical treatments like monoxidil or some of the other treatments we'll talk about in a little bit like finasteride and caffeine.

caffeine is being used to regrow hair.

Yes, believe it or not.

Very interesting, get into that in a moment. But if you're interested in using microneedling alone or in combination with some of these other treatments, there's a wonderful review that was just published this last year. Wonderful because it's very comprehensive. Not so wonderful, not to the fault of the authors because most of the studies out there on microneedling are not superb. There are ways of gauging the strength of a study, mainly related to their duration, whether or not there were control groups, et cetera. But the review itself is excellent. And the title of the review is microneedling and its use in hair loss disorders, a systematic review. We will provide a link to this in the show note captions. And this review did a very good job of highlighting both the strengths and drawbacks of the various studies looking at microneedling. It also explored the use of microneedling in both men and women and of various ages. And it does appear to be the case that microneedling shows some positive benefit in both men and women regardless of age, especially when used in combination with the various other treatments that we're talking about. I was also able to glean from this review and some of the papers described within it that needle lengths of about one millimeter to 2.5 millimeters seem to be more effective than shorter needle lengths. So if you're scared of the needles and the needle lengths, keep in mind that done properly, microneedling shouldn't be too painful. Some people experience a little bit more pain than others, but it's not considered a very painful procedure. It is, however, a procedure that can cause some bleeding of the scalp and that bleeding of the scalp can be very apparent,

or if you're already quite bald in a given region. So keep that in mind.

I suppose one could wear a hat or a wig

especially if it's in the front of the head,

as opposed to in the top of the head and hidden by some hair,

or something of that sort

if they were really self-conscious about it.

But the microneedling itself

is causing a physical disruption to the scalp,

some degree of bleeding, inflammation.

And again, all of that is part of the process

by which microneedling can actually improve hair growth.

And of course, there's healing that occurs

of the bleeding and the damage to the follicle.

This is a transient thing,

but understanding the cosmetic implications

in the short term as well as in the long term

is certainly worth knowing.

One thing that's very clear

is that the combination of microneedling

and minoxidil treatment together

is far more effective than either of those treatments alone.

In addition, the combination of microneedling and minoxidil

has been shown to be effective

in recovering what are called dead zones.

So these are regions of the scalp

that are either completely bald or mostly bald

for which there is essentially no stem cell population there.

And the combination of minoxidil plus microneedling

is somehow able to recover those stem cell populations

and allow new hair to grow.

Although the growth of that hair

in those dead zone regions can take a very long time,

30 to even 50 weeks.

Neither minoxidil treatment alone nor microneedling alone

has been shown to be effective

in recovering these so-called dead zones

when those treatments are done separately.

So this I would say is a strong reason

to consider combining microneedling and minoxidil

as opposed to just doing minoxidil or just microneedling.

I should also mention that minoxidil treatment

if you pursue it is likely something

that you are going to have to do for the rest of your life

if you want to hold on to the hair growth

that you obtain with minoxidil

or if you want to maintain the hair

that you are already maintaining with minoxidil.

Some people have been successful in taking minoxidil, maintaining some hair growth or even stimulating some hair growth and then coming off minoxidil. But most everyone who goes on minoxidil has to stay on minoxidil because when they cease taking minoxidil, even if they're doing other treatments, they lose the hair that they gained with minoxidil. So that is an important consideration. The decision to go on minoxidil is likely a decision to be on minoxidil for the rest of your life. I'd like to just take a brief break and thank one of our sponsors, which is Element. Element is an electrolyte drink that has everything you need and nothing you don't. That means plenty of salt, sodium, magnesium and potassium, the so-called electrolytes and no sugar. Now, salt, magnesium and potassium are critical to the function of all the cells in your body, in particular to the function of your nerve cells, also called neurons. And we now know that even slight reductions in electrolyte concentrations or dehydration of the body can lead to deficits in cognitive and physical performance. Element contains a science-backed electrolyte ratio of 1,000 milligrams, that's one gram of sodium, 200 milligrams of potassium and 60 milligrams of magnesium. I typically drink Element first thing in the morning when I wake up in order to hydrate my body and make sure I have enough electrolytes. And while I do any kind of physical training and after physical training as well, especially if I've been sweating a lot and certainly I drink Element in my water when I'm in the sauna and after going in the sauna because that causes quite a lot of sweating. If you'd like to try Element,

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So as you're probably starting to realize, there's a relationship between mechanical stimulation of the follicle and blood flow, both of which turn out to be critical for maintaining hair and for stimulating hair growth. Not surprisingly then, an increasingly common treatment for hair loss is Botox.

Botox is the brand name for what is otherwise known

as botulinum neurotoxin.

What is botulinum neurotoxin?

Well, botulinum neurotoxin,

sometimes just called botulinum for short,

is a toxin that's a bacterial toxin

that serves to cut the protein

that leads to synaptic vesicle release.

What in the world did I just say?

Well, when your nerve cells communicate with one another, they do that by way of electricity,

but the electricity within those nerve cells, those neurons,

triggers the release of chemicals from neurons

into the synapse, the little gap between neurons.

And the release of those chemicals

allows the next neuron to be chemically active

or in some cases,

it will suppress the electrical activity

of that next neuron.

Botulinum neurotoxin serves to cut a protein present in neurons so that neurons cannot release the chemicals that cause other neurons to be active.

So this actually is pretty serious.

If you were to get botulinum neurotoxin injected into your musels, you would be parelyzed.

into your muscle, you would be paralyzed

because the nerves that control contraction of the muscles would not be able to control the release of that chemical onto the muscle, which makes it contract.

Well, botulinum neurotoxin is commonly used

in what's called Botox.

Botox is something most people are familiar with because people get injected in and around their wrinkles because many wrinkles are triggered

by not just loss of tensile strength in the skin, but rather the nerves around the skin and in the skin are hypercontracted, which causes wrinkles.

So for instance, I have crow's feet. I like to think that's because I've laughed a lot and smiled a lot.

And it's probably also because I've squinted a lot in my lifetime.

I have crow's feet because the nerves there have pinched the skin on either side of my eyes and that's given me little creases there that are sometimes referred to as crow's feet. Botox injections can be applied to the scalp in order to relieve tension of the scalp. And in hearing that, it should be obvious why Botox is being used to try and offset hair loss.

It's decreasing the squinting, if you will, or the tensile nature of the scalp skin so that more blood flow can arrive at that stem cell follicle area.

So Botox treatment to the scalp is actually becoming pretty common. There are a couple of requirements

with this Botox treatment.

First of all it has to be done by someh

First of all, it has to be done by somebody who's really skilled.

There are numerous images online and websites online of so-called Botox fails where people have gotten too much Botox or the injections have been done too deep or not at the correct locations on people's face or scalp and it can give them droopy scalp or droopy eyes. All sorts of cosmetic nightmares can occur with Botox. The second thing to understand is that Botox does eventually wear off.

That botulinum neurotoxin doesn't stick around forever and provided it's done correctly at the correct dosages, it doesn't actually kill the neurons

that cause that tension of the skin.

So Botox injections have to be done repeatedly.

The efficacy of Botox for offsetting hair loss is not clear.

There aren't a lot of large-scale clinical studies

on this just yet, but it does seem to be

at least one reasonably safe alternative

to things like monoxidil.

Although I think if one were to just want

to increase blood flow to the scalp, things like low-dose to dalafilm, which doesn't seem to carry any of the side effects that monoxidil can carry.

We talked about those side effects earlier.

That would probably be the better alternative.

Botox is a fairly invasive procedure,

but some people opt for Botox treatment.

In fact, there's a syndrome called cutis verticus girata.

Some of you have probably seen this.

It's more typical in men,

although it does occur in women.

It literally means a lumping of the skin on the scalp or gyri of the scalp.

Gyri or gyris pertains to the Latin word knee,

so it means bump or knee.

And so at any time you hear the word gyra

in neuroscience or in biology, you're talking about a bump.

Sometimes see people will have ridges in the back

or it looks as if the skin was pushed together

kind of like a Sharpay dog, but on the scalp.

People with cutis verticus girata

almost always experience pattern hair loss.

Now part of the reason for that is cutis verticus girata

is also associated with some androgen

or testosterone related hormone issues

that we'll talk about in a little bit.

But in addition to that, it has been shown

that relieving some of those girata by injections of Botox

to allow those folds to sit flatter, A is effective.

It can lead to less of those gyri, those bumps,

and can improve hair growth in those regions,

even if those people don't take on any additional treatments

to address the hormone issues.

So that's really how people arrived at this understanding

that Botox might be a good treatment in general

for reducing the kind of squinting of the scalp

that can occur and the resulting hair loss in those regions.

I now like to turn our attention to the chemical variables

that control the duration of the growth phase of hair,

the duration of that catagen phase,

which is when that hair essentially recedes

from the inside out,

and the quiescent or semi-quiescent telogen phase. There are a couple of key chemical players here that we should all be aware of. First of all, the growth factor IGF-1, insulin growth factor one, which is produced by the liver, but that receives stimulation from the brain and pituitary to be released, is a strong regulator of hair growth. And we can think of it as the accelerator on hair growth. So it does that by extending that antigen or growth phase for longer. It doesn't necessarily speed up growth, but it extends it for a longer period of time. In addition, cyclic AMP, which is part of what's called a second messenger pathway, in fact, cyclic AMP is a second messenger, is also a key player in stimulating growth of the hair follicle. Now, cyclic AMP does many different things in many different cell types in the body. It really acts, as the name suggests, as a messenger between signals that arrive at the surface of cells and transmitting or conveying those signals to things that happen deep within the cells, such as the turning on and off of various genes. So when you hear second messenger, don't let that confuse you or overwhelm you. Just understand that the whole process of getting signals from the outside of cells into the center of cells and controlling gene expression, for instance, causing a stem cell to continue to give off daughter cells or causing a hair cell to continue growing for longer. That whole process is a bit like a bucket brigade of handing off water or a bucket from one component to the next or along a chain. It's like an assembly line.

I think that's probably the simplest way to think about it.

So for sake of this discussion,

IGF-1 is known to increase the growth of hair

by extending that antigen phase, as is cyclic AMP.

So those are going to be considered the accelerators, at least in this conversation.

The breaks on hair growth are going to be the things that either shorten the antigen phase

or that extend the catagen phase or this quiescent phase,

which is the tillogen phase.

And the two major breaks on hair growth

that we want to think about are PDE,

which is a phosphodiesterase.

Anytime you hear ACE, it's likely to be an enzyme.

And TGF-beta-2, all right?

So this is a particular growth factor

that somewhat counterintuitively doesn't stimulate growth.

It actually stimulates lack of growth or shortens growth.

So with all of that in mind,

and please do also keep in mind

that you don't need to remember all those specific terms.

Just understand that there are some factors

like insulin growth factor,

one that act as accelerators on growth.

And there are factors that act as breaks on growth.

We can start to think about why, for instance,

half of all people by age 50 start to lose their hair.

Well, they start to lose their hair

because of something called androgen-related alopecia,

which translates to English,

means testosterone and testosterone derivative-induced hair loss.

This is true in men and women.

So hearing that,

you should probably be wondering the following thing.

Young men have higher levels of testosterone

than old men, right?

Well, the answer is yes.

Although some older men in their 40s, 50s, even 80s

maintain testosterone levels similar to many men

in their 20s, but most don't.

It's a downward slope.

Starting at about age 40,

how steep that downward slope is depends.

Women too have testosterone.

In fact, women have higher levels of testosterone

than they do estrogen.

That's right.

A healthy woman has higher levels of testosterone

than she does estrogen.

However, women on average have lower testosterone

than most men, okay?

So they still have far more estrogen and far less testosterone than most men, but the level of testosterone that they have within their body is higher than the level of estrogen they have.

Androgens, such as testosterone and its derivatives, such as dihydrotestosterone,

which will be much the topic of what we're getting into next,

inhibit IGF1 and cyclic AMP.

Again, androgens such as dihydrotestosterone inhibit,

prevent the action of IGF1 and cyclic AMP,

which you just learned a few moments ago,

act to extend the antigen or growth phase of hair,

which then raises the question,

well, if young people, both male and female,

have higher levels of testosterone than they do $% \left\{ \left(1\right) \right\} =\left\{ \left($

when they're older,

why would people lose their hair when they're older

and not younger?

Ah, the answer lies in the conversion of testosterone to dihydrotestosterone.

Testosterone, most people have heard of,

dihydrotestosterone or DHT is made from testosterone.

There's an enzyme called five alpha reductase

that converts testosterone into dihydrotestosterone

in both men and women.

Dihydrotestosterone binds to the androgen receptor

at five times the affinity of testosterone.

So it is the most powerful androgen in humans

and it is responsible for a number of things

that we all really want and like,

such as mental vigor, physical vigor,

strength, healing capacity, drive, libido, and on and on.

So DHT itself is not bad.

So if we take a step back and we acknowledge

testosterone levels are higher in males and females

at younger ages as opposed to older,

but as they get older,

there's more five alpha reductase activity.

which is converting more of that testosterone

to dihydrotestosterone and dihydrotestosterone

inhibits hair growth by reducing IGF-1 and cyclic AMP.

Well, then we should all be aboard why it is

that by age 50, about 50% of people experience pattern hair loss. That is androgen dependent alopecia, okay? But translated to normal English is pattern hair loss. And in a moment, you'll understand why some people lose their hair from the crown region. I'm in the back of the head or back and top of the head, whereas other people lose their hair in the front of their head, in the flanks right on either side of the midline or maybe in the midline and front altogether. And that's because different people, depending on their genetic lineage, have different patterns of androgen receptors on their scalp. And the pattern of androgen receptors that you inherit indeed does come from your mother's side. And so this is what gave rise to the myth that if you want to know if you're going to go bald or not, just look at your mother's father. Doesn't quite work that way. In fact, if you think about the logic, you should really look at your mother's mother if you want to know your pattern of androgen receptors on your scalp. However, most women don't lose as much hair from their scalp or they have ways of covering up the hair loss in their scalp because their hair is just generally longer or they're using other approaches so that you never really get a clear picture of what the androgen dependent hair loss was in your grandmother, okay? Now we don't want to go too far down the genetics rabbit hole because as you know, you can't select your parents anyway. But if you want to know why, for instance, I'm losing a bit of hair on either side of the midline in the front, it almost certainly has to do with the fact that I have a higher density of androgen receptors there as opposed to say on the crown of my head where for whatever reason, my hair seems to grow thickest. Other people lose hair on the crown in the back and top but not in the front and some people lose it all over. Now you understand why hair loss occurs in certain regions of the body.

You should also understand that the androgen receptors on the face are also what are responsible for beard growth. And this is where it can get a little bit tricky but a lot of things will start to make sense if you can understand this and internalize this. If you have a high density of androgen receptors on your face, well then as your DHT levels go up with age, you will be able to grow a thicker and thicker beard. In fact, it is rare to see someone who can grow a thick beard in their youth but not so much as they get older. In fact, the reverse tends to be true. So the pattern of androgen receptors differs between the scalp and the face and the back. Okay, on your back, you have androgen receptors and their DHT stimulates hair growth. So if you know someone who has a very hairy back or if you have a very hairy back, that means you have a high density of androgen receptors on your back. If you have a beard and that beard is thick, well then you have a high density of androgen receptors on your face. However, a high density of androgen receptors anywhere on your scalp is going to predispose those regions to androgen dependent alopecia or hair loss in those particular regions, which is going to allow us to understand why all of the rest of the treatments for halting hair loss and for stimulating hair growth, almost all of those center on inhibiting either DHT directly

or five alpha reductase,
the conversion of testosterone to DHT.
So now I'd like to discuss the ways
that one can chemically adjust certain things
within the hair growth pathway,
things like IGF-1, PDE, TGF-beta, et cetera,
in order to stimulate hair growth or halt hair loss.
The first thing on this list
is actually going to be pretty surprising
to a number of you and that's caffeine.
We all think of caffeine as a stimulant that we drink.

I certainly drink coffee in Yerba Mate,

the occasional energy drink, things of that sort. Caffeine does many things besides stimulate our central nervous system and make us feel less sleepy, however. One of the things that caffeine does is it is a fairly potent PDE inhibitor. By being a potent PDE inhibitor, it indirectly stimulates IGF-1. Why? Well, because PDE can suppress IGF-1 and by ingesting caffeine or by applying topical caffeine ointment or cream to the scalp, you can suppress PDE sufficiently enough to increase IGF-1 and increase some hair growth or at least maintain hair growth in that region. This may come as a shock, it might seem a little bit esoteric or even outside the margins of typical treatments, but head-to-head, topical caffeine application can be as effective as minoxidil application without actually lowering things like blood pressure and potentially increasing prolactin and some of the other negative, let's call them negative because they are side effects of minoxidil.

So caffeine ointments and caffeine present in various hair treatments and creams, et cetera, is starting to become a more prominent theme out there. I will include a reference to caffeine

and its uses for offsetting hair loss.

Keep in mind that topical caffeine ointments shouldn't necessarily be applied every single day.

So this is the sort of thing you might do three times a week.

The concentration of caffeine in different ointments varies tremendously.

Most of the studies of caffeine on the stem cell niches

that control hair growth and extension

of the antigen phase of hair growth

have been performed in vitro in a dish.

Although there are some clinical studies exploring this,

they are not nearly as extensive in number or duration

as the studies of minoxidil

because this approach just hasn't been around quite as long.

However, when comparing side effects of minoxidil, cost of minoxidil, comparing the efficacy of caffeine and minoxidil, I think caffeine as a topical treatment for offsetting hair loss, stands as a pretty good choice if you're going to start exploring this pathway. And there's no reason to think that if you were to try the caffeine ointment and it didn't work for you or you didn't like it for some reason or you needed to stop it for some reason, that you couldn't stop it safely because it doesn't carry all the other and a blood pressure related effects and prolactinemia effects than minoxidil does. So if you look out there into the hair maintenance and hair replacement literature, you look at the treatments that are being sold, don't be surprised to see caffeine there. And also don't be surprised when I tell you what I'm about to tell you now, which is no, you can't simply just drink more caffeine in order to accomplish the goal of offsetting hair loss. It is true that when you ingest caffeine, it goes systemically. However, you have so many adenosine receptors throughout your body. Those adenosine receptors and the parking of caffeine in those adenosine receptors is the main way in which caffeine exerts its stimulatory effects, making you feel less sleepy. So it does that because then adenosine can't have its effects. which are to make you sleepy. Well, those adenosine receptors soak up so much of the caffeine that you would ingest orally that very, very little would make it to the scalp and to the hair follicles at the concentrations that you would want. So that's why you have to rely on the application of these caffeine ointments about three times a week. Keep in mind that no one has really explored the dosages of caffeine in these ointments

in a systematic way. We're still in the early stages of all this, but I do think it's important to mention caffeine because of the lower incidence of side effects. at least reported side effects, and the general safety margins and the head-to-head, essentially comparable efficacy with minoxidil because minoxidil has a bunch of other issues. Now keep in mind that both minoxidil and caffeine are generally used as a preventative for reducing hair loss over time. They are not expected and they do not, as far as we know, create new hair growth to any sufficient degree. If any of you have used caffeine ointments for minoxidil and observed new hair growth that was robust, please put that in the comment section. I'd be curious about those experiences, but as far as I know and from the clinical literature that I read, there's no examples of that. One other point about caffeine, it does appear that caffeine can not only indirectly stimulate IGF-1 in the antigen phase of hair cell growth by way of reducing PDE and TGF-beta, but it also seems to reduce apoptosis, which is naturally occurring cell death of that stem cell niche. We've been talking a lot about the antigen or growth phase of hair. We also talked about the catagen, where the recession of that hair from the inside out. But remember that third phase, the tillagen phase, where that whole bulb down at the bottom, the bulge as it's called, gets pinched off

off to the grave with it.

It appears that caffeine can offset the death of that niche and potentially maintain the stem cell population longer. Making caffeine a really good choice to think about in conjunction with the various chemical treatments aimed at directly attacking the DHT pathway that we'll talk about next.

and the whole thing dies and takes the stem cells

So there's one very direct way to increase hair growth and maintain the hair that you have on your head.

And that's to increase IGF-1.

That can be accomplished through prescription drugs, such as growth hormone and things that stimulate

the release of growth hormone and IGF-1.

Keep in mind, growth hormone is released

from the anterior pituitary during the first hours

of sleep, especially when you haven't eaten anything

for the two hours prior to sleep.

And especially when you get regular bedtimes.

Yes, this is a real thing.

If you are going to sleep at variable bedtimes,

especially if you go to sleep much later

than your usual habitual bedtime,

you will miss that growth hormone pulse

that normally occurs in the first two to three hours of sleep.

This does not mean that you need to be neurotic

about getting to sleep at the exact same time every night.

There's probably a plus or minus 30 minute window.

And it doesn't mean that you can't stay out late

or have a bad night's sleep every once in a while

or get woken up.

Your hair isn't going to fall out.

My dad's story, not withstanding.

Your hair isn't going to all fall out because of that.

But you do want to try and get that natural growth hormone release each night.

And as I mentioned, there are prescription approaches

and those are growth hormone itself

and things like sermorellen, which is a peptide

that increases growth hormone.

It's actually a secretogog.

It causes the secretion of growth hormone

and thereby increases in IGF-1.

This is well documented.

Both growth hormone and IGF-1,

as I mentioned, are available by prescription.

They are not without their side effects.

So they do cause growth of all tissues.

They do increase height in children.

They increase bone mass and density.

They can increase mainly fat loss,

not so much muscle size, but they can increase leanness.

And they increase hair growth.

They can increase turnover of skin. They can make skin look more youthful. All these things sound wonderful. And yet they also have problems because they can increase growth of small tumors that normally might be eliminated. So there is an increased cancer risk with growth hormone treatments in IGF-1 or anything that increases IGF-1, such as sermorellen, hypermoralen, any of the peptides that you hear about nowadays. That said, a number of people are using sermorellen as an indirect way to increase IGF-1 and thereby to increase hair growth. It does seem to be fairly effective in doing that, especially when done in conjunction with other treatments that we'll talk about. But keep in mind, these are prescription drugs and they do carry their own risks. The other thing about stimulating the natural production of IGF-1 is that as its name suggests, insulin growth factor one, it is insulin dependent in many ways. If not for its secretion, then for its action at the hair follicle and stem cell. So one of the things you really wanna pay attention to is to avoid being insulin resistant or rather you want to try to be insulin sensitive. So if you're carrying a lot of excess body fat, if you're obese or if you have type two diabetes, many are insulin resistant, you're going to want to deal with that. There are a number of prescription treatments to deal with that. Nowadays, people are very excited about all the GLP-1 agonists. But in addition to that, regular cardiovascular exercise and resistance exercise, making sure that your body fat percentage is not in excess of where it needs to be. And everyone has a slightly different idea of where it needs to be, both cosmetically and for health. So that's a conversation between you and your doctor

and you and you, frankly.

But you want to avoid being insulin resistant

because being insulin resistant and being obese

can indeed lead to hair loss.

And there are many people out there who are not obese,

who nonetheless are experiencing hair thinning and hair loss

because they are insulin resistant

by way of reduced IGF-1 activity.

There are a few supplements.

So these are over-the-counter supplements,

such as myoenositol,

taken at dosages of about 900 milligrams before sleep,

which by the way can also assist in sleep.

Or things like berberine or metformin,

which are known to improve insulin sensitivity.

Each and every one of these has its own profile

of positive and negative effects.

The lowest number of side effects is associated

with 900 milligram myoenositol,

taken about 30 minutes before sleep,

most because it can make people a little bit drowsy.

And it does aid sleep in some really great ways.

But really exercise and maintaining

a healthy nutritional program are also going to be key.

You just can't discard those.

Berberine, which is a substance made from tree bark,

is sometimes called the poor man's metformin,

metformin being the equally effective prescription version

of berberine, or put differently,

berberine is the non-prescription,

but equally effective version of metformin.

Keep in mind that both berberine and metformin

dramatically reduce blood sugar levels.

And so if you're not going to take them with carbohydrates,

it can lead to some feelings of discomfort.

That's why I'm not a fan of them.

But if your goal is to really improve insulin sensitivity,

they are considered very potent tools

on the list of things that can do that.

The other thing that's really important

for maintaining proper hair growth,

this antigen phase, is that you need sufficient iron.

This is because iron and ferritin play a key role

in the cell growth pathways that go from the stem cells to the stimulation of keratin within the hair itself. Don't have time to go into this pathway in a lot of detail, but you can have your blood levels of iron measured. This turns out to be pretty important because you don't just want to start popping iron supplements because too much iron can be toxic, too little, means you're anemic. For women, the levels of iron that you want are somewhere between 25 and 100. and for men, somewhere between 30 and 150. Fortunately, the tests or the blood tests for iron are usually a very inexpensive add to your current blood panel. So if you're going in for a blood panel for LDL, HDL, kind of typical things, or if you're doing the more elaborate hormone testing, which I do recommend people do if they can afford it, or if their insurance covers it. do ask for an iron test as well. And if your iron is low, you may need to supplement your iron. If your iron is too high, that's not good either, but iron plays a key role in the antigen phase of hair growth. So you want to make sure you at least have sufficient iron, and if you don't, you want to make sure that you're getting it from nutrition and or supplementation. Okay, so earlier we were talking about dihydrotestosterone. Dihydrotestosterone, just to jog your memory, is a form of testosterone that can bind the androgen receptor at five times the affinity of regular testosterone. Both men and women have a lot of testosterone relative to estrogen. It just so happens that men tend to have more testosterone than women do overall. But both men and women, as they age, convert more of the testosterone they have to dihydrotestosterone.

And dihydrotestosterone does two things

that are bad for hair maintenance and hair growth.

First of all, it shortens the antigen phase,

that growth phase.

So whatever hair is going to grow

is going to occur over a shorter period of time. Second of all, because of the presence of androgen receptors on the stem cell niche area and around it, it actually miniaturizes the follicle and the stem cell niche. In other words, it makes the population of cells that give rise to more hair protein, smaller, and can eliminate it altogether. And that is why anything that reduces five alpha reductase is going to reduce DHT is going to maintain or extend the growth phase, the antigen phase of hair growth, and is going to offset or prevent some of the tillagen phase, the pinching off and the removal of that stem cell niche. Now, one substance that we know can inhibit five alpha reductase, although it does it pretty weakly, is solpalmetto, which is an extract of the solpalmetto berry. To be honest, I don't know how this was initially discovered. If anyone knows, please put it in the show note captions. When I looked online, I found a bunch of conflicting stories about who was the first person to start extracting the extract of the solpalmetto berries. So I have no idea which one of those is true. Maybe somebody can tell me. The good thing about solpalmetto treatment is that it is known to have very few, if any, side effects. There might be some side effects in about 1% of people, but it's not associated with a lot of side effects. It's also known that when taken at about 300 milligrams per day, and here it doesn't have to be that strict. Most of the studies that I explored involved taking anywhere from 200 to 500 milligrams of solpalmetto, but most of them focused on about 300 milligrams of solpalmetto per day, divided into two or three doses. Why? Well, it has a relatively short half-life,

meaning it's gonna get cleared from the bloodstream,

and more importantly, its biological action is gonna be very short-lived. So if you can get a hold of 50 or 100 milligram capsules or tablets of solpalmetto and divide those up, take them in the morning, afternoon, and evening, or even just in the morning and evening to achieve a total of about 300 milligrams per day, that's going to very likely help you hold on to some of the hair that you would have otherwise lost, maybe even grow some new hair. And I say maybe give you some new hair growth because solpalmetto is not known to be a particularly robust treatment for the reversal of hair loss. It's known to have some effect, but it's generally taken in conjunction with a bunch of other things. And so it's really hard to tease out just what amount of hair growth or prevention of hair loss is due to solpalmetto specifically. But I mention it here because the mechanism of action is logical and fairly well known, which is this weak effect in reducing five alpha reductase and thereby reducing DHT. So again, because it has relatively low side effects, even though it's not super effective, and because it's fairly low cost and it's available over the counter, I felt it was important to include. Now, as soon as people hear things like solpalmetto bury they're probably also thinking, oh boy, here come all the herbals. Now, keep in mind that there are some herbal compounds that have pretty robust biological effects. And we talked before in our optimizing testosterone episodes about things like tonga ali and phytogeogrestis, which taken in correct dosages and in the correct way can be pretty potent for increasing luteinizing hormone and free testosterone. Not gonna have huge effects. It's not like taking exogenous anabolic steroids, but they can have real effects.

And so herbal compounds can be potent.

However, I do acknowledge that there's a vast market out there of herbal compounds and plant-based compounds that people are arguing,

mostly the people who sell them are arguing,

can increase hair growth.

And there are some decent studies of these things.

The hard part about studying these herbal compounds

and these plant-derived compounds for increasing hair growth $% \left(\mathbf{r}\right) =\left(\mathbf{r}\right)$

is that they are often taken in combination with one another.

In fact, most of the hair growth supplements

that involve these herbal compounds and plant compounds $% \left(x\right) =\left(x\right) +\left(x\right)$

include five or 10,

sometimes even more things altogether.

So teasing out which ones are effective

and which dosages are effective is nearly impossible.

They're just too many variables.

So you will hear, for instance, that green tea extract,

racy mushroom, pumpkin oil, zinc, curcumin,

that all of these things can increase hair growth.

Perhaps the only thing on that list

that makes logical sense with respect to the biochemistry

is that curcumin is known to, in some people,

be a potent inhibitor of five-valve reductase and DHT,

so much so that I can personally say,

for me, I once took curcumin, turmeric, it's also called,

in high dosage, this is about a gram to two-gram capsules, $% \left(1\right) =\left(1\right) \left(1\right) \left$

and I felt absolutely terrible.

I mean, just dreadful.

I don't care if it allowed me to keep my hair forever.

I would rather lose all my hair three times over.

Well, I guess that means it would have grown back.

I'd rather lose all my hair.

Let's just put it that way.

Then ever take curcumin or turmeric in high dosages again.

And in saying that,

I know that many people take turmeric and curcumin

and really like its anti-inflammatory properties

and don't experience any side effects.

I just happen to be particularly sensitive to curcumin

by way of this DHT pathway,

and I know this by way of blood work,

so I'm never going near that stuff again.

That said, a lot of these herbal compounds and cocktails

probably will have some minor marginal, infinitesimally small, somewhere in that range, effect on maintaining hair growth or in stimulating new hair growth. It's just that there really aren't clinical studies to support any one of them. And that's why I singled out Sol-Palmetto as one of the few for which the biochemical pathway of inhibiting file file for reductase and the low incidence of side effects and the fact that many people have used it with some degree of success makes it a standalone, I wouldn't say recommendation, but a consideration. Another commonly discussed and used commercial compound for offsetting hair loss and stimulating hair growth is ketoconazole.

Sometimes this is known as Nizrol, where Nizrol is the brand name of a shampoo. Ketoconazole is an antifungal that was initially developed to treat dandruff

and severe psoriasis.

So ketoconazole has been shown to be effective in increasing hair number.

It's also been shown to be effective in increasing hair diameter, which is somewhat surprising

because one of the common side effects of ketoconazole

is drying, shining, and brittle hair.

So what's going on there is a little unclear.

We'll return to that in a moment.

The mechanism of action for ketoconazole is pretty interesting.

Remember earlier we were talking about the sebaceous gland and the production of sebum, that oily stuff,

whose very name seems to evoke disgust in certain people? Well, ketoconazole can disrupt some of the fungal growth

that frankly we all have on our scalp all the time.

I know this is a surprise to many of you, but you are constantly bombarded

but you are constantly bollibarded

with viruses, bacteria, and funguses all the time.

But we manage to battle those off with our immune system, either by physical barriers, such as an oil barrier, like the sebum, or through antimicrobial action.

So chemical approaches, our immune system,

the sebum, et cetera.

Ketoconazole acts as an antifungal

that in some way seems to reinforce the properties of sebum

at keeping out other fungal infections.

And the net effect, at least as far as we know,

is a mild reduction in DHT.

Now exactly how this happens isn't really clear.

What is clear is that the use of ketoconazole shampoos

two to four times per week,

with a scalp contact time of about three to five minutes,

has been shown to give about an 80% response rate

of maintaining hair that would otherwise be lost.

So that's pretty dramatic, 80%.

What is less clear is whether or not ketoconazole shampoo

can actually stimulate new hair growth.

But as you're probably starting to realize,

this is always a bit of a tough thing

to disentangle maintenance of hair

that you would have lost versus new hair growth.

Certainly that's an easy thing to disambiguate

if you have a patch of scalp

where there is absolutely no hair.

These so-called dead zones that you can resurrect

with certain treatments.

But what about areas of your scalp where hair is thinning?

So for instance, on the top of your head,

this is where many women will first experience

pattern hair loss alopecia is right at their midline.

You know, especially if they have a part right there,

they'll start to notice that under very bright light,

fluorescent lights in particular,

they'll notice a thinning of their hair there

or in the forehead region.

They'll start using ketoconazole shampoo.

Again, the typical recommendation is two to four times

per week with a scalp contact time of three to five minutes

and really rubbing it in and then rinsing it out.

You don't need to be super vigorous,

but you want to make sure

that it gets down into the follicle

and around the follicles,

not just sitting on top of the hair,

which is going to be especially important for people who have long hair to really massage it in. Well, they will experience a growth of hair in that particular region that almost certainly were due to miniaturization of the hair follicle and reduction of the total population of stem cells in that follicle, as opposed to complete loss of the stem cell population.

The reason we say this is that there's no reason to think that ketoconazole can actually stimulate IGF-1 or activate growth itself.

It's probably offsetting some of the reductions in the antigen phase and some of the exacerbation of the telogen phase.

Now, if you decide to use ketoconazole as an approach to offsetting hair loss, it's very important that you get a hold of a shampoo that's at least 2% concentration of ketoconazole. This is important because a lot of the ones that are available out there, especially online, are going to be 1% or lower.

So you want to try and obtain a ketoconazole shampoo of 2% or higher concentration of ketoconazole because it has other things in it, of course. I should mention that there are occasional side effects with ketoconazole, the rates of side effects from the meta-analysis and reviews that I read, where somewhere between one and 8% of people will experience some side effects.

But those side effects tend to be pretty mild, things like irritation of the scalp, things like finning and brittleness of the hair that sometimes can be offset by using shampoos that contain things like biotin.

I know many people are probably curious about biotin, which is a protein that can be incorporated into the keratin.

Whether or not different biotin and rich shampoos can really enhance the total amount of biotin that gets incorporated into the hair isn't clear, but it is clear that having sufficient biotin around is important.

So if you get a little bit extra from your shampoo,

you can imagine how that would quote-unquote tap off the amount of biotin in that hair. And there are people out there saying that biotin and rich shampoos have done wonders for them. Who knows, I'm not going to dispute their experience. So if you're going to use Ketoconazole, keep in mind that the more typical brand names that are out there, you know which ones they are, oftentimes don't have 2% or if they do have 2%, they can be very drawing and lead to brittle hair. There are newer and now fortunately, a greater variety of Ketoconazole containing shampoos. We as a podcast and I don't have any affiliation to any of these, but I will provide a links to a couple of the more prominent ones that are known to have 2% concentration of Ketoconazole as well as some other things in them known to offset some of that dryness and brittleness that Ketoconazole shampoos can trigger. So by now, I think it will be abundantly clear why inhibiting five alpha reductase and thereby reducing DHT should increase hair growth because of the negative impact that DHT has on the hair follicle. The major player in this whole story around inhibiting five alpha reductase and reducing DHT to maintain or increase hair growth is going to be finasteride and it's close cousin, dutasteride. Finasteride is effective in reducing DHT because of its actions in reducing the type 2 isoenzyme or isoform of five alpha reductase. It turns out there's three different isoforms or what are sometimes called isoenzymes

of five alpha reductase.

This is getting pretty far down in the weeds.

What I think most of you just need to know

is that finasteride reduces DHT.

That's the net product of finasteride.

And in doing so, it can increase hair count

by as much as 20%, pretty remarkable if you think about it.

In addition, finasteride treatment done properly,

which we'll define in a moment,

can reduce hair loss in 90% of all people that take it.

That is, I mean, near staggering.

I mean, there aren't many pharmaceuticals out there

that have that kind of efficacy, really dramatic.

And in addition, it's known to increase hair thickness

by about 20 to 30% overall.

So not just create new growth of hairs and thicker hairs,

but whatever hair you do happen to have on your head,

it can further thicken those.

So the finasteride story is one, I think, of general success.

I mean, it really seems to improve hair growth

and help you hold on to the hair that you have.

The issue with finasteride is twofold.

First of all, it is known to have

some pretty significant side effects

if it's not dosed properly

and in particular populations of people.

And this is because there is a wide variation

in the amount of the different isoenzymes that people make.

This is why I brought up the isoenzymes earlier.

Some people make more of isoenzyme one and three.

Some people make more of isoenzyme two and three

and every variation thereof.

So when people take finasteride,

some people are very strong responders

and they achieve really effective hair regrowth

and maintenance of hair.

Other people less effective,

although still pretty impressive,

but the catalog of side effects

that people experience at a given dose varies widely.

So there's a lot of trial and error that has to take place.

Also should point out that finasteride

comes in two major forms.

There's an oral form and there's a topical form.

So this is not unlike our discussion

of minoxidil earlier.

Topical finasteride is typically taken

in 1% solution or ointment and rubbed into the head.

Sometimes it's now also incorporated into shampoos,

but typically it's put into a solution

that people rub into their head.

And it is thought that the 1% solutions are equivalent

to one milligram of systemic finasteride. Now we need to take a step back and ask why was finasteride developed in the first place? Well, finasteride as a fairly potent five-alpha reductase inhibitor, it's great at lowering DHT. It was developed for treatment of prostate enlargement and various issues of the prostate that are associated with elevated DHT that occurs with age. The topical finasterides were designed with the hope that the finasteride would make it into the hair follicle and would inhibit DHT there and allow for more growth of the hair, which apparently it does, but not make it into the systemic circulation or at least not at concentration sufficient enough to cause as many side effects as with the oral dosing. Now the problem is it does make it into systemic circulation. The issue is also that topical application of finasteride is harder to dose than oral finasteride. I'm not saying you should be taking oral rather than topical finasteride, but keep in mind that the dosages of finasteride that have been shown to be effective for inducing hair growth cover an enormous range. So as low as 0.01 milligrams per day and as high as five milligrams per day, which is a just staggering range. Now when trying to simplify the problem of how much finasteride to take, either by way of oral tablet or by way of topical solution, we can get a bit of leverage on this by thinking about how much DHT reduction occurs as a function of dose. And there finasteride shows this really interesting, what's called logarithmic distribution. What it means is that for a dosage of 0.01 milligrams of finasteride, you're going to achieve approximately 50% reduction in DHT and that's systemic DHT. So this is a blood draw measuring your DHT, then taking 0.01 milligrams of finasteride, again, 0.01, very low dose of finasteride,

or a period of couple of weeks, measure people's DHT in their blood. Again, you see that it's reduced by 50%. However, at increasing dosages of 0.2 milligrams, 1 milligrams, 5 milligrams of oral finasteride per day over the same period of time, the increase, or I should say the reduction in DHT doesn't increase linearly. It's not that you go from 50% to 60% to 75% to 100% with each increasing dose. It tapers off, it kind of flattens out. It tends to increase a little bit, but it's sort of a gradual slope increasing as you head from 0.2 milligrams out to 5 milligrams. So what this means is that given that reducing DHT can cause very side effects, sexual side effects, reductions in either sexual function or sexual drive, as well as overall drive and motivation, sometimes even some depressive symptoms, that everything points to taking the lowest effective dose of finasteride and starting with a very low dose of finasteride because low doses of finasteride, even at that 0.01 milligrams taken orally daily, are already leading to a 50% reduction in dihydrotestosterone and thereby, taken for a long enough period of time, should offset hair loss and stimulate hair growth. One of the problems, however, is that people will start taking finasteride at a low dose, you know, 0.1 milligrams or 0.2 milligrams, maybe even 0.01 milligrams, and there will be a reduction in their DHT, but because of the long duration of that antigen phase, they don't see a lot of change in hair growth in the first month or even two months. And so what they end up doing is increasing their dosage and then they start to see hair growth, but then they start to experience more side effects. Now, the side effects of oral finasteride are serious enough and common enough in people that take finasteride that the topical solutions were developed. But there too, one needs to exercise caution

because if we are going to translate
between topical finasteride and oral finasteride,
with the understanding that topical finasteride
can actually make it into the systemic circulation,
we need to look at what's been shown in clinical studies,
which is that, for instance,
taking one milliliter of 0.25% finasteride
applied to the scalp,
this is a very typical recommendation,
translates to the same thing that would be achieved
with 2.5 milligrams of oral finasteride.
And when I say translates to the same thing,
what I mean is it leads to the same concentrations
in the blood.
Now, consider that 0.2 milligrams,
0.2 milligrams of finasteride in the blood
is known to be effective in generating new hair growth

Now, consider that 0.2 milligrams, 0.2 milligrams of finasteride in the blood is known to be effective in generating new hair growth and maintaining hair that one already has when they start the treatment.

So if you think that the topical finasterides are actually creating lower overall systemic concentrations of finasteride, that is not necessarily the case. Again, one milliliter of topical finasteride at 0.25% leads to a 2.5 milligram concentration in the blood when the effective dose within the bloodstream by taking it orally as a pill is 0.2 milligrams. That might not seem like a big deal to you, although it is a big deal, right?

We're talking 2.5 milligrams versus 0.2, but it is a huge deal when you consider that the side effects of finasteride increase as you increase the concentration of finasteride in the bloodstream.

So where does this leave us?

Should people who are interested in taking finasteride take the oral form at low dose or take the topical form and simply try and apply it less often or guess at what their systemic concentration of finasteride is?

Well, it's going to vary from person to person.

Well, it's going to vary from person to person. Some people are very sensitive to finasteride and not in the good sense.

The side effects just really show up quickly

and they tend to be dramatic.

Other people, not so much.

The dosage recommendations that I was able to arrive at based on the clinical studies and frankly in discussion with some doctors who prescribe finasteride were the following.

0.5 milligrams to one milligram of finasteride as a tablet per day seems to be an effective

and pretty safe starting place for most people.

Now, some people will find that even that 0.5 milligram dosage

is just going to cause side effects

that are not going to work for them

and they're going to either have to reduce

their dosage of finasteride or move to the topical

or maybe cease taking finasteride altogether.

But for many people out there,

that's going to be pretty well tolerated.

The key thing here is that one is going to have to wait

some period of time to see whether

or not any hair growth occurs.

It is a naive and frankly a foolish approach

based on what we know about the duration

of that antigen phase of the hair

to do one of these treatments,

wait a week or two and then decide to up your dose.

Now, it is not foolish to reduce your dose

if you're experiencing bad side effects,

but to simply increase your dose

because you're not getting results guickly enough,

that's not going to be the best approach.

I really encourage people who are going to explore

the finasteride route to think of this as a long-term project

and to really ratchet up slowly, if at all,

starting initially with a low dose

taken for a long period of time,

maybe even as long as 25 weeks

before considering going up any further.

Certainly, as I mentioned before,

if you need to go down further,

that's not going to be a problem,

at least not in terms of reducing side effects.

I mean, you're not going to get additional hair growth,

but you're certainly not going to increase your side effects

if you reduce your dose. However, I will talk a little bit later about post-finasteride syndrome, which is something that's getting increasing attention nowadays, that's something that occurs after people have taken finasteride for an extended period of time. Now, some of you have perhaps heard, and I'll just tell you right here, that the topical forms of finasteride are associated with far less side effects. Now, that might come as surprising given that topical application of finasteride can lead to systemic distribution of finasteride, but the numbers that are out there right now are it's that topical finasteride is associated with 30 to 50% fewer side effects, or 30 to 50% less severe side effects than oral finasteride. Now, there are several things probably responsible for that. One is that people tend to ratchet up their dose of oral finasteride pretty quickly, but keep in mind that the effective dose of finasteride in the blood is 0.2 milligrams. Now, earlier I said, the typical topical finasteride solutions are 0.25%. People are taking one mil of it, that equates to 2.5 milligrams, and so it seems like a massive overdosing. Ah, but here's the discrepancy, and here's where we can arrive at some reasonable recommendations. If you decide that finasteride's right for you, you get a prescription, I would hope that you're monitoring your DHT levels and other hormone levels, that would be ideal and working with a doctor, please. 0.5 milligrams to one milligram per day of oral finasteride seems to be the best starting place. For topical finasteride, it's going to be that one milliliter of 0.25% that we talked about earlier, but that's taken only one time per week,

and you can fully expect that right after the application, you will have higher levels of finasteride in your bloodstream and therefore lower levels of DHT, and that will alter across the week. Most people are not going to be able to measure their DHT on a day-by-day or even weekly basis, it's just too expensive and labor-intensive, but I think those dosing regimens ought to get people into more or less the same category of optimizing hair maintenance and hair growth while minimizing finasteride side effects. One point about finasteride taken either alone or in combination is that in recent years, really in the last five or so years, there's been increasing discussion about so-called post-finasteride syndrome. Now, post-finasteride syndrome is indeed a new phenomenon in the sense that finasteride has been prescribed for a very long period of time for treatment of the prostate at dosages of about five milligrams per day. That's a very high dose, sometimes lower, but as high as five milligrams per day. And for many years, there was no discussion about this post-finasteride syndrome. What is post-finasteride syndrome? Post-finasteride syndrome is when typically it's males, this is where it's been described, will take finasteride at any range of dosages from one milligram to five milligrams per day. They're either doing this for prostate or more likely they're doing it to offset hair loss and increase hair growth. And then they stopped taking finasteride for whatever reason, financial or it wasn't working for them or the side effects were not to their liking. And they start to experience some very severe, what can only be called syndrome effects, such as very reduced libido, very reduced erectile function, very reduced mood to the point of depression, even suicidal depression. So this is pretty scary stuff, especially since it's occurring at an age

when most of these things are not typically occurring in males, they can occur, but they're not typical of younger males in their 20s and 30s and early 40s. So we have to ask ourselves, what's going on here? After all, people have taken finasteride for the prostate at fairly high dosages without this post-finasteride syndrome when they've stopped. Now these young males are taking finasteride, they're coming off finasteride and they're getting this very severe, very debilitating post-finasteride syndrome. This has become a kind of hot topic, enough so that medical doctors who've been prescribing finasteride for a very long time have been forced to address this. I think at first they were sort of perplexed and thought, I don't know, this might be psychosomatic, whatever that means, I mean, as a neuroscientist who works on mind-body connection, we know that nothing is truly psychosomatic, everything is of the mind and body. But the point is that enough medical attention has been placed on post-finasteride syndrome and trying to unravel exactly what that is, where there are now a few general conclusions about what might be going on. First of all, it seems that younger males taking finasteride, in particular high dosages, to improve hair growth or offset hair loss, seems to be one of the key variables. We're not seeing this post-finasteride syndrome as much in older males. In fact, it seems to occur more in males in their 20s and 30s than males in their 40s and older. So that's one thing that may relate to the ways in which dihydrotestosterone, we know has a very key role in early embryonic development, it's actually what's responsible for the male genitalia. It's also responsible for certain things in female development, but mainly in utero, it's responsible for male development and development of the penis in particular. And then around the time of puberty,

dihydrotestosterone acts again in what's called its activating effects to further increase growth of the genitalia, increase the musculature, bone growth, et cetera, and increase libido and a number of other things. It's probably also involved in the activation of puberty. It's certainly not the only hormone involved in the activation of male puberty, but it's certainly one of the key players. So dihydrotestosterone has these known early roles in embryonic development and in puberty. But what post-finasteride syndrome seems to indicate is that dihydrotestosterone is likely having further effects on male maturation, in particular maturation of the hypothalamus and areas of the brain that continue well into one's 20s and maybe even one's 30s. And here I just want everyone to keep in mind that we tend to think about development as childhood, teenage years, young adulthood, adulthood, but really development never stops. Development is something that starts at conception and birth, of course, and then extends all the way out until the point when we die. So even if we live to be in our late 90s or achieve 100 years of age, development is occurring that entire time and these different hormones such as dihydrotestosterone are having different impact across the lifespan and in different ways across the lifespan. So there isn't a clear conclusion about what post-finasteride syndrome is really all about, but it points to the fact that DHT is likely to be involved in development of the brain and the brain to genital axis because I mentioned that because so many of the side effects that are associated with this post-finasteride syndrome seem to center on sexual side effects, although they're also the depressive side effects, of course those can be related to one another in either direction. So while I do understand that loss of one's hair or potential loss of one's hair can be particularly troubling and anxiety provoking even cause depression in some cases, I am sensitive to that.

You also want to be sensitive to the fact that some of these treatments such as finasteride can carry very serious side effects even if you come off them, AKA post-finasteride syndrome. As long as we're talking about finasteride and this general pathway of five alpha reductase inhibition and thereby DHT inhibition and on and on, topics and themes and nomenclature you are now very familiar with, we have to talk about dutasteride. Dutasteride is vet another molecule similar to finasteride. but remember those three isoforms of the five alpha reductase enzyme? Well, it inhibits all three, mainly type one and type two, but also type three and it does it very potently. So as a consequence, the typical dosage of oral dutasteride, get this, 0.5 milligrams to 2.5 milligrams taken orally, works two to five times faster than typical finasteride at inducing hair regrowth and reduces DHT by, get this, 95%. So it's just near flat lines DHT and that can occur at concentrations as low as 0.5 milligrams, although you will see prescriptions and people taking dutasteride anywhere from 0.5 milligrams all the way to 2.5 milligrams orally. Now, not surprisingly, dutasteride is associated with a lot of side effects related to the DHT pathway. So things like reduction in sex drive, reduction in overall drive. It also tends to impact other hormone pathways. So increases in estrogen prolactin. That's why gynecomastia, male growth of male breast tissue sometimes occurs when people take dutasteride. And so you're probably asking, why would anyone take dutasteride? Why not just take finasteride and wait for that hair growth? Well, the answer is that people are often very impatient and it turns out that dutasteride works about two to five times faster than finasteride. So some people don't want to wait a full 30 weeks or 40 weeks or 50 weeks or more

in order to grow their hair back.

And they're very concerned about the hair loss that's occurring.

So they will take what I hope would be a very low dose of dutasteride.

I realized that there are ways to take dutasteride

that can be safer, offset some of these side effects.

But by my read of the literature,

if one is going to try to mildly inhibit the DHT pathway,

things like tall palmetto,

things like topical caffeine,

which has some effect on the androgen pathway.

But as we talked about earlier, tickles other pathways,

things like ketoconazole, mild reduction

in androgen receptor pathways in the follicle

and very direct because it's applied directly to the scalp.

Things like that are going to be the best route

for mild reductions in DHT as an attempt

to maintain hair or grow hair.

Whereas if one really wants a potent stimulus

for increasing hair growth,

that's very likely going to be finasteride

and hopefully low enough dosages of finasteride

and hopefully a patient enough patient person

that they are willing to wait the duration of time required

for that hair growth to come back

because they understand that that androgen phase

takes some time.

Now the holy grail of all this hair stuff

is in understanding that no one specific treatment is magic.

And in fact, there are now a number

of good meta-analyses comparing

the various treatments we've talked about today,

alone or in combination.

And we can summarize that pretty easily

by saying that combination treatments

that involve a mechanical stimulus

and a chemical stimulus are always going to be better

than either one alone.

And within the mechanical category,

the stimulus that seems to work best is microneedling.

So the combination of microneedling and finasteride

can lead to some pretty robust and impressive hair regrowth.

So much so that people that were,

I guess we'd only describe them as pretty bald or bald can regrow significant amounts of hair. I've never seen examples of people who were completely bald, meaning lacking all scalp hair to grow back a full head of hair. But the combination of microneedling and finasteride is probably the most effective way to go. And if you're going to do that, there's no reason why you couldn't also use ketoconazole shampoo, couldn't also use salt palmetto. There's no reason to think that these things collide with one another, although anytime you're inhibiting a DHT pathway or whether or not you're increasing or decreasing any hormone pathway for that matter, vou want to be careful about layering in too many different treatments because you don't want your DHT level to go too low. I mean, let's keep in mind that even if you take a very low dose of finasteride and reduce your DHT and don't have side effects, maybe just the mere addition of salt palmetto, which leads to a slight reduction in DHT combined with some caffeine ointment would be sufficient enough to start inducing some of the low DHT associated side effects. So you really have to see for you. And that's one of the major issues in this whole field of hair growth and regrowth is that people are highly individual in their response and in their side effect profile to these various treatments. But there's an enormous range there and unfortunately there's no way to know a priori before doing these treatments what your response is going to be in terms of side effects. So you're simply going to have to explore and I would hope that you would explore starting with minimal possible effective dosages and to do that in coordination with a medical professional. So you could really evaluate these things at the level of blood and cosmetic changes. And in fact, that's a pretty good motivator for thinking about the different treatments

that we talked about today. Everything from mechanical treatments, as simple as massage, which we've all heard about, but for which there really aren't a lot of data for supporting hair growth, but certainly things like microneedling, which is a mechanical stimulus for which there are pretty good data that it can improve hair growth. Also things like salt palmetto, a weak DHT antagonist. Also things like Ketoconazole shampoo, which again is a weak DHT antagonist and operates through some other pathways as well to stimulate hair growth. My suggestion is that anyone, young, old, male, female, who's thinking about embarking on various treatments for offsetting hair loss and stimulating hair growth, consider both mechanical approaches and the approaches that attack the chemical pathways that can stimulate hair growth and can inhibit the inhibitors of hair growth. In fact, that's the reason why we spend so much time on the biology of hair growth and what shuts down hair growth early on in today's episode. And then systematically went through each of the various treatments that relies on, and in some cases hinges entirely on either a mechanical stimulus or a chemical stimulus in order to exert its effects. My goal today was not to overwhelm you with a ton of biology about hair, although we did cover a lot of biology of hair and stem cells and hair growth. My goal in describing all that was really for you to be able to hear about various treatments. whether or not it's lasers or PRP or finasteride, dutasteride, or whatever is coming next that we're sure to hear about soon online and from the medical community, and to be able to place those into bins related to their known or potential mechanisms, and then to be able to step back and evaluate which, if any, of those treatments might be right for you.

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