A Master Guide To DHT Reduction

The *Four* Major Levers

PerfectHairHealth

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Disclaimer

Summary

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1

The DHT Master Flowchart

Summary

- Not everyone has time to uncover the science behind DHT, its connection to hair loss, and how to lower it. So here are the takeaways: ***The Master DHT Reduction Flowchart***.
- We'll answer: what is DHT, why is DHT associated with hair thinning, and what are all the ways (good and bad) to fight hair loss by lowering DHT?

The DHT Master Flowchart

Reducing DHT For Hair Loss

If you're reading this, you might be suffering from hair loss. You might also want to learn *how* the hormone DHT is connected to hair loss, and all the ways you can *lower* DHT to help slow or stop hair thinning.

But you also probably don't have the time to dive into *all* the science behind each way to lower DHT. You probably just want someone tell you, "Hey, here's how you can lower DHT. Here are the *safe* ways to do it. And here are the ways you should avoid."

That's what this chapter is for. We're *not* going to get how DHT is linked to hair loss, or the science behind each DHT-reducing mechanism (but we will in later chapters). Instead, we're just going to highlight all the major ways you can reduce DHT levels to fight hair loss, and which mechanisms are good (and bad) targets.

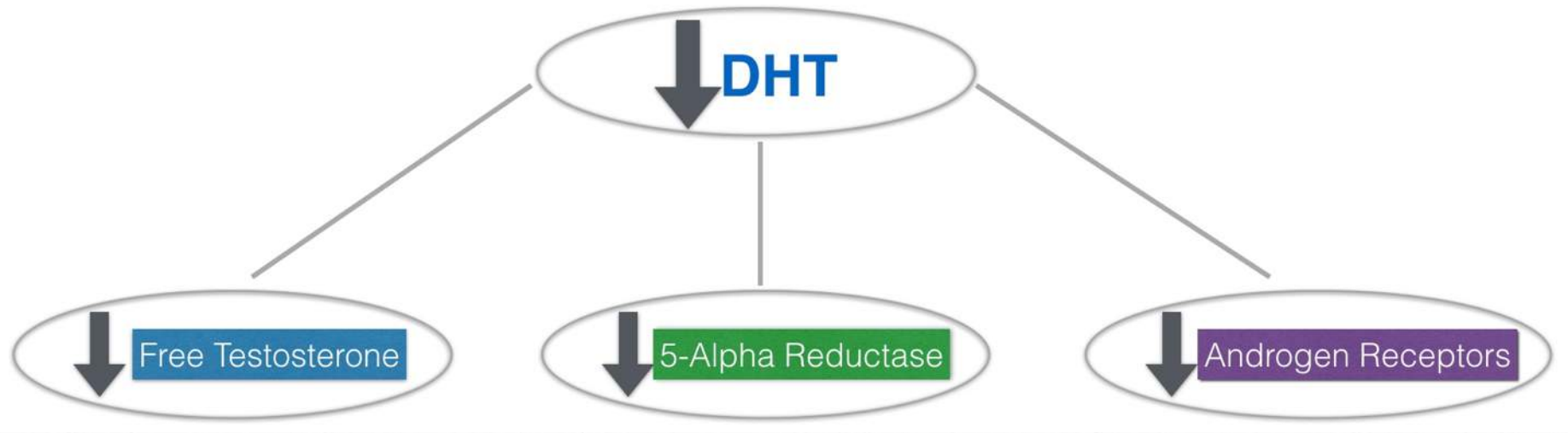
Now let's get into it. Again - this is a high-level overview. For better clarity, please read the later chapters.

Lowering DHT: The Four Major Levers

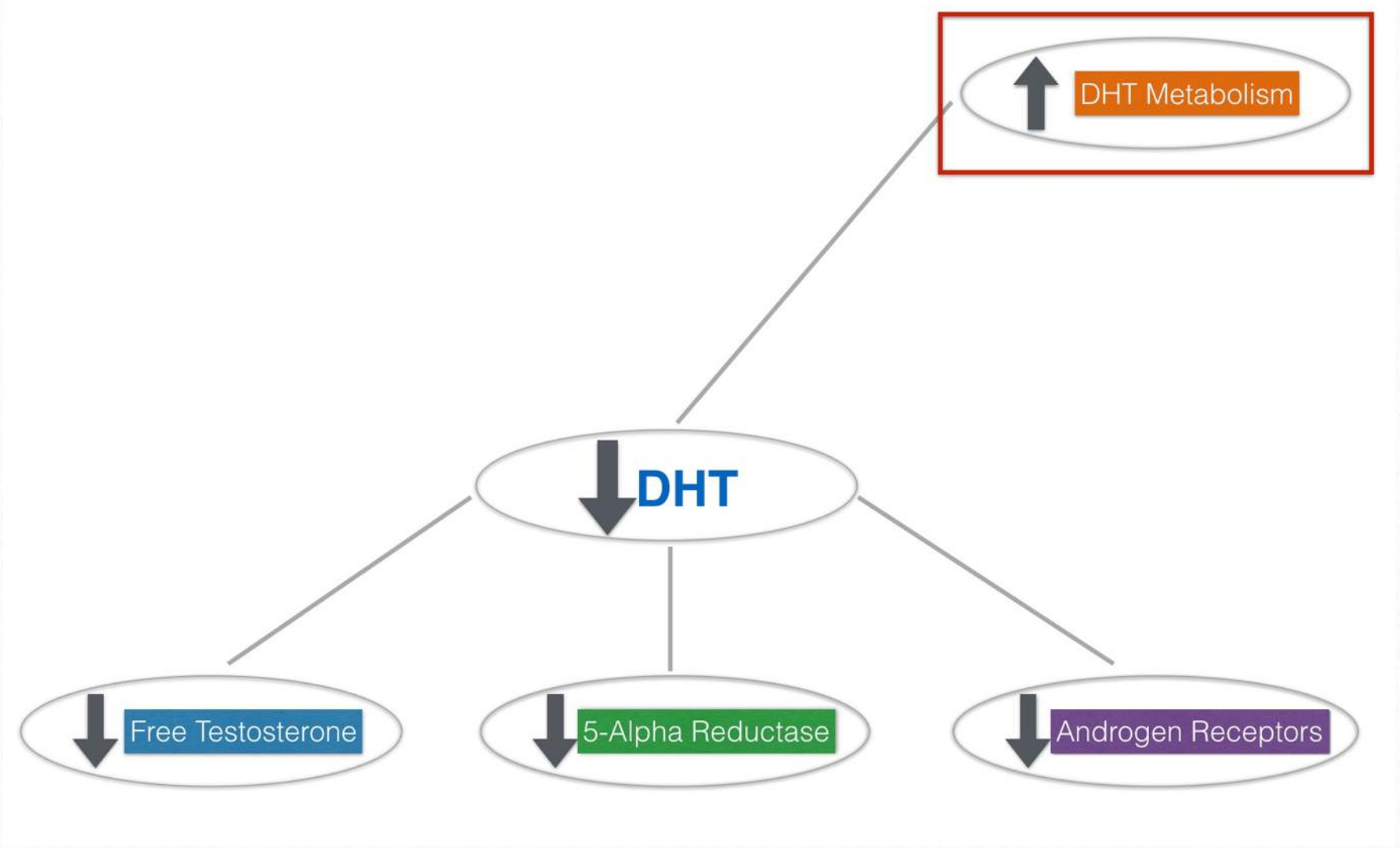
DHT is a hormone made from testosterone. And in order to make DHT, our bodies need three things:

1. **Free testosterone** (unbound testosterone)
2. **5-Alpha Reductase** (the enzyme required to convert unbound testosterone into DHT)
3. **Androgen Receptors** (the place where DHT binds to a cell, so that it can influence cell function)

This gives us **three** angles of attack to lower DHT levels for the purpose of fighting hair loss. We can reduce free testosterone, inhibit 5-alpha reductase, or decrease androgen receptors.



And on top of that, we can *also* reduce DHT by **increasing DHT metabolism** - the rate at which DHT is metabolized into other hormones and byproducts. In doing so, we decrease DHT's effects on a tissue.

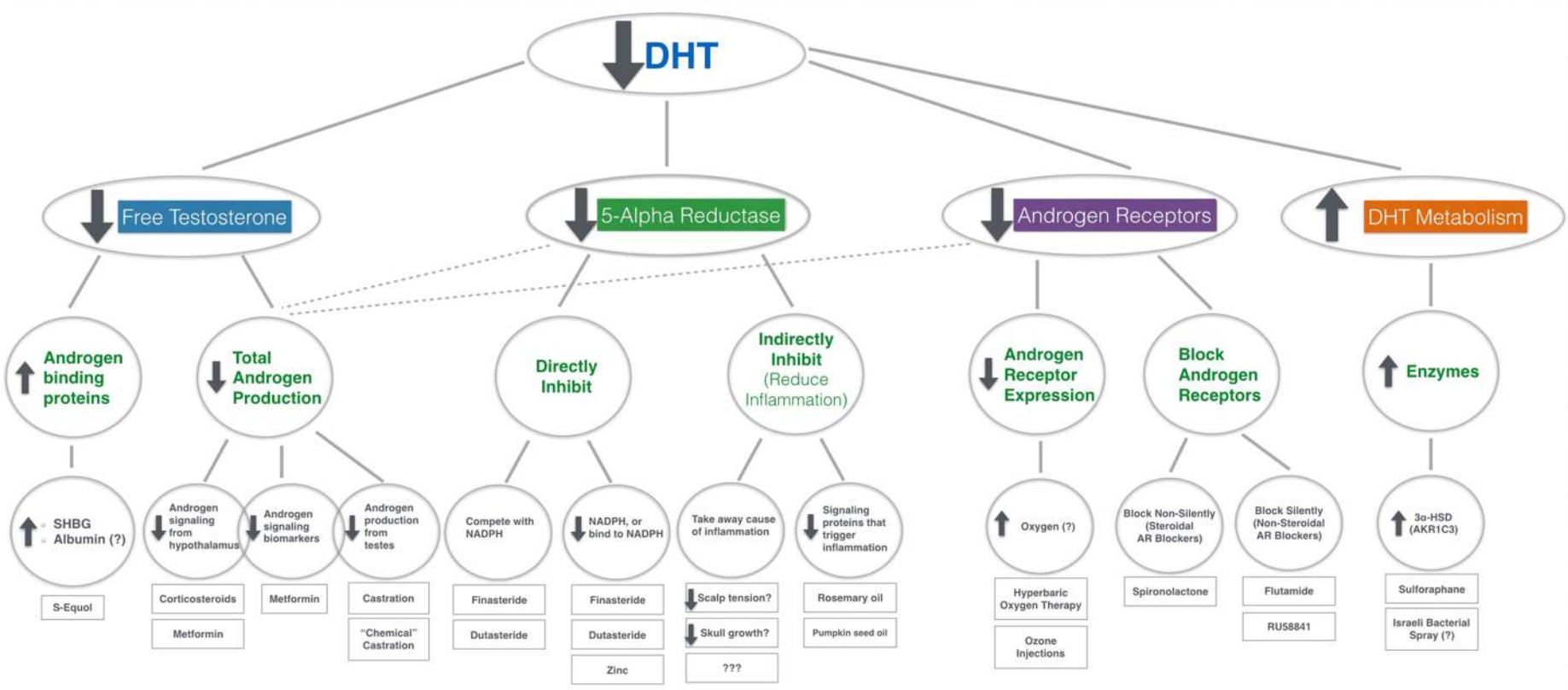


This gives us **four** levers to "attack" DHT:

1. Reducing Free Testosterone
2. Inhibiting 5-Alpha Reductase
3. Decreasing Androgen Receptors
4. Increasing DHT Metabolism

So how can we go about targeting these levers to lower DHT? There are *hundreds* of ways. I've summarized all the major ones inside this **DHT Reduction Master Flowchart**.

The DHT Reduction Master Flowchart



Yes, it's tough to read. So click here to enlarge!

Which DHT-Lowering Mechanisms Are The Best For Fighting Hair Loss?

It all depends on what matters to you. Here's the problem: DHT is associated with pattern hair loss, but it's also associated with male sexual function. As a result, most hair loss sufferers feel trapped between two terrible choices: do they take drugs and supplements that *reduce* DHT (and slow or stop hair loss), but at the risk of developing sexual side effects? Or do they avoid those drugs and supplements all together, at the consequence of losing more hair?

In my opinion, if you're going to reduce DHT levels to fight hair loss, safety matters most. There's no point in having hair if you can't perform sexually. And there's no point in having hair at the cost of your mental health.

So with that in mind, I have a few DHT-reducing mechanisms and treatments that I would absolutely avoid:

DHT-Reducing Mechanisms To AVOID:

1. Reducing total **androgen production**. Evidence here.

2. **Steroidal** 5-alpha reductase inhibitors. Evidence here and here.

3. **Steroidal** androgen receptor blockers. Evidence here.

And what about the DHT-reducing mechanisms that aren't so dangerous?

(Relatively) Safe DHT-Reducing Mechanisms:

1. Increasing **androgen-binding proteins**. Evidence here.

2. **Non-steroidal** 5-alpha reductase inhibitors. Evidence here.

3. Decreasing androgen receptor expression via **oxygen** therapies. Evidence here.

4. Increasing DHT **metabolism**. Evidence here.

What You Need To Know About DHT

Above all, remember this: if we reduce DHT levels to zero - even through herbal supplements - we're bound to develop sexual side effects. So reducing DHT to fight hair loss is a careful balancing act.

And remember this too: even if we reduce DHT levels to zero, we won't regrow *all* of the hair we've already lost. Why? Because DHT isn't necessarily the *cause* of hair loss... but DHT *does* play a causal role in the development of the two conditions that likely do cause hair loss: fibrosis and calcification.

That means if we want to make a full hair recovery, we need to stop focusing on DHT, and start focusing on reversing calcification and fibrosis. More evidence on that here.

And with that, let’s get into the science behind our **Master Flowchart**. We’ll start by breaking down each lever, diving into all the supporting mechanisms, and then uncovering a few foods, drugs, supplements, and topicals that influence them.

2

Attack DHT: Lower Free Testosterone

Summary

- What is DHT? How is it connected to hair loss?
- A deep-dive into lowering DHT by reducing **free testosterone**.

Lowering Free Testosterone

Reducing DHT For Hair Loss

We're going to dive into all of the best (and worst) ways we can lower DHT in hopes of slowing, stopping, or reversing pattern hair loss.

This chapter starts with the DHT-hair loss connection. By the end of these chapters, we'll uncover…

- The four major angles of attack against DHT – free testosterone, 5-alpha reductase, androgen receptors, and DHT metabolism
- The mechanisms behind each angle – and where things like bacteria, inflammation, and oxygen come into play
- The drugs, foods, and supplements targeting *each* mechanism – and which to AVOID
- The truth: Should we actually target DHT to reverse hair thinning? Maybe *not*

The objective: to create a **Master DHT Reduction Flowchart**. This is a systematic, scientific overview of nearly all the conventional (and unconventional) ways to reduce DHT. Some mechanisms *might* help reduce hair loss… most *won't*. But by the end, you'll have a concrete understanding of all DHT-reducing possibilities.

This way, the next time you read an article about a certain "DHT blocker" or "DHT reducer" – you'll instantly understand how it works, if it's effective against hair loss, and what the dangers are (or aren't) of trying it.

This series is educational. I do not endorse any specific mechanism as the "best" method (as you'll see – especially in this article – some of these mechanisms are downright horrifying). And after all, it's still unclear if reducing DHT is the best way to go about reversing hair loss.

So let's get started! Our focus for this article: reducing DHT by **reducing free testosterone** (more on this soon).

The DHT-Hair Loss Connection

Why Focus On DHT For Hair Loss?

Since the discovery of **testosterone** in 1935, researchers have believed that androgens (like testosterone or DHT) play some sort of role in pattern hair loss. Their rationale? Men bald more often than women, and coincidentally, men have much *higher* androgen levels.

It didn't take long for these beliefs to be confirmed. First, there was an observational study on men castrated *before* puberty. The findings: if a man is castrated before puberty (ie: *before* they start producing lots of androgens), androgen production remains suppressed throughout the remainder of his life – since the testes are responsible for producing 95% of a man's testosterone. And interestingly, men castrated *before* puberty never go bald later on — possibly a result of permanently suppressed androgen production.

It was an interesting observation… But the hair loss story was still incomplete. Why? Because testosterone isn't the *only* male androgen. There are other hormones made from testosterone that might be more at fault for hair loss. And if researchers wanted to create a viable treatment for hair loss, they'd need to get more specific and uncover the exact hormone causing the problem.

Then came an observational study on men with a rare genetic mutation: a type II 5-alpha reductase deficiency. This is the enzyme our bodies use to turn unbound testosterone into DHT in our scalps and prostate glands. The study's findings: men with this deficiency suffered from poor genital development and no body hair… but they also *never* went bald later in life.

This narrowed the scope: maybe it wasn't *testosterone* that caused hair loss… but rather DHT.

Many years later, researchers confirmed their suspicions after a breakthrough study confirmed that the hormone DHT is *elevated* in balding scalp regions – but *not* in non-balding scalp regions.

The key takeaway? It's likely that DHT plays some sort of *causal* role in pattern hair loss. And if we want to reduce hair loss (or even reverse it), maybe we should try to *reduce* our DHT levels.

This was the basis for hair loss drugs like **Propecia** (finasteride) and **Avodart** (dutasteride). These drugs reduce DHT, and unsurprisingly, they're clinically proven to help slow, stop, or even partially reverse pattern hair loss and hair thinning.

And this is why almost *everyone* focuses on reducing DHT to fight hair loss. And despite evidence that attacking DHT might not be the most effective treatment for hair loss, the concept remains cemented into doctors, researchers, and even hair loss sufferers.

How Is DHT Made?

There are many conversion pathways to making DHT. But when we boil it down, all (or nearly all) DHT is made from the hormone testosterone. And for the majority of DHT creation, our bodies *need* these three things:

1. **Free testosterone**. Testosterone comes in two varieties – bound and unbound. And in order for testosterone to convert into other hormones, it needs to be **unbound** (free) so that it can connect with other proteins or enzymes that change its structure.
2. **5-Alpha Reductase**. This is the **enzyme** our bodies use to convert free testosterone into DHT. When free testosterone comes into contact with the enzyme 5-alpha reductase, that enzyme converts the testosterone into DHT. Without the 5-alpha reductase, DHT can't form.
3. **Androgen Receptors**. In our cells, androgen receptors are the **landing pads** for androgens (like DHT). After free testosterone interacts with 5-alpha reductase and becomes DHT, that DHT needs to attach to a cell's androgen receptor in order to exert any effect on the tissue. Without androgen receptors, DHT has no home and can't exert its effects on cells.

If we had to break this down into a crude formula:

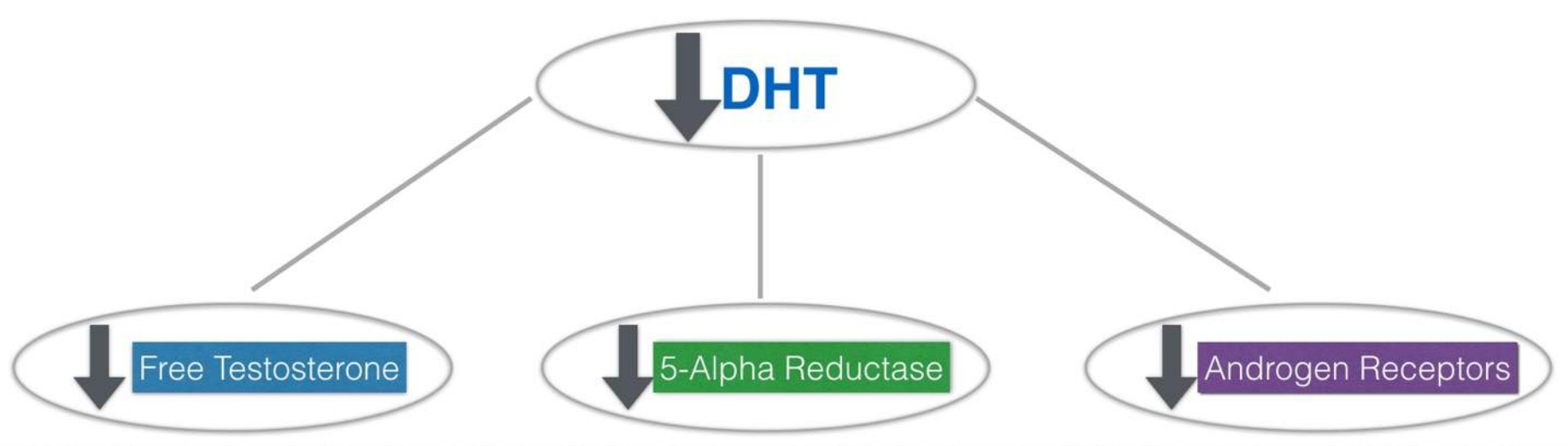
DHT = Free Testosterone + 5-Alpha Reductase + Androgen Receptors

Reducing DHT To Fight Hair Loss: Three Angles Of Attack

You probably picked up on this, but we just laid down three angles of attack *against* DHT:

1. Decreasing free testosterone
2. Decreasing 5-alpha reductase
3. Decreasing androgen receptors

Why? Because without free testosterone or 5-alpha reductase – DHT can't form. And without androgen receptors – DHT can't exert any effect on a tissue (like, for example, hair loss).

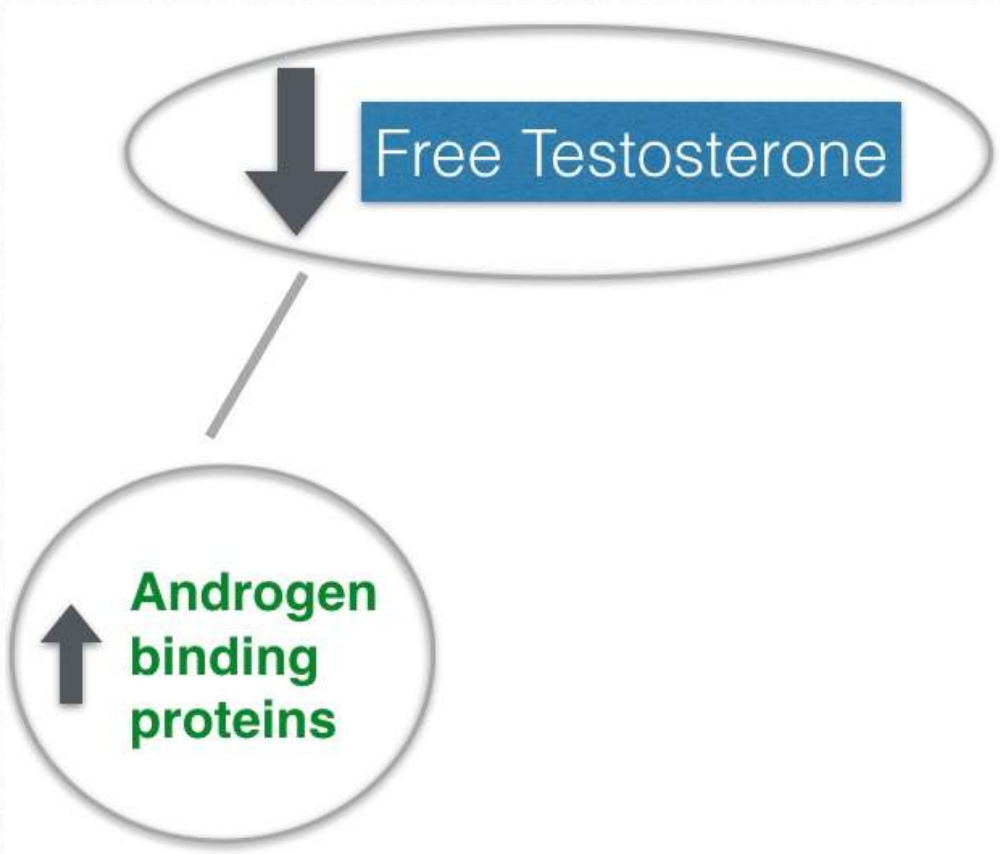


So let's dive into each angle of attack. This article *only* covers **free testosterone**. The next two will cover 5-alpha reductase, androgen receptors, and a lesser-known DHT reducing mechanism that very people ever consider.

DHT Attack Angle #1: Reduce Free Testosterone

Of all the ways to reduce free testosterone, there appear to be two *major* ones relevant to pattern hair loss.

The first: ***increasing*** **testosterone-binding proteins**.



1. Reduce DHT By *Increasing* Testosterone-Binding Proteins

Remember how testosterone must be *unbound* (free) in order to convert into DHT? Well, if testosterone is *bound*, it can't make that conversion. That means if we bind *more* free testosterone to certain proteins and enzymes, we can reduce the chances of free testosterone binding to the enzyme 5-alpha reductase and then becoming DHT.

Enter **sex hormone binding globulin** – a protein which *binds* to free testosterone and carries that bound testosterone throughout our blood. The benefit of this binding: this free testosterone is no longer free. And while that testosterone is bound, it cannot convert into DHT.

The Sex Hormone Binding Globulin-Hair Loss Connection

The more sex hormone binding globulin (SHBG) – the more SHBG binds to free testosterone, and the *less* free testosterone is available to convert into DHT.

It's unsurprising that low levels of SHBG are seen in young women with diffuse hair thinning, or that lower levels of sex hormone binding globulin are observed in completely bald men.

The takeaway: maybe by *increasing* SHBG, we can decrease free testosterone, maybe decrease DHT levels, and maybe even improve our pattern hair loss.

How to Increase Sex Hormone Binding Globulin (SHBG)

There are countless foods, supplements, and drugs that help increase SHBG (and decrease free testosterone). We're not going to cover all of them. But we *are* going to cover one of particular interest – a supplement known as **S-Equol**.

S-Equol is bacterially derived from daidzein, an **isoflavone** abundant in **soy foods**.

Isoflavones may increase the production of SHBG (sex hormone-binding globulin) in the liver and bind to biologically active testosterone. This results in the lowering of free testosterone.

The less testosterone in scalp tissue, the less likely it will be converted into DHT – *theoretically* reducing the risk of pattern hair loss. In fact, this has been validated.

One study demonstrated that short-term administration of soy isoflavones stimulated the production of serum equol and decreased the serum DHT (DHT in the blood).

But do soy isoflavones *also* decrease DHT in scalp tissues? Unfortunately, we don't know. There haven't yet been any studies to confirm this. And just because S-Equol reduces serum DHT doesn't mean we can say it *also* reduces scalp tissue DHT. And when it comes to fighting pattern hair loss, scalp tissue DHT is what really matters.

Can Other Proteins Bind To Testosterone And Decrease DHT?

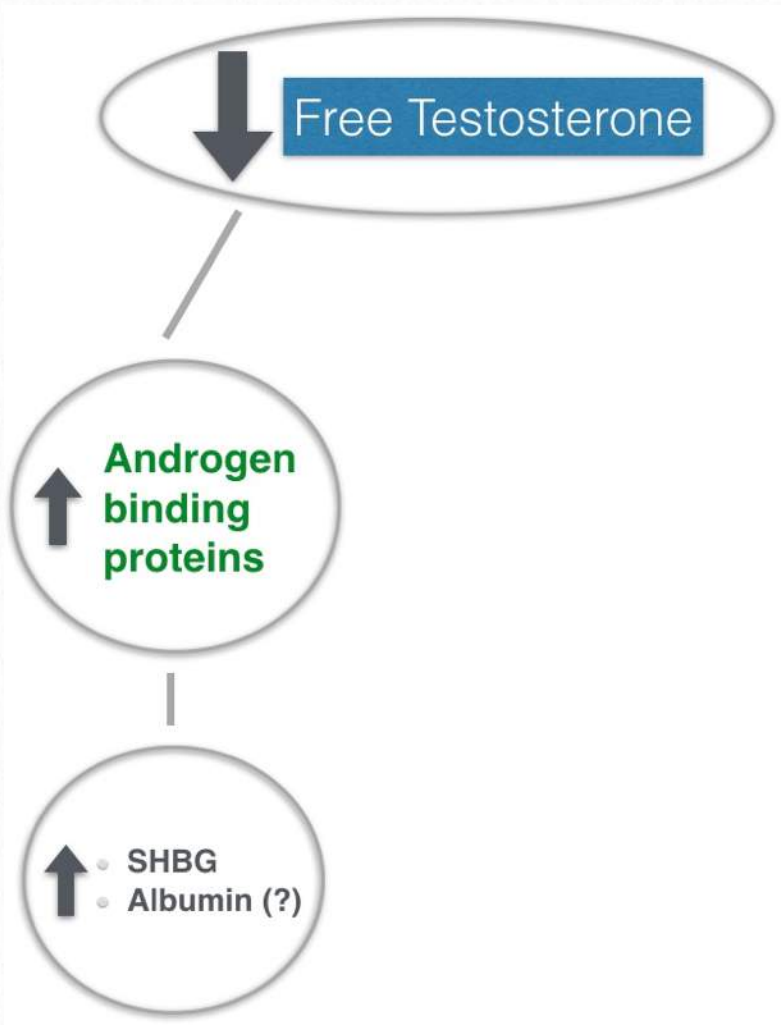
Maybe.

There are many large proteins in our blood that bind to hormones. **Albumin** – for example – is the largest protein in our blood, and is similar to SHBG in that it is made by the liver. It's important to consider if albumin might be more effective than SHBG at reducing DHT, *or* how substances like S-Equol affect albumin levels. Until more research is done, we just don't know.

Should We Take S-Equol To Reduce DHT And Fight Hair Loss?

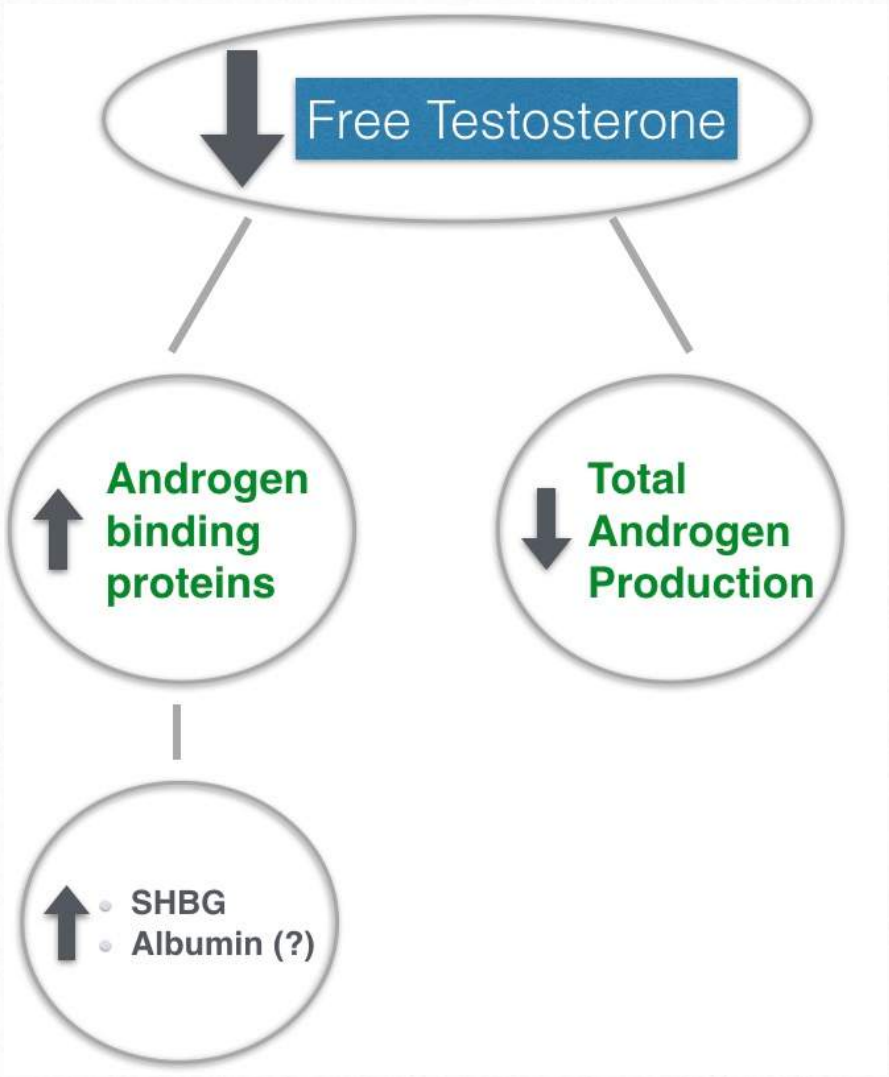
Until S-Equol is studied extensively for its effects on 1) scalp tissue DHT, and 2) pattern hair loss – we won't know if it's a viable treatment for hair loss sufferers. But within recommended dosages, it seems to be relatively safe and devoid of sexual side effects. So it probably won't hurt!

Here's a summary so far:



This is the first major way of *reducing* free testosterone (and thereby DHT). There's one more, and this one comes with *much* higher risk: ***suppressing*** **total androgen production**.

Please be warned: the following is educational. I don't endorse *any* of what's about to come.



2. Reduce Free Testosterone By Decreasing Total Androgen Production

Our brain – or specifically our hypothalamus – determines how much testosterone our bodies should produce. In fact, our hypothalamus sends this message to our testes – which produce 95% of testosterone for men. Together with this messaging, the testes then synthesize testosterone from cholesterol and send it out through our bloodstream. It's here that our testosterone then binds to proteins and enzymes – converting into different androgens and performing hundreds of bodily functions.

You might've already guessed it, but if we want to reduce DHT by reducing our body's *production* of androgens, we just laid out three more levers:

1. Reduce androgen signaling needs from the hypothalamus
2. Reduce cholesterol (and other testosterone production-signaling biomarkers)

3. Reduce our testes' ability to produce androgens

Let's take these one-by-one. And please, don't try *any* of these. Seriously. It's just a bad idea.

1. Decrease Androgen Signaling From The Hypothalamus

Certain steroids and drugs can *reduce* our body's desire (or ability) to produce testosterone. For example, steroids known as corticosteroids – through unknown mechanisms – can reduce the amount of testosterone our bodies decide to produce. This is likely due to these drugs muting androgen signaling needs from our hypothalamus.

2. Decrease Cholesterol (And Other Testosterone Signaling Biomarkers)

Unsurprisingly, cholesterol-lowering and insulin-lowering drugs (like Metformin) have also been shown to reduce total testosterone production. While the mechanisms aren't entirely clear, this may be due to brain signaling response changes. For instance, the hypothalamus might tell the testes to produce *less* testosterone if it senses we have lower levels of circulating cholesterol and insulin. And the less free testosterone we produce, the less there is to convert into DHT – the alleged "hair loss" hormone.

Note: these drugs and steroids are merely examples, and not meant to be misconstrued as the most potent free testosterone reducers, or the only free testosterone reducers.

The Problem With Suppressing Total Androgen Production? Shrunken Testicles

Unfortunately, when we mute testosterone production, we pay a steep price. When we manipulate our brain's signaling so that our hypothalamus tells our testes to produce *less* testosterone… our testicles can actually start *shrinking.*

This is called **hypogonadism** – a condition that's twice as prevalent in men taking statin (cholesterol-lowering) drugs. And if we suppress testosterone production for too long, our testicles can shrink to a size of complete dysfunction.

In a sense, this is "chemical castration" – taking testosterone-suppressing drugs at the consequence of rendering our testes lifeless…

…Which brings us to the extreme end up the spectrum: *cutting off* the ability for our testes to produce 95% of our body's testosterone.

3. Reduce Testes' Ability To Produce Androgens

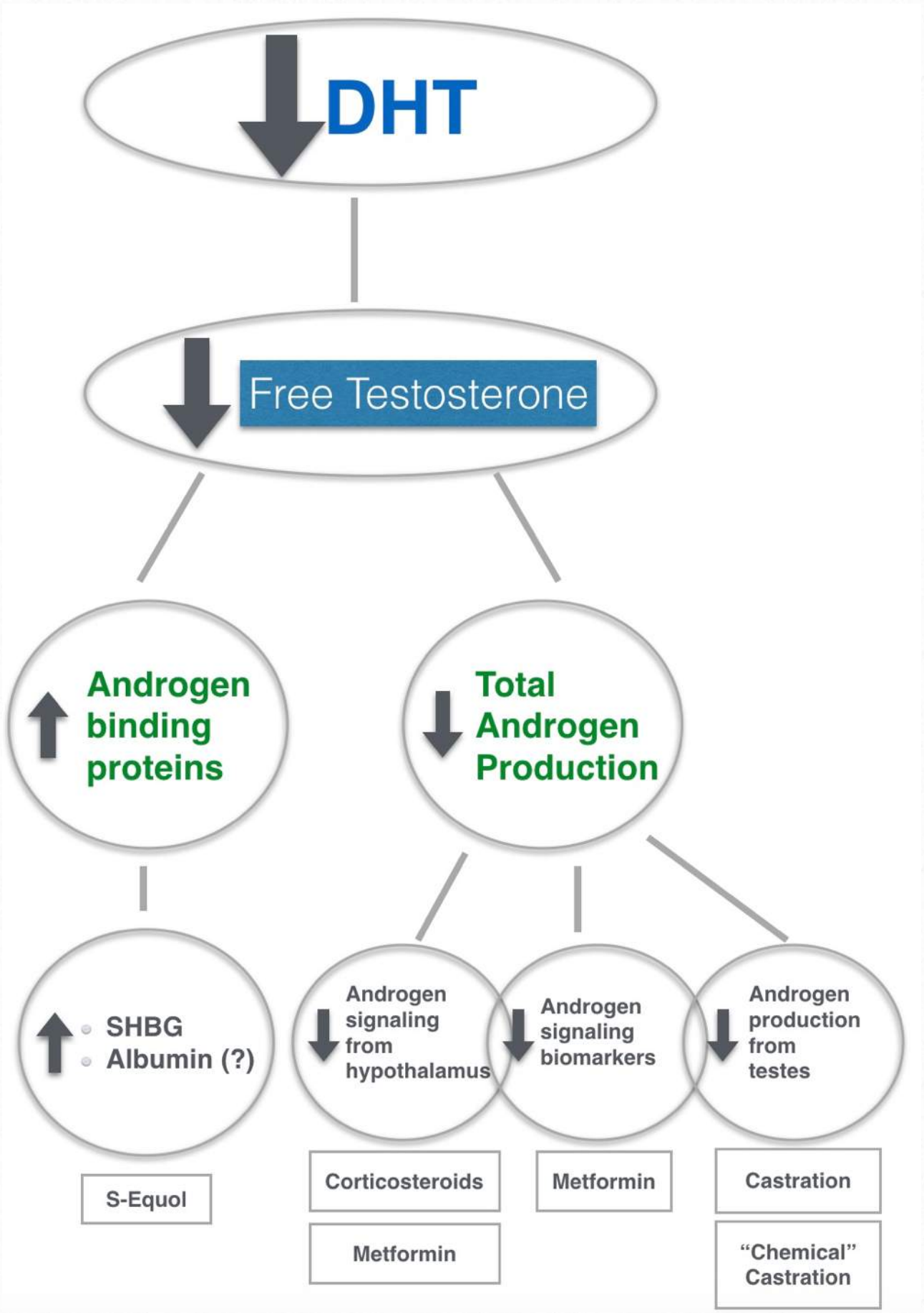
In some forms, this is just the end-result of long-term testosterone-suppressing drug use. But at its very extreme, this is removal of the testicles.

Yes, I'm talking about **castration**. Yes, this is the ultimate DHT suppressor. And yes, this a terrible idea. If you're looking to live a life with a near-absent libido, poor-to-no erection quality, depression, and possibly even a higher susceptibility to certain diseases and cancers – this is what life is like for some male castrates.

I don't know about you, but I'd choose baldness over castration any day – chemically-induced or otherwise. So please, *don't* get any ideas.

Summary So Far

We've just completed the *first* pillar of our flowchart… reducing DHT by reducing **free testosterone**.



The key takeaway: fighting DHT by reducing free testosterone is a bad idea… unless you're decreasing DHT by *increasing* androgen-binding proteins like sex hormone binding globulin or albumin.

Above all: stay away from drugs that suppress total androgen production. While it's not covered in this article, even treatments like **testosterone replacement therapy** can, over time, decrease your body's ability to produce endogenous testosterone – or in other words, testosterone from the testes. The end-result? Hypogonadism. Which is ironic when you consider that both *suppressing* testosterone production and *injecting* testosterone outside the body can both result in shrunken testicles.

The good news: the next article uncovers *slightly* better ways of going about reducing DHT for pattern hair loss. The third article dives into some very effective topicals. And the *final* article uncovers DHT-fighting breakthroughs almost no one is talking about.

What's Next…

In the next chapter, we'll uncover DHT's second "angle" of attack – reducing DHT by inhibiting the enzyme **5-alpha reductase**. And if you think Propecia, Avodart, or even "natural" supplements like saw palmetto extract or pumpkin seed oil are the *only* ways to reduce this enzyme… think again.

2

Attack DHT: Inhibit 5-Alpha Reductase

Summary

- A deep-dive into lowering DHT by inhibiting the enzyme **5-alpha reductase**.

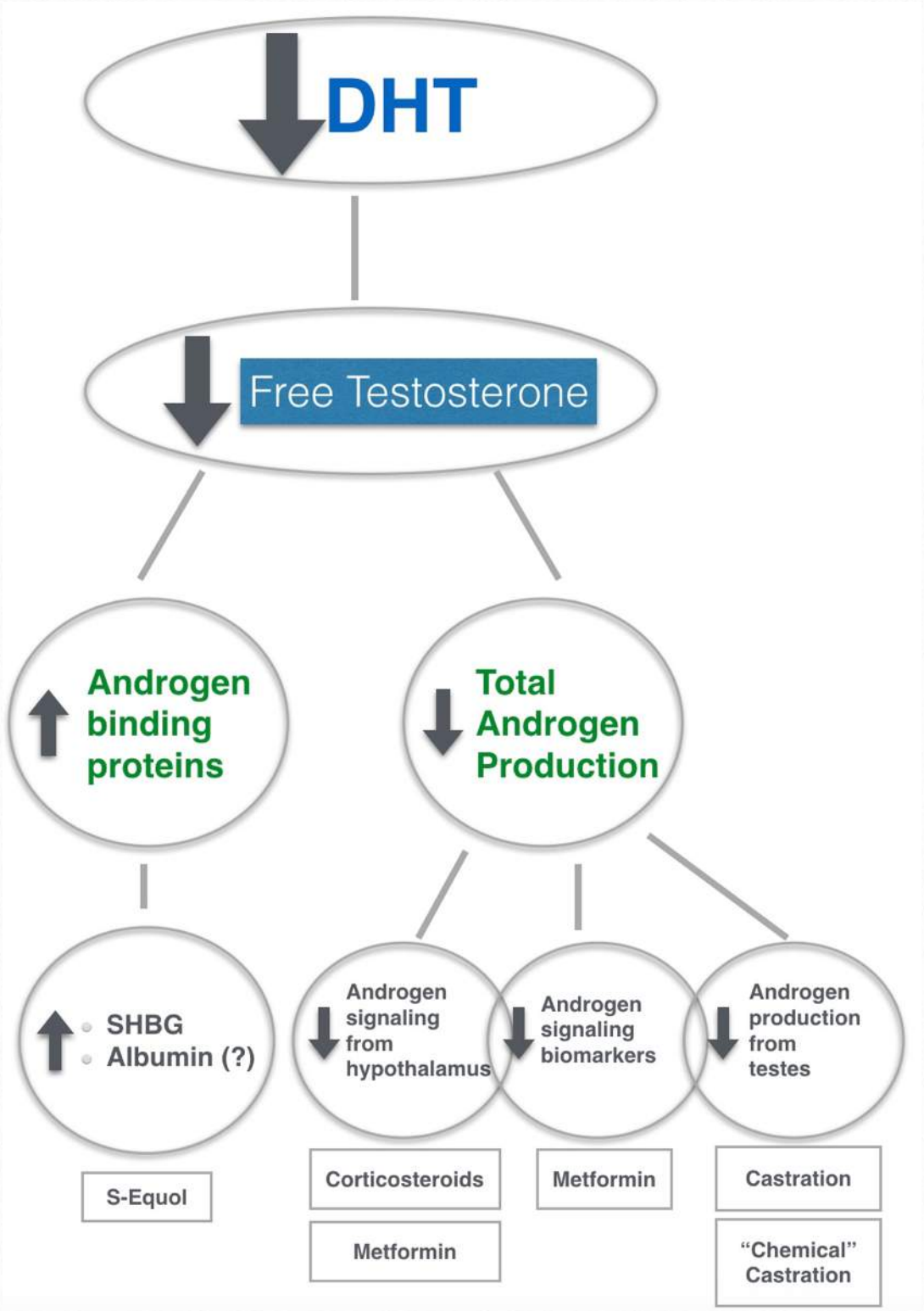
Inhibiting 5-Alpha Reductase

Summary So Far – Decreasing DHT By Reducing Free Testosterone

In the last chapter, we uncovered what DHT is, how it's made, the DHT-hair loss connection, and the **four** major levers to reduce DHT levels in hopes of stopping hair loss:

1. Decrease free testosterone
2. Inhibit 5-alpha reductase
3. Decrease androgen receptors
4. …and one more we haven't yet revealed

Then we dove into all the ways we can decrease DHT by using that *first* lever: reducing **free testosterone**. Here's a summary of the mechanisms (but not all the drugs, foods, supplements, and treatments that target these mechanisms):



Unfortunately, most of these approaches are bad ideas. For instance – *yes*, we can theoretically plummet DHT production via castration. And yes, castration has been shown to significantly slow or stop pattern hair loss. But the consequences of castration *far* outweigh the pain of losing our hair!

In fact, when it comes to decreasing DHT by reducing free testosterone – the *only* mechanism here that might have any merit for hair loss sufferers is **increasing androgen-binding proteins** (like sex hormone binding globulin). This strategy appears devoid of major side effects and can even be achieved with supplements (like S-Equol).

So when it comes to reducing DHT by decreasing free testosterone, we don't have many viable options…

The good news? There are still **three** other levers of attack against DHT.

The one we'll cover inside this article: inhibiting the enzyme **5-alpha reductase**.

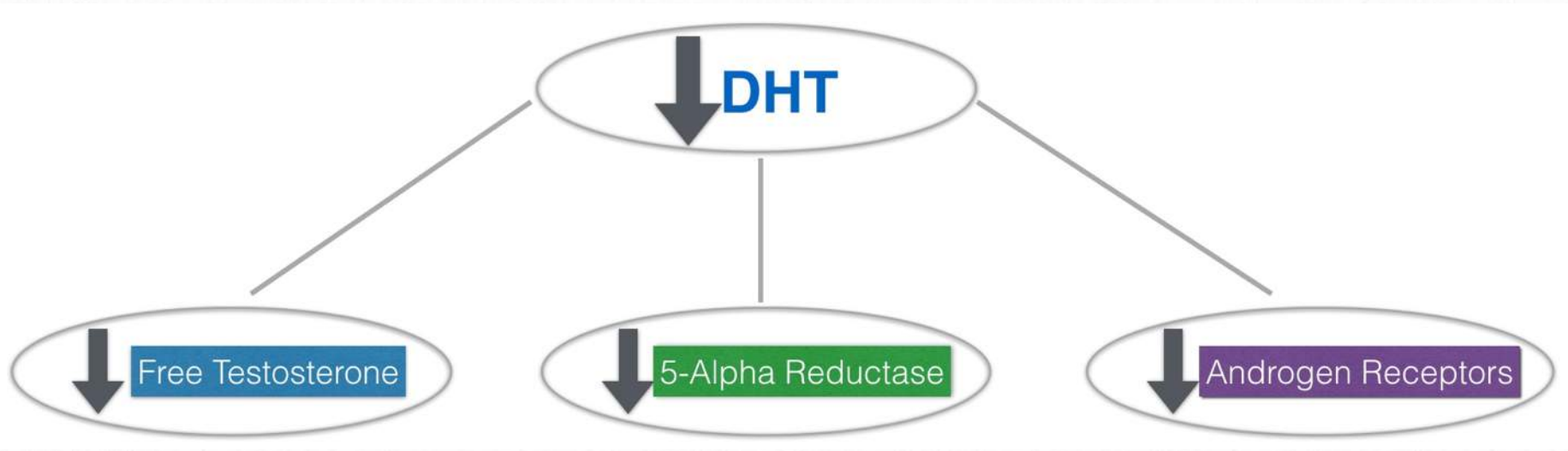
Part Two: Decreasing DHT By Inhibiting 5-Alpha Reductase

Review: Three Levers Of DHT Reduction

Remember: in order for dihydrotestosterone (DHT) to form, we need *all* of the following present:

1. Free testosterone
2. 5-alpha reductase
3. Androgen receptors

As a result, this gives us three major levers to reduce DHT levels: decrease 1) free testosterone, 2) 5-alpha reductase, and / or 3) androgen receptors.



We've already covered the major ways to reduce free testosterone (and thereby decrease DHT). Now it's time to move onto the enzyme **5-alpha reductase**.

What Is 5-Alpha Reductase?

5-alpha reductase is the enzyme our bodies use to convert free testosterone into DHT. And without the enzyme 5-alpha reductase, DHT cannot form.

There are many types of 5-alpha reductase, but when it comes to hair loss, the one that gets the most attention is **type II 5-alpha reductase**.

Type II 5-alpha reductase is the enzyme expressed in our scalp skin and prostate. Some men have a rare genetic mutation where their bodies can't produce *any* type II 5-alpha reductase. And interestingly enough, these men don't go bald.

The net: we need type II 5-alpha reductase to make DHT in our scalp skin. And that means if we can *reduce* the expression of type II 5-alpha reductase, we can also reduce our DHT levels (and possibly prevent or partially reverse pattern hair thinning).

Which brings us to our second angle of attack against DHT…

Angle Of Attack #2: Reducing 5-Alpha Reductase

There seems to be *at least* two major pathways to inhibiting (or reducing the presence of) this enzyme.

1. **Directly** (competitively inhibit 5-alpha reductase)
2. **Indirectly** (reduce inflammation)

Let's take these one-by-one.

#1: Direct 5-Alpha Reductase Inhibition

Competitive Inhibition

5-alpha reductase doesn't just arrive out of nowhere. In order for this enzyme to form and mediate the whole DHT conversion process, it needs the help of a coenzyme known as nicotinamide adenine dinucleotide phosphate… or in other words, **NADPH**.

5-alpha reductase needs NADPH to convert free testosterone into DHT. So an effective way to *stop* the formation of 5-alpha reductase (and reducing DHT) is to…

1. Compete with the coenzyme NADPH, or…
2. Block NADPH

These are two mechanisms of **direct** 5-alpha reductase inhibition – or for the lay person – reducing 5-alpha reductase by *stopping* it from forming. And while it's still up for debate, the hair loss drugs Finasteride and Dutasteride – two 5-alpha reductase inhibitors – appear to work in this way.

Mechanism 1: Compete With NADPH

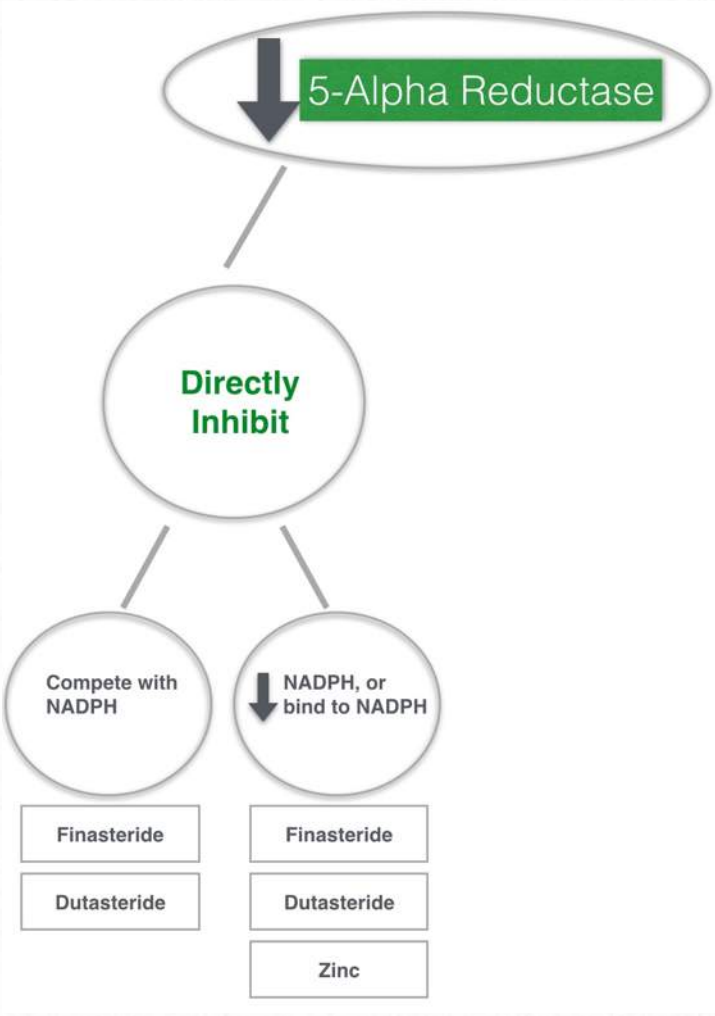
Some research shows that Finasteride *competes* with the coenzyme NADPH. Finasteride's molecules take the place of NADPH in a cell, and in NADPH's absence, 5-alpha reductase cannot form. The end-result? Less DHT. And another 5-alpha reductase inhibiting drug – Dutasteride – seems to do the same thing (using a slightly different molecule).

Mechanism 2: Block NADPH (Or Bind To NADPH And Change Its Structure)

There's also research showing that instead of competing with NADPH, Finasteride may instead *bind* to NADPH and change NADPH's structure into a *different* coenzyme – one that doesn't support the formation of 5-alpha reductase. The bottom line: free testosterone can no longer convert into DHT.

Interestingly, **zinc** may also reduce 5-alpha reductase and through a similar manner. Evidence suggests that zinc reduces NADPH production, thereby decreasing 5-alpha reductase activity. The less enzyme activity, the less DHT.

And that's a brief overview of how to reduce DHT levels by *directly* inhibiting the enzyme 5-alpha reductase.



Finasteride & Dutasteride For Hair Loss: Should You Use Them?

Finasteride and Dutasteride are *very* effective 5-alpha reductase inhibitors – with research showing these drugs can reduce DHT levels by up to 70-90% (depending on the dose). Finasteride and Dutasteride are also effective hair loss treatments – with most studies suggesting these drugs help slow, stop, or partially reverse pattern hair loss in the overwhelming majority of its users.

Unfortunately, these results can come attached to a long list of unintended sexual side effects – like poor erection quality, low libido, problems ejaculating, or even impotence.

This shouldn't come as a surprise. Why? Theoretically, anything that does *too* good a job of reducing DHT can cause sexual side effects – because healthy DHT levels seem to be a requirement for healthy sexual function.

But there's another problem with Finasteride and Dutasteride that often goes undiscussed. These drugs remodel our prostates (and likely our scalp skin). They increase the number of androgen receptors present in prostate tissue. And this remodeling seems to persist – even after stopping treatment.

This creates a hidden cost of Finasteride and Dutasteride: *dependency*. If our prostates and scalp skin remodel with *more* androgen receptors while on Finasteride, then if we *stop* taking these drugs, we may run of the risk androgens (DHT) flooding our scalps and prostate… more so than if we *never* took these drugs in the first place. In fact, this may men when we get off Finasteride, we might actually *increase* our rate of hair loss – which would explain why users who quit either drug typically lose all the hair they'd maintained (or regrown) in less than a year.

With that said, many men take Propecia (Finasteride) or Avodart (Dutasteride) for hair loss and experience zero issues. But if you're considering either treatment, you need to be aware of the side effects – since in some cases, they are irreversible.

This is why so many researchers are searching for non-steroidal 5-alpha reductase inhibitors. It's because steroidal 5-alpha reductase inhibitors (the ones synthesized from progesterone – like Finasteride and Dustasteride) – tend to come attached to irreversible prostate remodeling, and a higher potential for sexual side effects.

Fortunately, for those trying to fight hair loss by reducing DHT, non-steroidal 5-alpha reductase inhibitors *do* exist. And so far, the evidence suggests these non-steroidal 5-alpha reductase inhibitors *don't* cause permanent sexual side effects. And what's even more interesting is that these non-steroidal 5-AR inhibitors might work through different pathways than Finasteride or Dutasteride to inhibiting 5-alpha reductase. Which brings us to our *second* main mechanism to inhibiting 5-AR…

#2: Indirect 5-Alpha Reductase Inhibition

Reduce Inflammation

Studies show there's an association with DHT and inflammation. The net: DHT might regulate the inflammatory process. And in some tissues, increased DHT might even be a *response* to increased inflammation.

Theoretically, if we can *reduce* inflammation, we might also reduce 5-alpha reductase activity (and thereby DHT levels).

Interestingly, reducing chronic inflammation may be an *indirect* way of reducing 5-alpha reductase. This is because reducing inflammation doesn't *directly* inhibit 5-alpha reductase, but rather, inhibits the inflammation that *signals* 5-alpha reductase to arrive in certain tissues (like our scalp skin and prostates).

There are *hundreds* of ways to reduce chronic inflammation. But they all boil down to two main methods: we can either 1) take away whatever's causing the inflammation in the first place, or 2) stop the signaling proteins that tell our bodies to send inflammatory cells to injury sites.

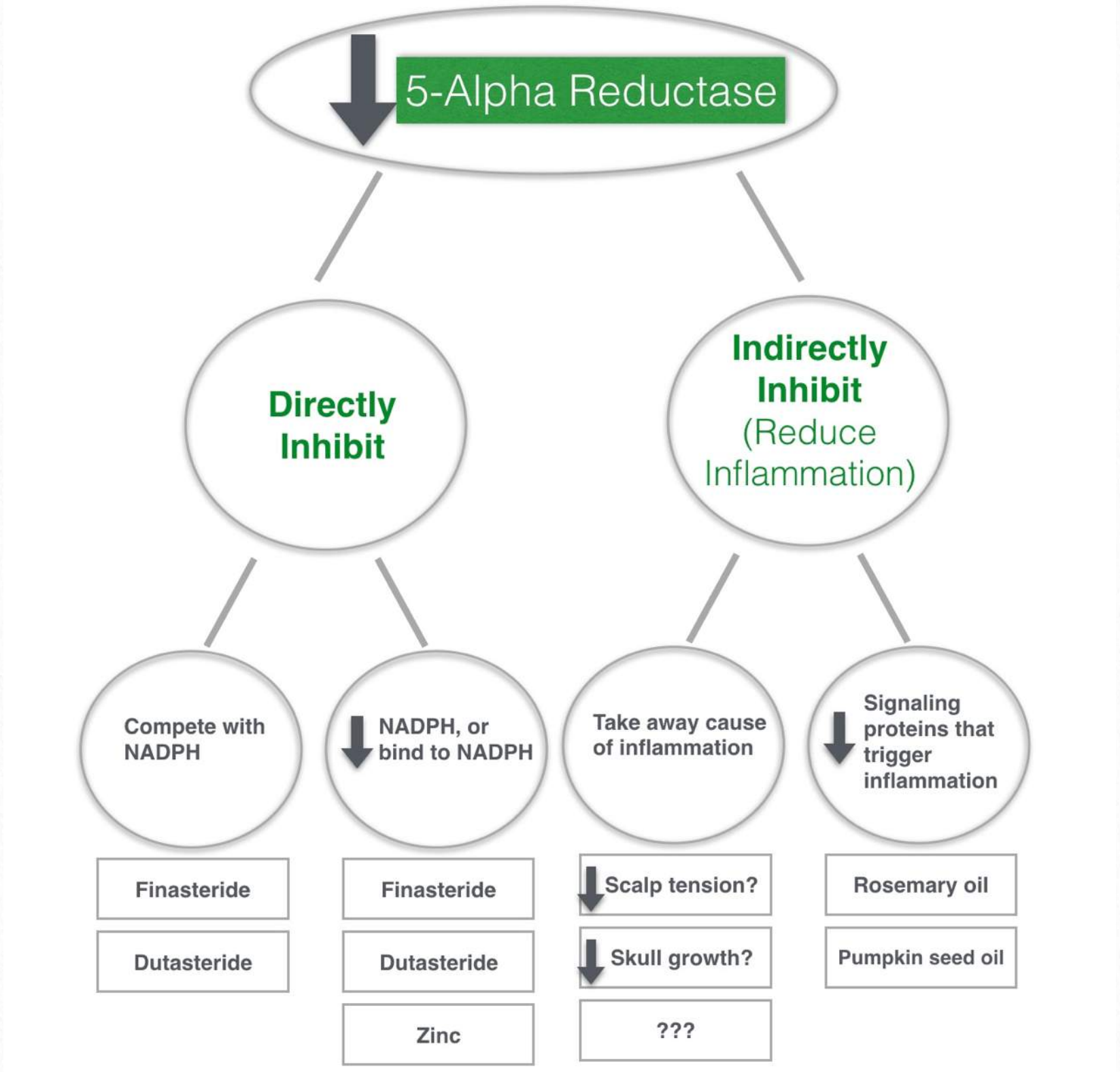
In the case of pattern hair loss, we don't really *know* what causes chronic inflammation in our scalps. It could be scalp muscular tension, protruded bone growth, skin tightening… the list goes on. But since we don't know the cause, we're more or less stuck with that second inflammation-reducing option: muting signaling proteins that channel inflammatory cells to injured tissues.

Fortunately, there are *hundreds* of substances that can do this. Covering each is out-of-scope for this article, so we'll instead highlight just two:

1. **#1: Pumpkin Seed Oil**
    1. Antioxidants: decrease oxidation, decrease transforming growth factor beta
    2. Linoleic acid: decreases COX-2 enzyme
2. **#2: Rosemary Oil**
    1. Polyphenols: decreases COX-2 enzyme
    2. Volatile oils: decreases COX-2 enzyme, interleukins, and tumor necrosis factor

Recap: Direct Vs. Indirect 5-Alpha Reductase Inhibition

We can decrease DHT by inhibiting 5-alpha reductase through two major pathways: **direct** versus **indirect** 5-AR inhibition. Direct 5-AR inhibition is how steroid-derived hair loss drugs like Finasteride and Dutasteride work. Unfortunately, while these drugs are effective at stopping hair loss, they potentially come attached to potentially irreversible side effects like prostate remodeling and decreased sexual function.



Conversely, we may be able to **indirectly** inhibit 5-alpha reductase by *reducing* inflammation in tissues. Inflammation and hair loss are closely linked, but since we don't yet know what *causes* the inflammation that triggers hair loss, we're more or less limited to reducing scalp inflammation by simply inhibiting the signaling proteins that send more inflammatory cells to those tissues. Rosemary oil and pumpkin seed oil have these anti-inflammatory properties. Unfortunately, they're less studied in terms of hair loss, and probably aren't as effective at stopping hair loss versus Finasteride (Propecia) or Dutasteride (Avodart).

Any Other Ways To Reduce 5-Alpha Reductase – Directly Or Indirectly?

Absolutely.

There's evidence that polyunsaturated fatty acids like **linoleic acid** may act *directly* and *indirectly* on 5-alpha reductase by 1) reducing inflammation, and 2) altering lipid bilayers in cell membranes to decrease 5-alpha reductase formation.

There's also evidence that vitamin B2- also known as **riboflavin** – may decrease 5-alpha reductase activity, though the mechanisms aren't completely understood.

Even the polyphenols inside **green tea** may inhibit 5-alpha reductase.

The pathways these substances take to reduce 5-alpha reductase are complex, and they're still being explored. As a result, I've omitted these from the flowchart until studies can confirm their exact mechanisms.

Finally – we can also reduce 5-alpha reductase activity by ***decreasing*** **total androgen production**. The less androgens our bodies produce, the less 5-alpha reductase is activated. This was covered in the first article about reducing free testosterone, and as a result, we won't cover it again here.

Summary Of Series (So Far)

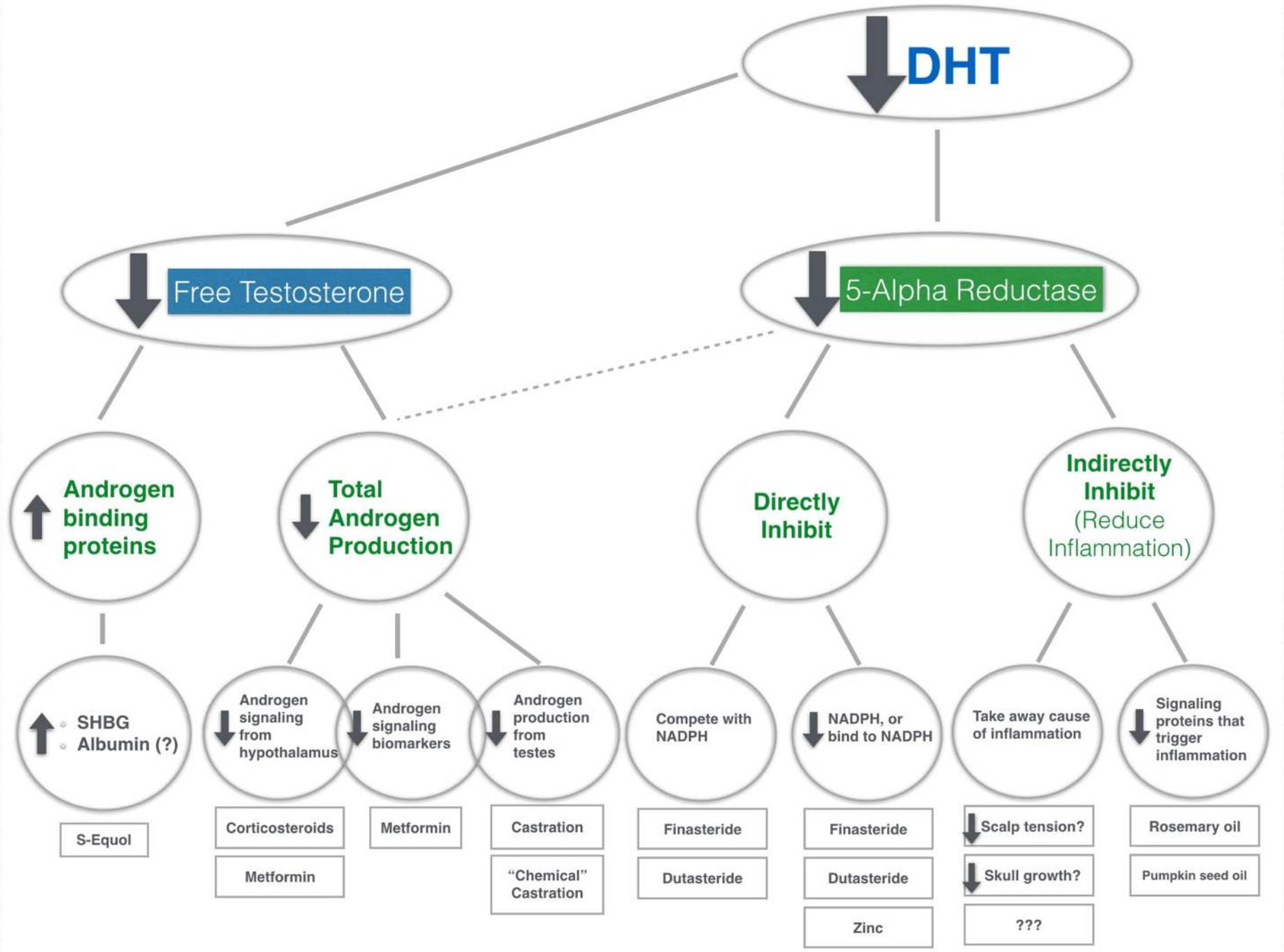
When it comes to fighting hair loss by reducing DHT, there are four main levers of attack:

1. Reducing free testosterone
2. Inhibiting 5-alpha reductase
3. Blocking androgen receptors
4. A mystery lever (we'll get to that soon)

In the last capter, we covered the major ways of reducing DHT by reducing free testosterone, and provided some examples of the drugs and supplements which achieve this (inadvertently or not).

In this chapter, we uncovered how we can reduce DHT by inhibiting 5-alpha reductase – and through a variety of mechanisms.

So let's combine what we know so far into one major flowchart. So far, we're 2/4's of the way to a complete **DHT Reduction Master Flowchart**:



Remember, the drugs and supplements listed above are just *examples*. These are by no means the most *effective* drugs and supplements within their respective categories, nor are they the *only* drugs or supplements that can achieve these effects. This flowchart is educational and *not* endorsing of any specific treatment.

What's To Come...

The third chapter of a ***Master Guide To The Mechanisms Behind DHT Reduction*** uncovers how to decrease DHT by decreasing **androgen receptors**. And this is where things like oxygen come into play... so read on!

2

Attack DHT: Decrease Androgen Receptors

Summary

- A deep-dive into lowering DHT by decreasing **androgen receptors**.

Decreasing Androgen Receptors

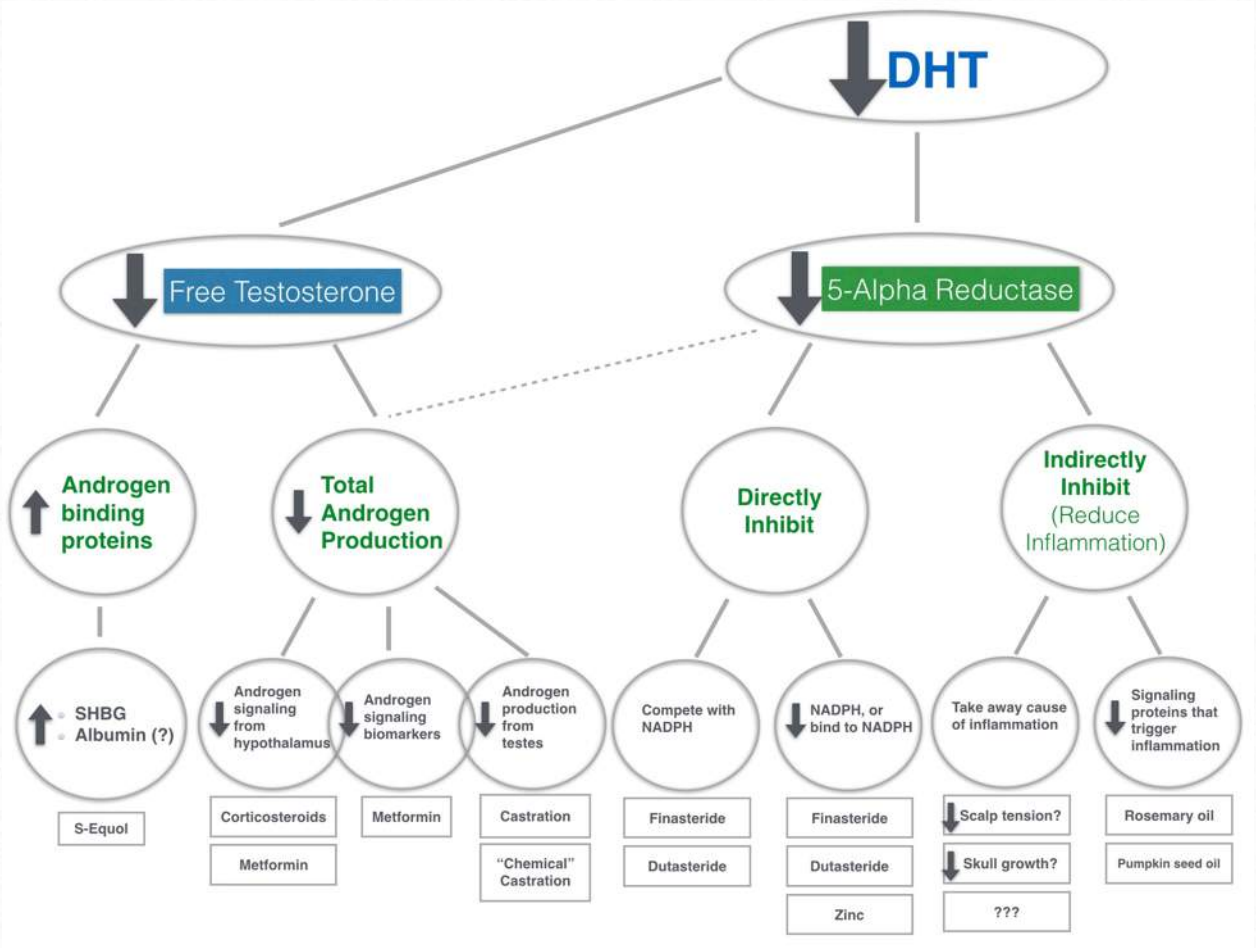
Summary So Far - Decreasing DHT With Free Testosterone, 5-Alpha Reductase

In the first chapter, we uncovered what DHT is, how it's made, the DHT-hair loss connection, and how we can reduce DHT (and maybe fight hair loss) by using **four** major levers.

1. Decrease free testosterone
2. Inhibit 5-alpha reductase
3. Decrease androgen receptors
4. …and one more we'll reveal in the next article

Then we dove into all the mechanisms by which we can decrease DHT by using the first and second lever: reducing **free testosterone** and inhibiting **5-alpha reductase**.

By the end of the [second article], we summarized the mechanisms (but not all the drugs, foods, supplements, and treatments) targeting those first two levers:



But we still have two DHT-fighting levers left!

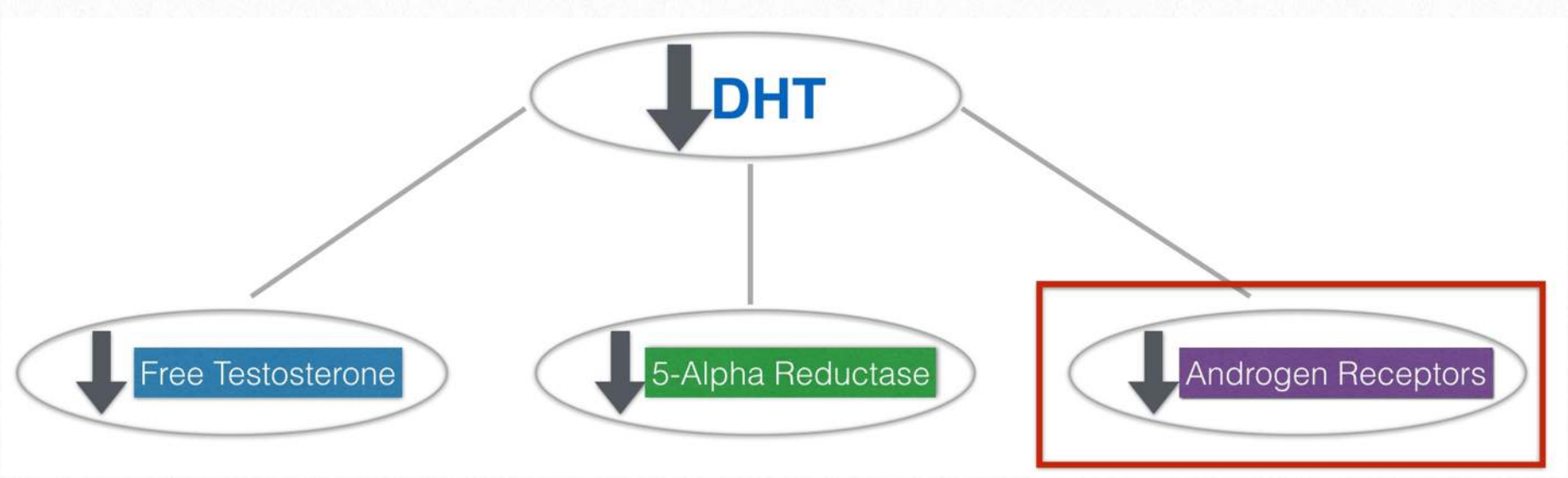
1. Decreasing **androgen receptors**, and…
2. A **mystery** lever (for the next and final article…)

This chapter is the third installment to our DHT-reducing mechanism series. We'll uncover the third major DHT-reducing pathway – and its known mechanisms – in hopes of reducing DHT to slow, stop, or reverse pattern hair loss.

It all builds into our ***Master Flowchart: A Guide To All Major DHT Reducing Mechanisms To Fight Against Hair Loss.***

What we're covering now: decreasing DHT by reducing **androgen receptors**. And the research here is pretty exciting (at least to me).

Reduce DHT: Decreasing Androgen Receptors



What Are Androgen Receptors?

Androgen receptors are the places inside a cell where androgens (testosterone, DHT, etc.) attach themselves. After an androgen attaches to an androgen receptor, these androgens can then influence a cell's function.

Remember: DHT forms when free testosterone interacts with the enzyme type II 5-alpha reductase and converts that free testosterone into DHT. Then that DHT *binds* to a cell's androgen receptor, where it influences that cell (and tissue). In the case of pattern hair loss, the kind of DHT people want to reduce is scalp tissue DHT.

Think of androgen receptors like a landing pad for DHT. Without an androgen receptor, DHT can't attach to the cell and influence its function. And in the case of pattern hair loss – without androgen receptors, DHT can't attach to scalp tissue DHT (the kind of DHT associates with hair thinning).

How Can We Decrease Androgen Receptors?

There are three main ways to decrease androgen receptors…

1. Decrease total androgen production
2. Decrease androgen receptor expression
3. Block androgen receptors

Let's dive into all three.

#1: Decreasing Total Androgen Production

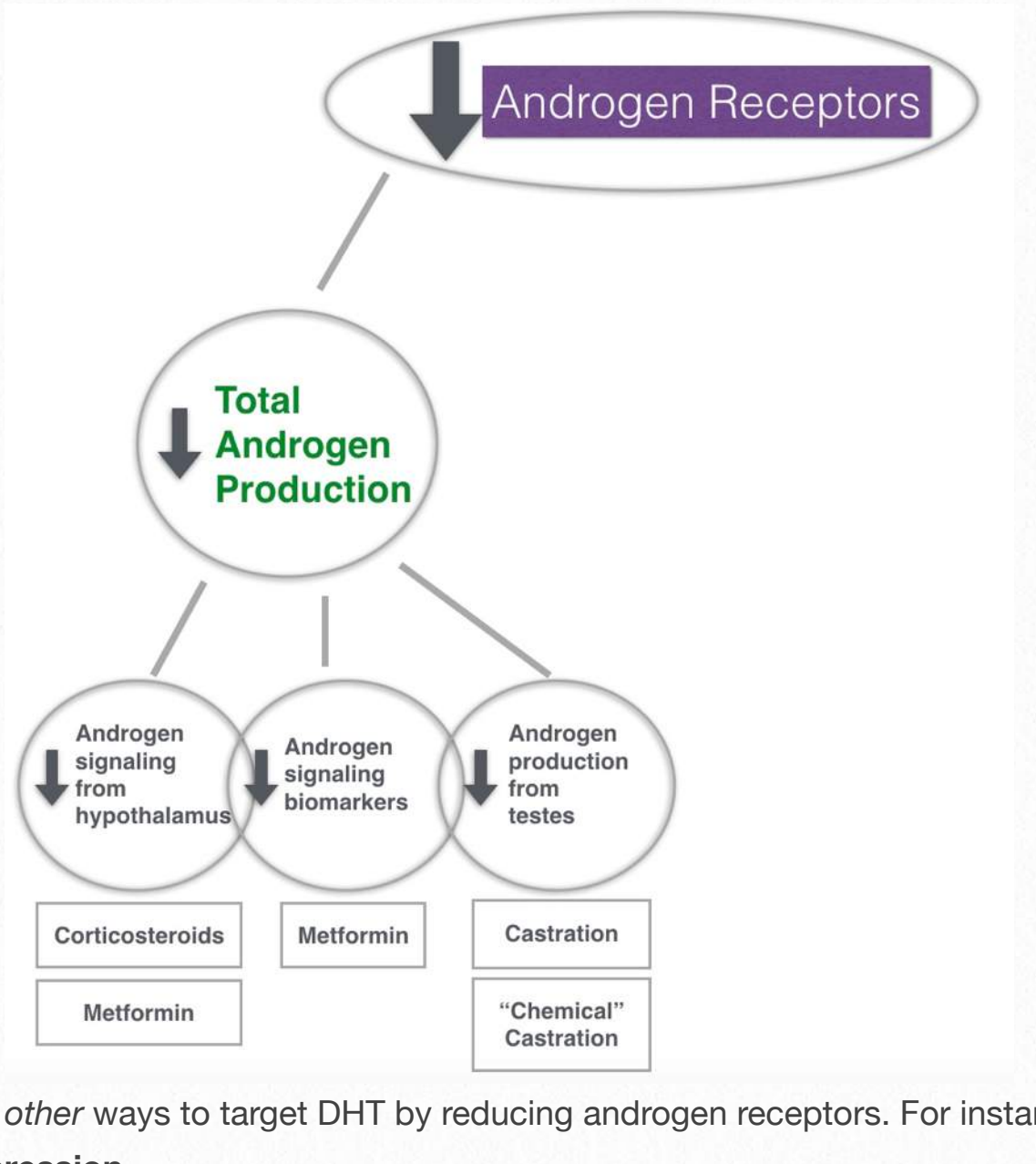
We've actually covered this method before – albeit for reducing DHT via free testosterone. And to reiterate: this is a *bad* idea.

For instance, one way of blocking our body's ability to produce androgens (and thereby *reducing* androgen receptor expression) is castration. Another way is to take drugs that change the brain signaling pathways in our hypothalamus so that our bodies convince themselves they need to produce less testosterone to thrive.

Yes, blocking total androgen production *significantly* decreases androgen receptor activity (likely because there are fewer total androgens available). But doing so comes attaches to serious side effects. And the costs of these side effects far outweigh any benefit to DHT reduction and hair health.

The consequences of this kind of DHT-reducing approach? Low/no libido, depression, sexual dysfunction, the list goes on. So when it comes to *safely* decreasing androgen receptors, please consider all other options aside from reducing total testosterone production.

In any case, here's what this looks like in a flowchart:



Fortunately, there are *other* ways to target DHT by reducing androgen receptors. For instance: **decreasing androgen receptor expression**.

And this is where it gets interesting.

#2: Decreasing Androgen Receptor Expression

When we talk about *decreasing* androgen receptor expression, we're *not* talking about manipulating androgen receptor activity by taking away the thing that tells our bodies to activate them – the androgens themselves. That's the first mechanism – decreasing *total* androgen production.

This mechanism – **decreasing androgen receptor expression** – is about changing the actual *environment* of our tissues – so that fewer androgen receptors activate in those tissues.

I personally like this approach better. Why? Because rather than chemically (via drugs) or physically (via castration) reduce our androgen production to reduce androgen receptors, we're instead changing the environment of a tissue where there are androgen receptors – so that fewer androgen receptors express.

And based on the evidence, this method is much safer.

One potential way to decrease androgen receptor expression?

***Increase* tissue oxygen levels**.

Hypoxia (Oxygen Restriction) Increases Androgen Receptor Activity

In the prostate, reduced oxygen levels – in combination with DHT – *dramatically* increases androgen receptor activity. In fact, it increases androgen receptor expression six-fold versus DHT alone.

Why is this interesting? Well, an enlarged prostate and men's balding scalps have a lot in common.

For one, our prostates *and* our balding scalp regions both use the enzyme type II 5-alpha reductase to convert free testosterone into DHT – and *not* other forms of 5-alpha reductase.

In addition, high DHT levels are associated with both balding scalp regions *and* an enlarged prostate.

But even more interesting? Hypoxia (lower oxygen) is associated with both prostate cancer *and* regions of the scalp which are balding.

Could the increased DHT we see in balding scalps somehow be connected to hypoxia? Probably. Especially when we consider how androgen receptors, in the presence of DHT *and* hypoxia, express 6-fold higher than in the presence of DHT alone.

What does all this mean? We can probably reduce DHT levels by *decreasing* androgen receptor expression. And how can we do that? By *increasing* oxygen tissue levels.

If We Increase Scalp Tissue Oxygen, Can We Reverse Hair Loss?

The evidence on oxygen therapies and DHT levels is limited, and the evidence on oxygen therapies *regrowing hair* is even more limited.

It's not because the relationship doesn't exist. Rather, it's just under-studied.

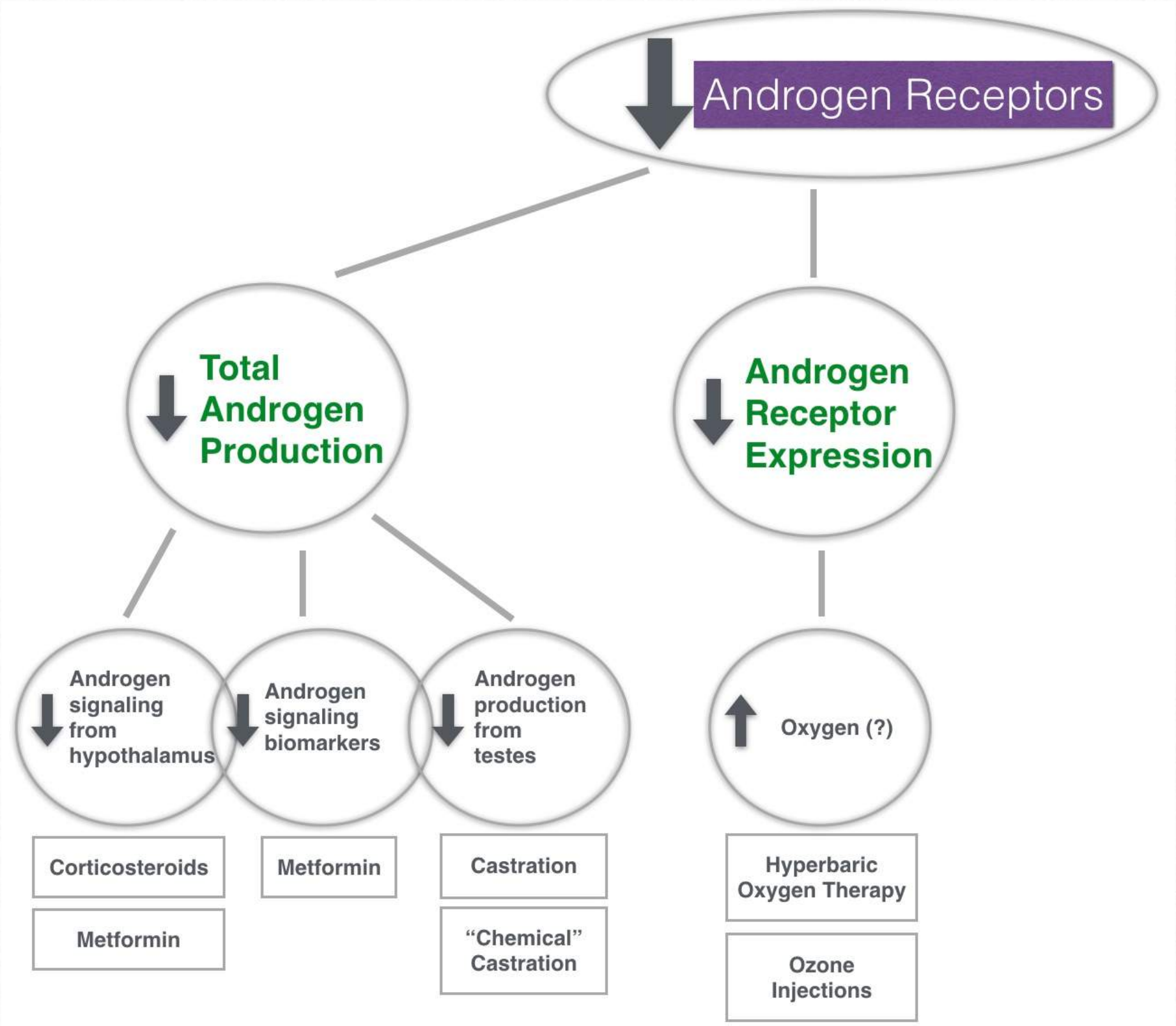
Anecdotally, I've spoken with two people who tried **hyperbaric oxygen therapy** and said that it regrew their bald vertexes over a period of four months. And there's also a patent on injectable ozone for hair loss sufferers, with cited case studies.

With that said, there's not enough evidence to say with *certainty* that increasing oxygen is a viable option for 1) reducing androgen receptors, 2) reducing DHT levels, or 3) regrowing hair. There's anecdotes, but no hard data.

Another challenge with oxygen: delivery. Just because we inhale pure oxygen *doesn't* mean we actually raise **tissue** oxygen levels. This is probably why future hair loss therapies using oxygen will come in the form of injections rather than hyperbaric chambers – if at all.

But the bottom line: if we increase tissue oxygen levels, my bet is that this will 1) decrease androgen receptor activity *and* 2) encourage hair regrowth.

So let's summarize our mechanisms (so far) for decreasing androgen receptors:



This brings us to our last mechanism to reducing androgen receptors: **blocking them**. And if you've tried many hair loss drugs or keep up with hair loss research, there's a good chance you know what's coming.

#3: Blocking Androgen Receptors

What does it mean to block androgen receptors?

In simple terms, it means to *bind* something to an androgen receptor so that the androgen receptor is "blocked off" from binding with actual androgens, like testosterone or DHT.

That's how androgen receptor blockers reduce DHT: the AR blockers bind to a cell's androgen receptors and *prevent* DHT from binding to that same cell. In effect, that DHT can no longer influence that cell's function.

Androgen receptor blockers come in two forms: **steroidal** and **non-steroidal**. And like steroidal 5-alpha reductase inhibitors, steroidal androgen receptor blockers are also synthesized from hormones like progesterone.

#1: Steroidal Androgen Receptor Blockers – Spironolactone

When it comes to hair loss (and reducing DHT), the most popular androgen receptor blocker appears to be a drug called **spironolactone** (branded as Aldactone). This is an androgen receptor blocker derived from the hormone progesterone.

Spironolactone reduces DHT by blocking androgen receptors, and doctors often prescribe an *oral* form for women suffering from female pattern hair loss or even hirsutism – unwanted body and facial hair *growth*. This is because increased DHT is associated with hair loss in the scalp, but ironically, hair growth in the body and face.

But spironolactone is a powerful anti-androgen. In fact, most men are advised against taking it *orally* as a hair loss treatment. Why? Because it's feminizing. In fact, oral spironolactone is the same drug some men use to transition genders and become female.

The good news for male hair loss sufferers? Spironolactone also comes in *topical* form – so we can concentrate its anti-androgen receptor effects to our scalps and minimize the risk of feminization.

So, if you're a man and considering reducing scalp DHT by blocking androgen receptors, I would choose topical spironolactone over oral spironolactone.

With that said, spironolactone still comes with risks.

The Risk: Steroidal Androgen Receptor Blockers (Like Spironolactone) Activate Androgen Receptors… And Can Be Overpowered By DHT And Testosterone

Let's break this down.

Each cell often has *many* androgen receptors. But not *all* of these androgen receptors are always active. In fact, in order for an androgen receptor to activate, they often need the presence of actual androgens – like testosterone or DHT.

Spironolactone is derived *from* steroids – or in other words, androgens. And as a result, our bodies read the presence of spironolactone as they would testosterone or DHT. And the response? To activate androgen receptors in cells where spironolactone is nearby.

This isn't necessarily a *huge* problem… because in a perfect world, spironolactone would *bind* to all of those androgen receptors and render them unusable. However, there are two caveats.

The first: spironolactone may not bind to all the androgen receptors it activates.

And the second: steroidal AR blockers like spironolactone *aren't* strong androgen receptor blockers. In fact, in the presence of *too much* DHT, spironolactone might dissociate from the androgen receptor it's supposed to block, allowing androgens like DHT to come in and bind to that same androgen receptor.

Do you see the problem here?

1. Spironolactone can *activate* many androgen receptors
2. Spironolactone then *binds* to some of those androgen receptors
3. …But if too much testosterone or DHT is nearby, it'll also bind to those other activated androgen receptors. And in some cases, those androgens can dissociate spironolactone and then *attach* the androgen receptor it just activated!

The result? DHT flooding. And this isn't theoretical. It's been documented.

Some men taking steroidal AR blockers to treat an enlarged prostate inadvertently end up *increasing* the weight of their prostate (prostate growth) during treatment.

Moreover, some steroidal AR blockers can accelerate the *growth* of prostate cancer – the exact opposite of their intended effects.

This is why, for men especially, steroidal AR blockers like spironolactone are finicky, tricky drugs – especially for the purpose of DHT reduction and to fight hair loss.

This is why researchers' focus on androgen receptor blockers – at least lately – is all about finding good **non-steroidal androgen receptor blockers**. And there might be a few good contenders.

#2: Non-Steroidal Androgen Receptor Blockers – RU58841

Aside from *not* being derived from hormones, the major difference between steroidal vs. non-steroidal androgen receptor blockers is that non-steroidal AR blockers are what we call "silent" androgen receptor antagonists. In other words, they block androgen receptors *without* actually activating them.

This makes them, in my opinion, a better option for anyone trying to fight hair loss by reducing DHT through androgen receptor blocking.

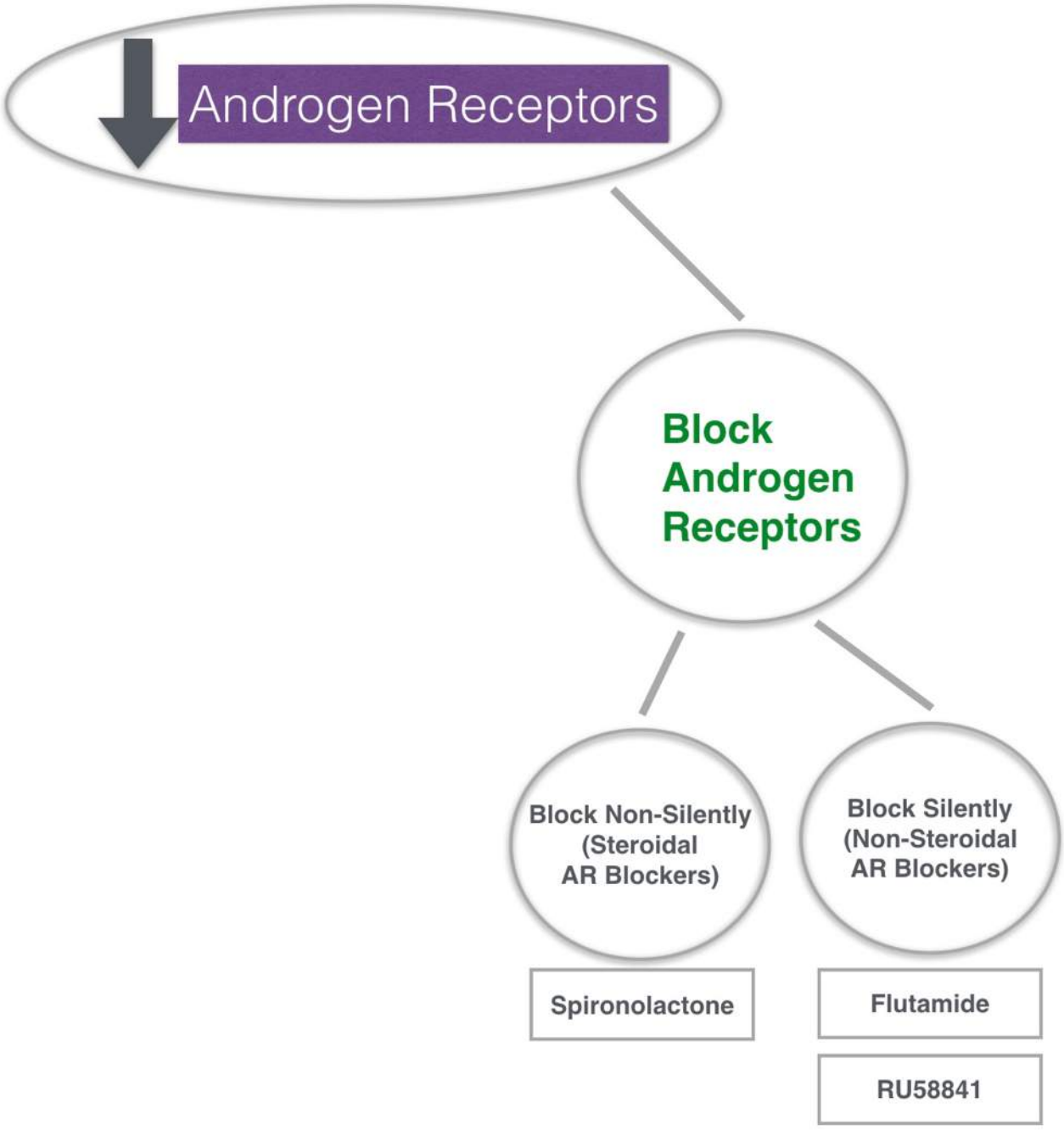
And remember those unintended, DHT-flooding effects of steroidal androgen receptor blockers? Based on the evidence, non-steroidal AR blockers don't evoke the same response. They don't likely cause growth in either the prostate or prostate cancer.

Two examples of non-steroidal androgen receptor blockers for hair loss?

1. Flutamide. Historically, this drug was mostly geared for female pattern hair loss sufferers and men with advanced stage prostate cancer. But recent advents in topical delivery via nanoparticles might make this drug effective for hair loss – and maybe even devoid of major side effects.
2. RU58841. In the past few years, RU58841 made the rounds on hair loss forums, but it has yet to *legally* make it to the US market (technically, you can still get your hands on it – albeit for "research" purposes only).

The side effects of non-steroidal androgen receptor blockers aren't fully understood, so unfortunately I can't say much. What I will say: when it comes to *any* anti-androgen – do your research, understand the risks, and exercise caution.

Now let's add all of this to a flowchart:

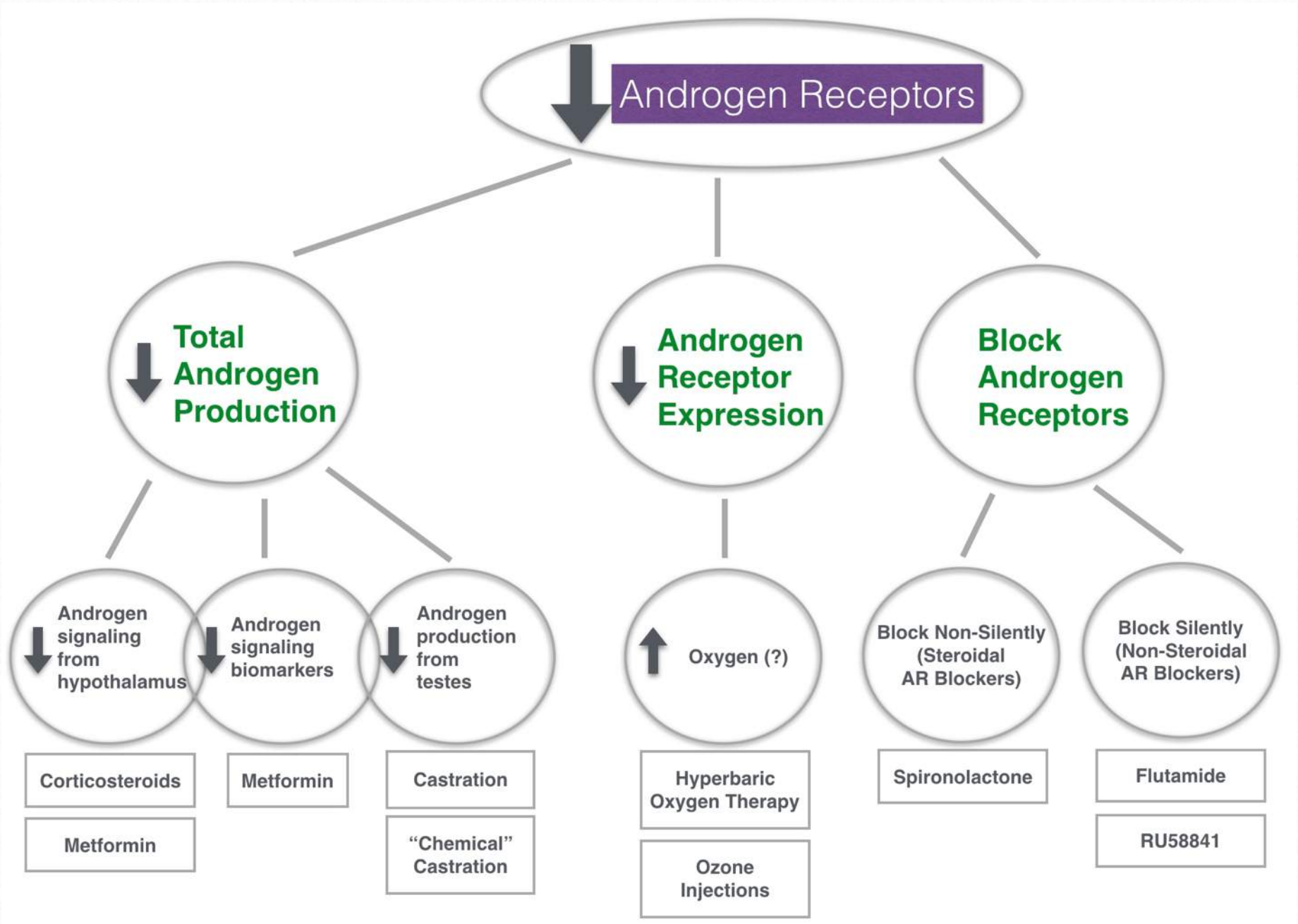


Reduce Androgen Receptors: Summary

When it comes to reducing DHT by decreasing androgen receptors, there are three major ways we can go about doing this:

1. Decrease total androgen production
2. Decrease androgen receptor expression
3. Block androgen receptors

Here's a summary of the major mechanisms behind each approach:



Unfortunately, when it comes to fighting hair loss, not *all* of these AR-reducing mechanisms hold promise.

For instance, the consequences of reducing DHT by reducing total androgen production far outweigh any potential benefits to our hair health. In addition, using steroidal androgen receptor blockers may evoke feminization *or* potentially lead to DHT flooding of the prostate and scalp tissue – the exact opposite of what we want to achieve.

So what *does* hold promise? It's hard to say. But from the looks of it, I think more research should be done on increasing oxygen levels and non-steroidal androgen receptor blockers. Those mechanisms appear to have fewer side effects, but may be extremely powerful in reducing tissue DHT levels (and potentially reversing hair loss).

So let's add these discoveries to our **master flowchart**, which is just one chapter away from completion.

What's Next…

When it comes to reducing DHT in hopes of stopping hair loss, we've covered…

- Free Testosterone
- 5-Alpha Reductase
- Androgen Receptors

But there's still a fourth DHT-reducing pillar we *haven't* discussed. What is it?

Increasing DHT metabolism.

In fact, research in increasing DHT metabolism might hold promise for hair loss sufferers looking to decrease scalp tissue DHT but *avoid* the sexual side effects of DHT reduction. This is all covered in the next (and final) chapter – where we will complete our ***Master Guide To The Mechanisms Behind DHT Reduction***.

2

Attack DHT: Increase DHT Metabolism

Summary

- A deep-dive into lowering DHT by increasing **DHT metabolism**.
- Should we even target DHT to reduce hair loss? Maybe *not*.

Increasing DHT Metabolism

Summary So Far - Decreasing DHT With Free Testosterone, 5-Alpha Reductase, Androgen Receptors, And…

In the last three chapters, we've uncovered what DHT is, how DHT is connected hair loss, and three angles of attack to reducing DHT levels to help slow or stop hair thinning:

1. Decrease free testosterone
2. Inhibit 5-alpha reductase
3. Decrease androgen receptors

Now we're going to uncover the fourth (and final) major lever to reducing DHT for the purpose of fighting hair loss: **increasing DHT metabolism**.

Increasing DHT metabolism often goes entirely overlooked, and without good reason. We'll dive into new breakthroughs to increase DHT metabolism using **bacteria** and even certain foods and supplements. And in doing so, we'll finish the final leg of our **Master DHT Reduction Flowchart**.

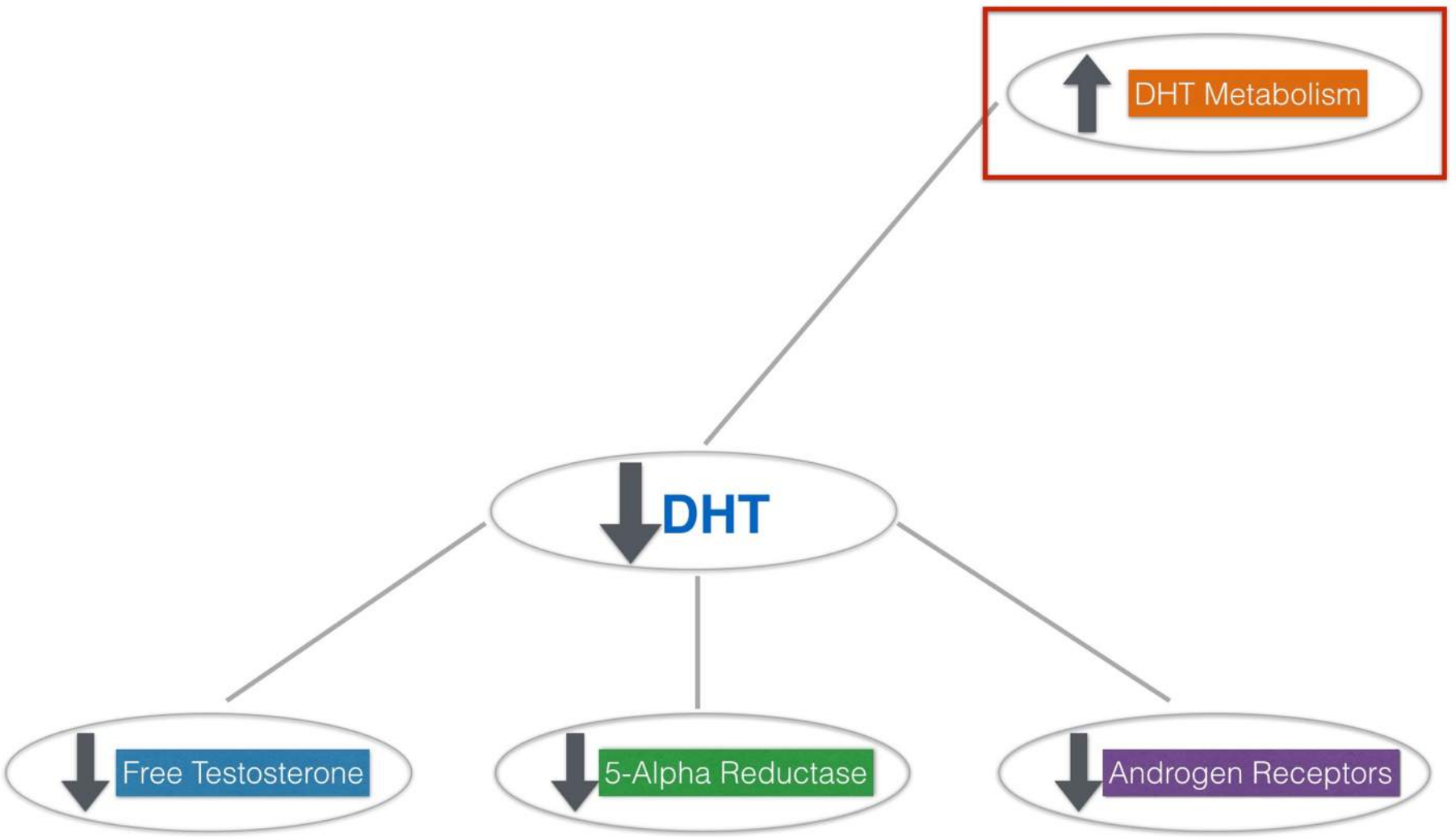
Why is this chart important? Well, using this completed flowchart, we'll be able to…

1. Recognize (and categorize) the DHT-reducing mechanisms of any drug, food, supplement, or hair loss treatment…
2. Weigh the costs (and benefits) of each DHT-reducing lever for hair loss, and…
3. Know which DHT-reducing mechanisms are worth our time and which *aren't.*

Finally, we'll cover the most important part of all: why we should look *beyond* lowering DHT if we want to achieve a full hair recovery.

Now let's get started.

Lowering DHT By Increasing DHT Metabolism



Increasing DHT Metabolism: What Does That Mean?

Dihydrotestosterone (DHT) is made from testosterone. When free testosterone interacts with the enzyme 5-alpha reductase, it gets converted into DHT. Then that DHT binds to a cell's androgen receptor. And that point, DHT can begin influencing that cell's function – from sexual health to hair loss.

We know that DHT levels are higher in balding regions versus non-balding regions of the scalp. And we also know that men who can't produce much (or any) DHT don't suffer from patter hair loss. And this is why most hair loss sufferers focus on *lowering* DHT in hopes of slowing, stopping, or reversing hair thinning.

But here's the catch: once DHT attaches itself to an androgen receptor, it doesn't stay there forever.

The reality is that this DHT eventually interacts with *other* enzymes, and then gets converted into other byproducts.

The implication for hair loss sufferers? Maybe we don't need to go about *lowering* DHT by decreasing free testosterone, inhibiting 5-alpha reductase, or decreasing androgen receptors.

Maybe instead, we can go about *increasing* the chances that DHT interacts with enzymes that turn it into other testosterone byproducts.

In other words, we *increase* the speed of DHT metabolism. We decrease the length of time in which DHT stays in our scalp tissue, and in doing so, we decrease its effects on our hair health.

That's what increasing DHT metabolism is all about. And while lowering DHT (to the extreme) by decreasing free testosterone, inhibiting 5-AR, and decreasing androgen receptors have all been shown to cause sexual side effects… The same *can't* be said about increasing DHT metabolism (at least not yet).

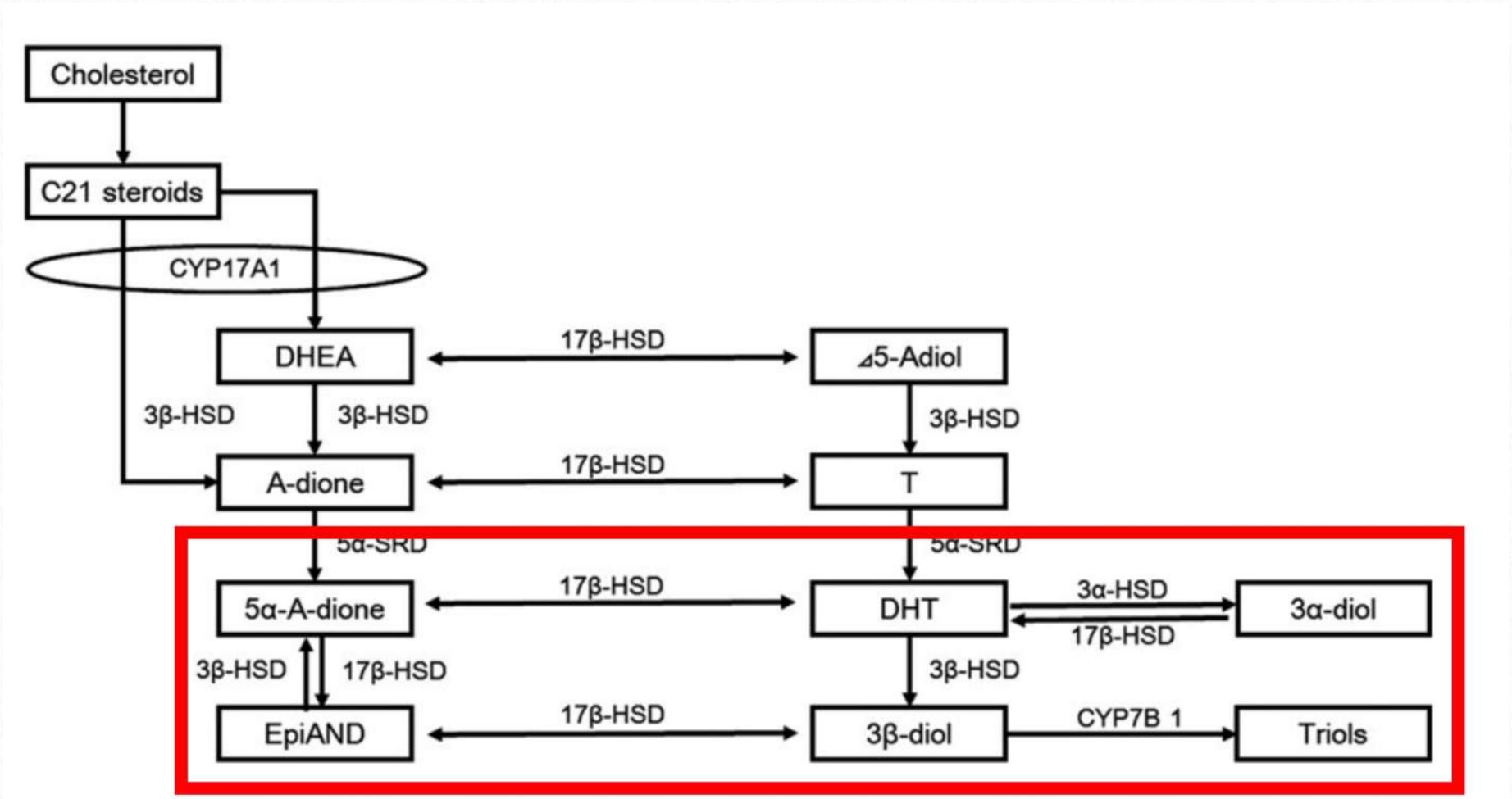
So how do we go about doing this?

One way is to increase the expression of the **enzymes** which metabolize DHT.

There are several enzymes that turn DHT into other byproducts. For example:

1. Increasing 3-alpha–hydroxysteroid dehydrogenase (3α-HSD)
2. Increasing 3-beta–hydroxysteroid dehydrogenase (3β-HSD)

And these enzymes are a part of this chart on the DHT metabolizing pathways:



But as we'll soon see, increasing some of these DHT-degrading enzymes might *not* help our hair. In fact, doing so might even hurt our hair.

So let's dive into it. For simplicity, we'll stick with these enzyme's short names: **3α-HSD** and **3β-HSD**.

Increasing DHT-Metabolizing Enzymes

3-Alpha-Hydroxysteroid Dehydrogenase (3α-HSD)

3α-HSD is one of the enzymes that metabolizes DHT into different byproducts. And interestingly, there are substances inside foods that can *increase* the activity of 3α-HSD – and thereby increase the rate of DHT metabolism. One of these substances is called sulforaphane.

Sulforaphane is found in cruciferous vegetables, but its highest concentrations is found inside **broccoli sprouts**. Sulforaphane *increases* the expression of DHT-degrading enzymes like 3-alpha hydroxysteroid dehydrogenase – at least in murine (rodent) studies. And in rats, sulforaphane supplementation *increases* hair growth!

So we should expect the same thing to happen in humans, right? If we supplement with sulforaphane, could we'll see hair regrowth too?

Maybe. Maybe not. This whole equation actually gets a lot *more* complicated.

Problem: If We Increase The Wrong *Form* Of 3α-HSD, We Might Actually Increase DHT (And Hair Loss)

Remember the enzyme 5-alpha reductase – the one that converts testosterone into DHT? Well, that enzyme comes in many different forms: type I, type II, type III, etc. However, there's only *one* form of 5-alpha reductase that's associated with pattern baldness: type II 5-alpha reductase.

That same logic can be applied to the 3α-HSD enzyme. Research shows there are *many* forms of 3α-HSD, and two important isoforms are: **AKR1C3** and **AKR1C2**.

Both AKR1C3 and AKR1C2 metabolize DHT into other androgen byproducts – like the hormone 3α-androstanediol. This is a good thing! We want to increase DHT metabolism so that we can lower the effects of DHT on our scalp skin – and reduce hair loss.

But there's a problem.

One of these isoforms – **AKR1C2** – can also convert 3α-androstanediol *back into* DHT. When it comes to hair loss, that's *not* a good thing. The more AKR1C2, the higher the chances that 3α-androstanediol will convert back into DHT (which is exactly what we're trying to avoid).

Summary So Far

- Tissue DHT is higher in balding versus non-balding scalps
- If we increase DHT metabolism, we may *decrease* the effect of DHT in our tissues
- We can increase DHT metabolism by increasing the expression or activity of the **enzymes** which convert DHT into other byproducts.
- One enzyme that converts DHT into other byproducts is called **3α-HSD**. But this enzyme comes in many forms: **AKR1C3** and **AKR1C2** (among others). And depending on the form, this enzyme will behave differently.
- When it comes to decreasing DHT levels, we *only* want to increase AKR1C3 – *not* AKR1C2. Why? Because both AKR1C3 and AKR1C2 can convert DHT into other hormonal byproducts… but *only* AKR1C2 can convert those byproducts *back into* DHT.

For us humans, we have to be selective about which enzyme isoform of 3α-HSD to increase.

If we increase AKR1C3, we *might* increase DHT metabolism and help our hair. If we increase AKR1C2, we might *increase* the conversion of 3α-androstanediol into DHT, and actually hurt our hair.

So… Does Sulforaphane Increase AKR1C3 Or AKR1C2?

We don't know! While the sulforaphane-hair regrowth study on rats saw a dose-depending increase in two 3α-HSD isoforms (AKR1C21 and DHRS9), that study didn't measure AKR1C2 or AKR1C3.

And until we know more about sulforaphane's effects on AKR1C3, AKR1C2, and other 3a-HSD isoforms, we can't say for sure that it's a viable hair loss treatment for humans.

Personally, I think sulforaphane *might* help – and that in the future, studies will demonstrate its hair growth promoting effects on humans too. But until there's stronger evidence, we have to exercise caution.

With that said, some research teams are putting big bets on the idea that if we increase 3a-HSD, we'll decrease DHT and help slow, stop, or reverse hair loss.

In fact, *one* research team is taking this concept and developing a hair loss product. The most interesting part? It's made entirely out of **bacteria**.

Increasing DHT Metabolism With Bacteria

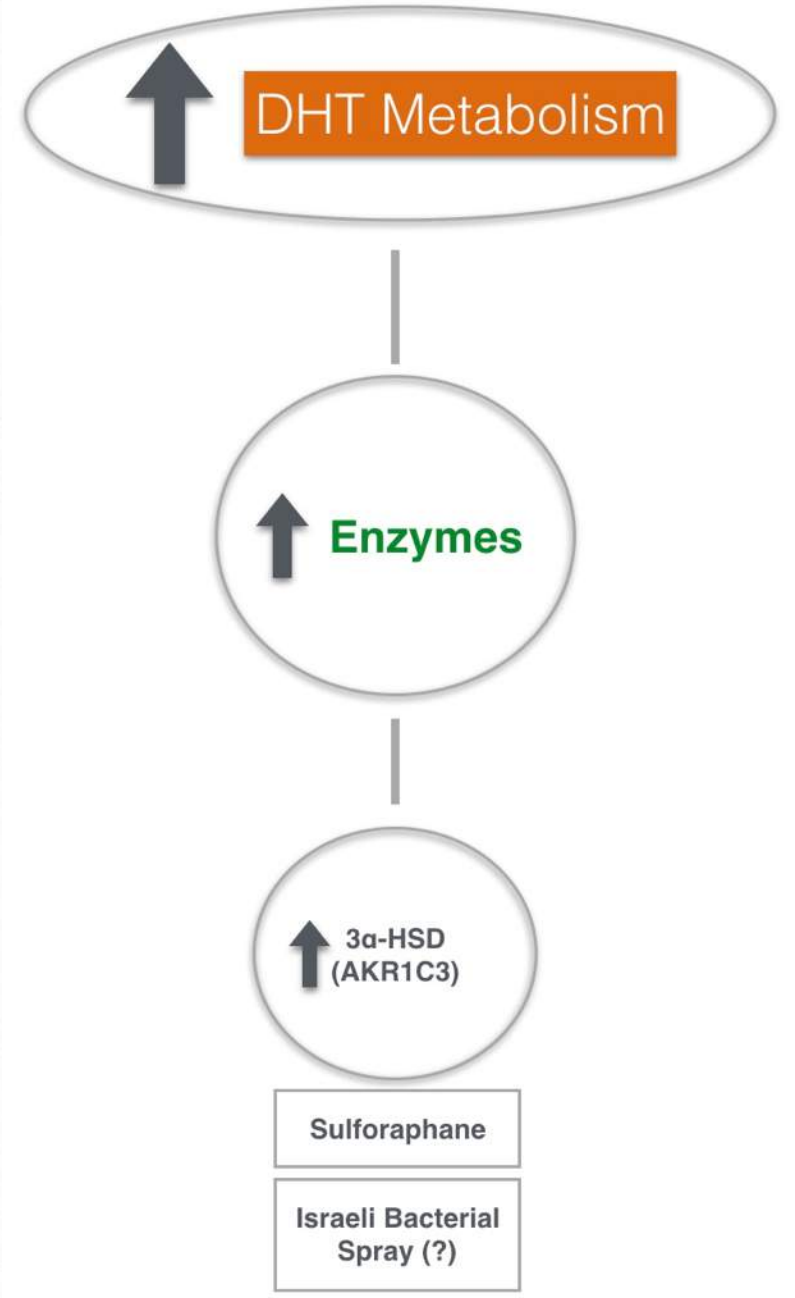
One breakthrough in the biomedical field is that by using genetic engineering, we can now *manipulate* bacterial strains to begin *overproducing* certain enzymes… enzymes like the DHT-metabolizing enzyme **3α-HSD**.

This is exactly what a group of Israeli researchers are doing. They're manipulating the bacteria *Bacillus subtilis (B. subtilis)* to overproduce 3α-HSD in hopes of 1) decreasing tissue DHT, and 2) promoting hair growth. The concept is as follows:

- *B. subtilis* is a bacteria that already lives naturally on our scalps
- If we introduce the gene for 3α-HSD into *B. subtilis,* then colonize the manipulated bacteria on our scalp skin, then that bacteria will *overproduce* the 3α-HSD enzyme in our scalp tissues…
- …and increase the metabolism of DHT – hopefully promoting hair regrowth

I'm interested to see how the trial turns out. If this treatment works, it would be as simple as a bacterial spray. Let's just hope they're increasing the 3α-HSD enzyme isoform AKR1C3, and not AKR1C2.

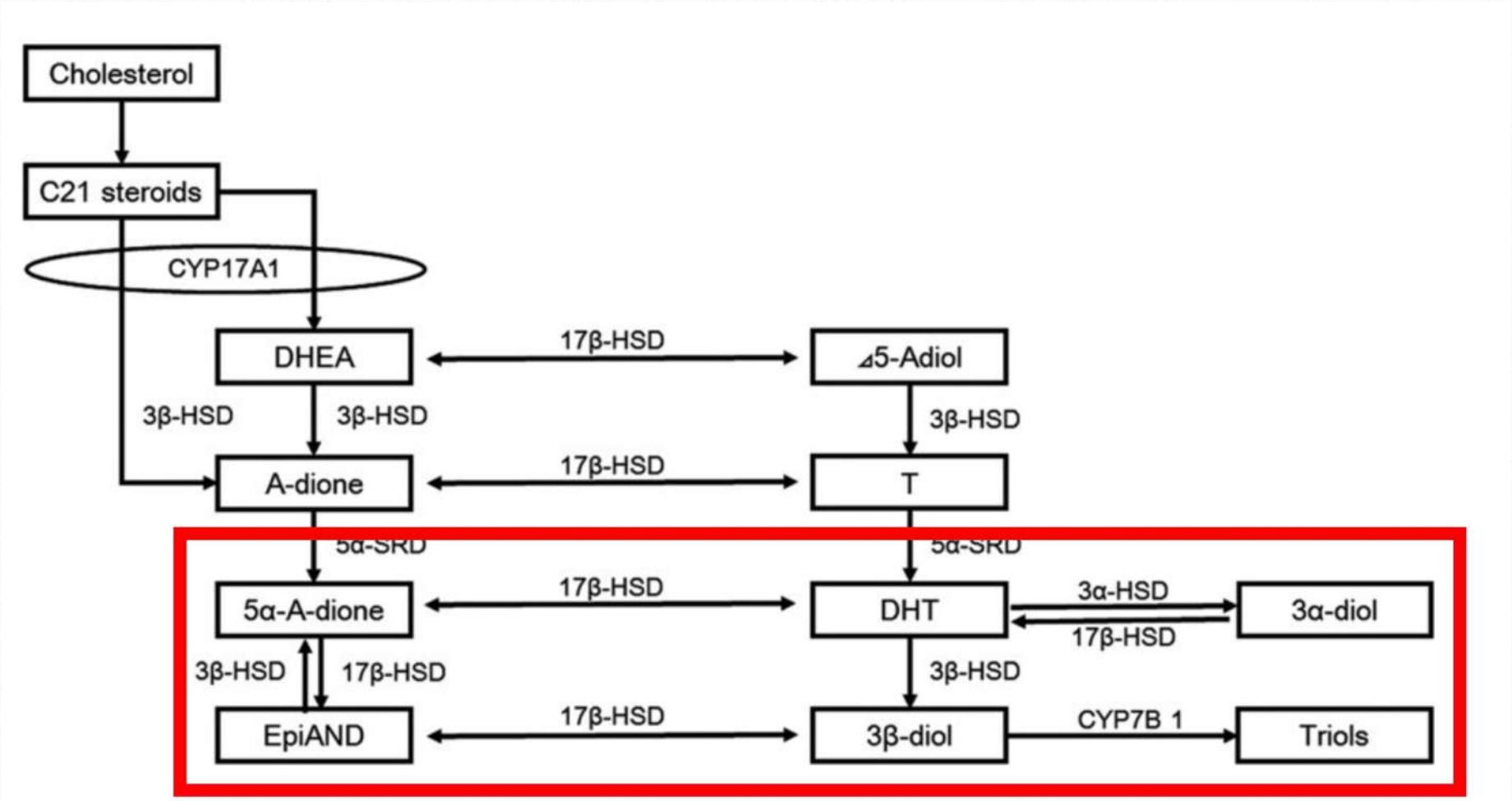
Now let's add the 3α-HSD enzyme into our flowchart.



What About That *Other* Enzyme… 3β-HSD?

This is where things get even more complicated.

Let's refer back to that DHT metabolism chart. We've already covered one DHT metabolizing enzyme: 3α-HSD. So this time, let's focus on **3β-HSD**.



Theoretically, if we increase 3β-HSD, then we should increase DHT metabolism, and in doing so, maybe help fight hair loss… Right?

Unfortunately, evidence is mixed on whether increasing 3β-HSD will actually decrease DHT levels or have any impact on our hair.

In fact, other researchers have even stated that abnormal expression of *any* of these DHT metabolizing enzymes (3α-HSD, 3β-HSD, or even 17β-HSD) might increase scalp DHT… and possibly even accelerate the balding process!

Why is this the case? How is it that *increasing* enzymes that metabolize DHT can *also* inadvertently increase DHT levels? That seems paradoxical.

It is. Until we refer back to our chart and realize that even if we increase DHT metabolism, we *might not* decrease DHT levels. Why? Because **DHT can recycle back into itself**.

Problem: DHT Can Recycle Back Into Itself

After DHT is metabolized by the enzyme 3α-HSD, that metabolite only requires a *single* enzyme to then convert back into DHT. That enzyme: 17β-HSD. That's a one-step recycling process back to DHT.

But what about 3β-HSD?

Well, if DHT is metabolized by the enzyme 3β-HSD, this whole step-process is much longer and requires multiple enzymes. But… the DHT recycling can *still* happen.

Unfortunately I haven't found evidence that, all else equal…

1. Increasing 3β-HSD will decrease DHT (in otherwise healthy people), or…
2. Increasing 3β-HSD will help fight hair thinning.

So I've excluded this enzyme (and a couple others related to DHT metabolism) from the flowchart.

Should We Increase DHT Metabolism To Lower DHT (And Keep Our Hair)?

I don't think anyone knows.

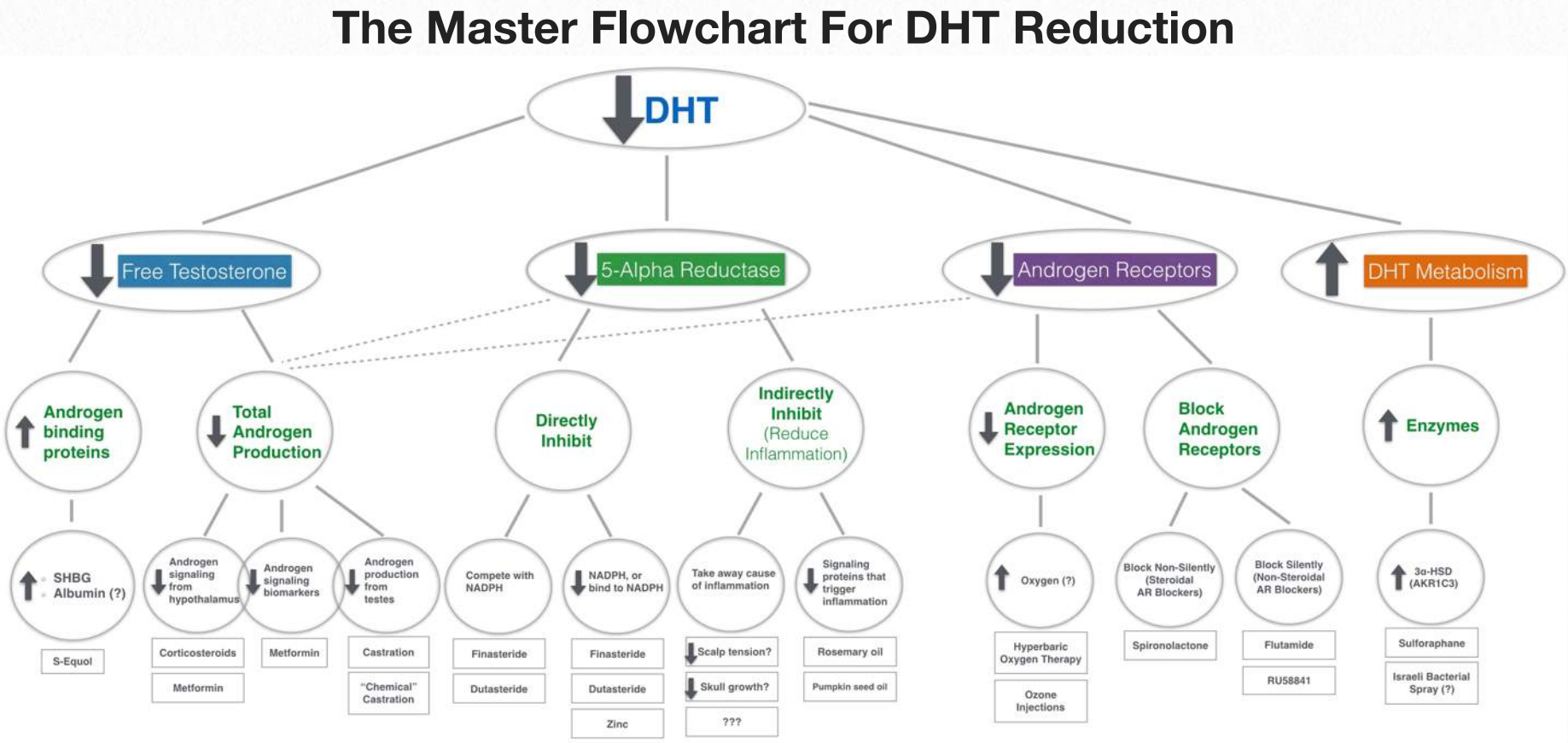
The truth is that we barely understand these enzymes, and even less so their relationships to pattern hair loss. And while there's evidence that increasing DHT metabolism via 3a-HSD can promote hair growth in *rats* – we're not sure yet if this will carry over to humans.

The good news? Within expected doses, sulforaphane appears to be safe. If you want to include a sulforaphane supplement or sulforaphane-containing foods in your diet, go for it! It's not hard to find broccoli sprouts at the grocery store. And it's pretty easy to grow those sprouts yourself.

And the better news? We've just completed our final lever of our **DHT Reduction Master Flowchart**.

The Final Flowchart: A Master Guide To DHT Reduction For Hair Loss

We can now add DHT metabolism to our flowchart. So without further ado, here is the completed **Master Flowchart For DHT Reduction** (click to enlarge).



Now let's put this chart to use.

How To Use The Flowchart: Customize Your DHT-Lowering Protocol!

Are you considering any DHT-reducing supplements to include in your fight against hair loss? Use this chart to better understand how these supplements work and if they're worth trying.

Better yet, use this chart to **customize** a DHT-reducing plan to maximize 1) your angles of attack, 2) efficacy, and 3) safety.

For example, which approach do you think is better?

First Approach: you want to reduce DHT levels to fight hair loss, so you buy *tendifferent supplements.* Every single supplement does the same thing: it targets DHT by inhibiting the enzyme 5-alpha reductase.

Second Approach: you want to lower DHT levels to fight hair loss, so you buy *ten different supplements*. One supplement that inhibits 5-alpha reductase *indirectly*, one supplement that *increases* the expression of androgen-binding proteins (S-Equol), one topical that targets 5-alpha reductase through lipid bilayer permeation (pumpkin seed oil), one supplement increases DHT metabolism, and maybe one experimental topical possibly increases tissue oxygen levels in the scalp, and in doing so, maybe decreases androgen receptor expression.

Both approaches likely cost the same. But I'd bet your money is better spent on the *second* approach. Why? Because you're not *just* targeting **one lever** of DHT reduction. You're targeting all **four**.

In doing so, you're 1) targeting hair loss from multiple angles, 2) not hammering any lever *too* hard, and as a result, 3) likely spreading out your risk for side effects.

The big takeaway: we can target DHT more broadly by targeting multiple *levers*, rather than many supplements that only target the *same* lever.

And now that we've covered this, here's a cold hard truth about DHT and hair loss.

Final Thoughts...

Should We Target DHT To Reverse Hair Loss?

Maybe not. And here's why.

Yes, Finasteride can plummet DHT to near-castration levels. And yes, castration (and Finasteride) have an incredibly high success rate for stopping pattern hair loss dead in its tracks.

But here's the catch: those taking Finasteride don't see full hair recoveries. Rather, the regrowth that they get is modest, with some studies showing merely a 10% increase in hair count over 48 weeks.

That's not very impressive. And it begs the question: if DHT *causes* hair loss, then how come after we *take away* DHT, all of our hair doesn't grow back? How come our hair loss only stops, or our hair thickens a bit, with some minor recovery in recently lost hairs?

No researcher is asking this question, and I don't know why. Maybe it's because pharmaceutical companies are financially bound to the DHT-hair loss connection. Maybe it's because nobody really cares to answer this. Maybe it's because in most scientific fields, 90% of funding goes toward treatment testing and *just* 10% goes toward understanding the pathology of a disease. The end-result? We're all shooting in the dark, clawing to create the *best* treatment for a condition we barely understand.

But if we take a step back, and look at DHT's relationship to other conditions – like heart disease, atherosclerosis, and wounding-healing – one thing becomes clear.

DHT plays a causative role in the formation of two conditions: **fibrosis** and **calcification**. And unsurprisingly, fibrosis and calcification are observed all over balding scalps. These conditions reduce blood flow, oxygen, and nutrient supplies to tissues. And if we take away the *triggers* of fibrosis and calcification (like DHT), we *don't* take away the damage already done to those tissues – and the scar tissue already formed.

Maybe if we want to reverse hair loss and see full hair recoveries, we need to focus less on DHT, and *more* on ways to reverse calcification and fibrosis. Yes, DHT-reducing drugs, foods, supplements, and topicals will help slow or stop hair loss. Yes, maybe they'll grow back a little bit of hair. And yes, maybe they'll cause some sexual side effects (after all, DHT is critical to male sexual function).

But if we want *full* hair regrowth, we better develop ways to undo the damage already done. And that means we better find ways to reverse calcification and fibrosis.

Want Strategies To Reverse Hair Loss By Targeting Fibrosis And Calcification?

I've written a free email course which gives you the basics. You can sign up here.

Or, you can pick up a copy of my recent book: *Perfect Hair Health: A Guide To Natural Hair Recovery*. Inside, you'll find my before-after photos, plus the exact regimen I used to reverse my hair thinning - all without drugs, supplements, topicals, or surgeries. You'll also find several case studies of other hair loss sufferers who worked with me and achieved even *better* results. Access everything right here.