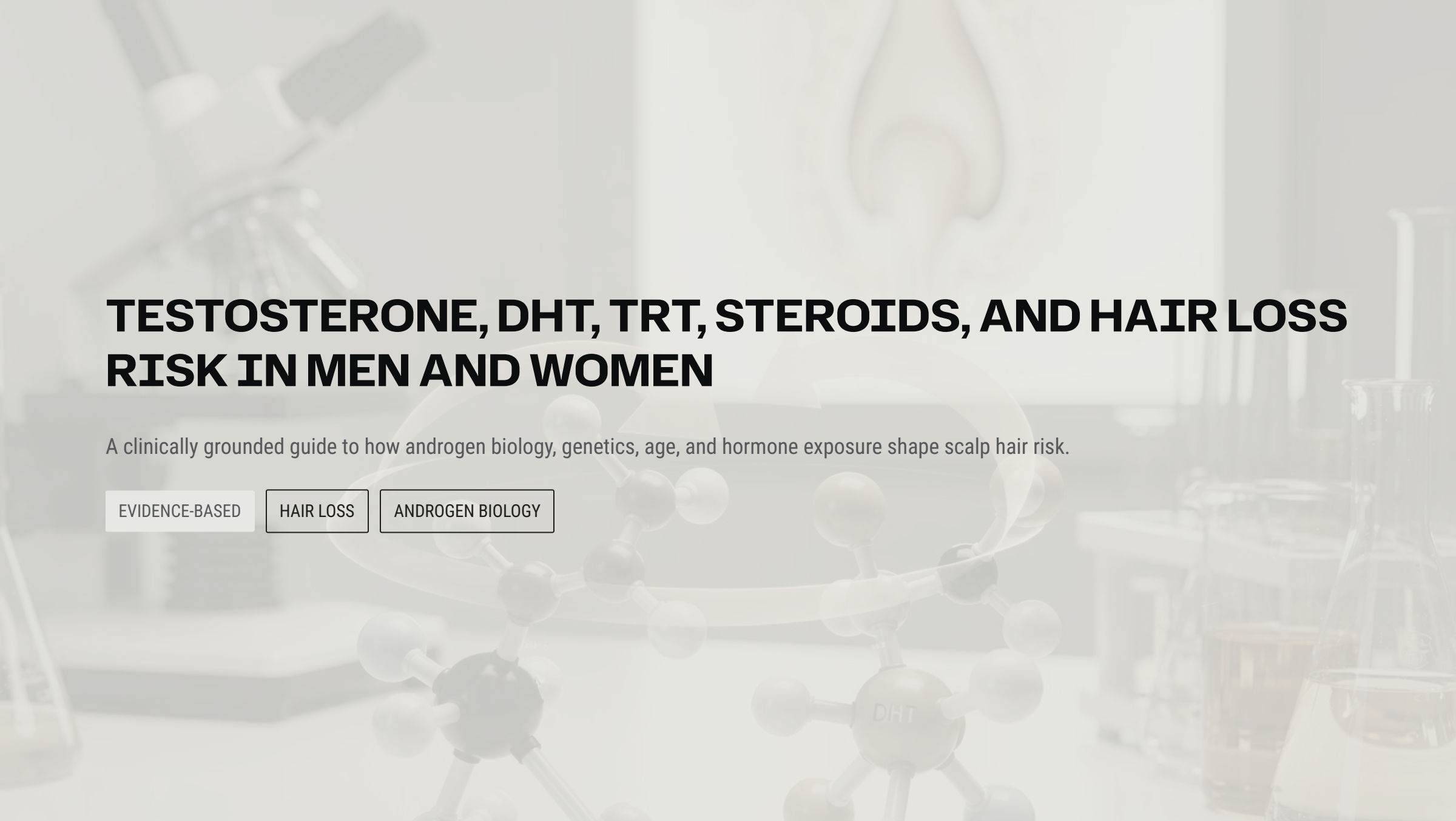

TESTOSTERONE, DHT, TRT, STEROIDS, AND HAIR LOSS RISK IN MEN AND WOMEN

A clinically grounded guide to how androgen biology, genetics, age, and hormone exposure shape scalp hair risk.

EVIDENCE-BASED | HAIR LOSS | ANDROGEN BIOLOGY

WHY THIS TOPIC IS SO MISUNDERSTOOD

THE OVERSIMPLIFICATION PROBLEM

Few topics in hair medicine are as routinely oversimplified as the relationship between testosterone and hair loss. The phrase "testosterone causes baldness" has become cultural shorthand — repeated in gym locker rooms, on social media, and even in some clinical settings — despite being a fundamentally incomplete description of the biology.

The result is a persistent fog of misinformation. Patients are left poorly equipped to understand their own risk, and sometimes poorly advised about the interventions they are considering.

WHY THE BIOLOGY IS MORE COMPLEX

A key source of confusion lies in the failure to distinguish between total testosterone, free testosterone, dihydrotestosterone (DHT), and androgen receptor sensitivity. These are not interchangeable terms.

A man with high total testosterone but elevated sex hormone-binding globulin (SHBG) may have less biologically active androgen circulating than a man with moderate total testosterone and low SHBG. And neither of those figures, in isolation, tells you what is happening at the level of the scalp follicle, which is ultimately where androgenetic alopecia (AGA) begins and progresses.

THE CLINICAL BLIND SPOTS

Gym culture and the rapidly expanding "testosterone optimisation" world have added a further layer of complexity. Discussions about TRT, hormone panels, and performance enhancement frequently proceed without serious attention to the scalp-hair question.

Men starting TRT are often not counseled on hair risk. Men beginning anabolic steroid cycles may have no awareness that androgenic pressure on follicles can be dramatically amplified beyond the physiological range. Women are almost entirely absent from these conversations, even when they have legitimate androgen-related hair concerns of their own.

WHY THIS MATTERS

This guide is written precisely to address that gap. The central argument is straightforward but important: **hair loss risk is usually about androgen response, not just androgen level.**

Takeaway for the reader: when assessing hair-loss risk, look beyond total testosterone and ask how much androgen is available, how strongly follicles respond, and whether DHT-driven sensitivity is likely to matter.

THE SIMPLIFICATION PROBLEM

"Testosterone causes baldness" is repeated widely but is biologically incomplete and clinically misleading.

THE TERMINOLOGY PROBLEM

Total T, free T, DHT, and receptor sensitivity are routinely conflated — even in professional contexts.

THE EXCLUSION PROBLEM

Women are rarely included in androgen-hair discussions, despite real and clinically relevant overlap.

THE CULTURE PROBLEM

Optimisation and gym communities often proceed without engaging the hair-loss trade-off at all.

THE ANDROGEN INDEX: A BETTER FRAMEWORK

The Androgen Index, as used throughout this guide, is not a laboratory test or a validated clinical scoring tool in the formal sense. It is a **clinical thinking model** — a structured way of holding multiple relevant variables in view at the same time, rather than anchoring the entire assessment to a single blood result. This matters because androgenetic alopecia is a multifactorial condition, and no one number can capture the full picture.

In practical terms, the model helps you ask the right questions: *How much inherited risk is present? How long has thinning been developing? Is there evidence of active miniaturization? How much androgen exposure is likely reaching the follicle?* The answer comes from the pattern across several domains, not from any one metric alone.

FAMILY HISTORY

Inherited susceptibility. A strong family history increases the likelihood that follicles will respond to androgens in a hair-loss pattern.

AGE AND PATTERN OF ONSET

When thinning began, and where it is occurring, helps distinguish early AGA from other causes of shedding or diffuse loss.

HAIR CALIBER CHANGE

Progressive miniaturization — hairs becoming finer over time — is one of the clearest signs that true AGA is active.

ANDROGEN EXPOSURE HISTORY

What the body has been exposed to over time, including endogenous variation and any exogenous use, provides essential context that a single blood test cannot.

DHT CONVERSION POTENTIAL

How readily testosterone is converted to DHT, largely through 5-alpha-reductase activity, affects the strength of the signal reaching the follicle.

SCALP OILINESS, ACNE, SEBORRHEA

Visible signs of androgenic activity that can help reinforce the clinical picture, especially when lab results are ambiguous.

TRT OR ANABOLIC STEROID USE

A modifiable exposure variable that can amplify androgen pressure on follicles and change the pace of loss.

HORMONAL CONTEXT

SHBG and the free androgen fraction help explain how much biologically active androgen is actually available to tissues.

FEMALE HYPERANDROGENIC CLUES

Irregular cycles, acne, facial hair, and PCOS features can point to androgen sensitivity even when routine bloodwork looks normal.

How to use the framework: look at the full pattern, not just the testosterone value. The goal is to estimate follicular risk with more nuance, more context, and less false certainty.

No single spoke of this wheel is sufficient to define risk. The value of the model lies in viewing all domains together — weighting them appropriately for the individual — and using that composite picture to guide assessment and action.

TESTOSTERONE VS. DHT: THE DIFFERENCE THAT MATTERS

TESTOSTERONE

A steroid hormone produced primarily in the testes in men and in smaller amounts in the ovaries and adrenal glands in women. It circulates in both free and protein-bound forms and supports a wide range of functions — muscle development, libido, red blood cell production, bone density, and mood.

DIHYDROTESTOSTERONE (DHT)

A more potent androgen formed when testosterone is converted by the enzyme 5-alpha-reductase, especially type II in scalp follicles. DHT binds the androgen receptor with about five times greater affinity than testosterone, which makes it far more biologically active at the follicular level.

The enzyme 5-alpha-reductase exists in two main isoforms: type I, found more broadly in sebaceous glands and the liver, and type II, concentrated in hair follicles, especially in the scalp. It is type II activity in the frontal and vertex regions that most directly influences androgenetic alopecia. This is why finasteride — a selective 5-alpha-reductase inhibitor — lowers DHT and can slow or halt miniaturization in predisposed men, usually without dramatically changing total testosterone levels.

The key clinical point is that **same testosterone does not mean same hair outcome**. Two men can have virtually identical testosterone levels and very different hair loss patterns depending on how much of that testosterone is converted to DHT, how sensitive their scalp follicles are to androgen signaling, and how densely those follicles express androgen receptors. Blood testosterone is therefore only a partial proxy for what is happening at the follicular level.

This also helps explain why scalp hair and body hair can behave in opposite ways under similar hormonal conditions. Beard and body hair are often stimulated by DHT, while scalp follicles in genetically susceptible areas become progressively miniaturized. Location and inherited receptor sensitivity determine how the same androgen signal is interpreted — a biological nuance that is central to understanding hair-loss risk.

→ **TESTOSTERONE PRODUCED**

In testes, ovaries, and adrenal glands. Circulates in free and bound forms.

→ **5-ALPHA-REDUCTASE CONVERTS TESTOSTERONE → DHT**

Primarily type II isoform in scalp follicles. Activity varies between individuals.

→ **DHT BINDS ANDROGEN RECEPTORS IN FOLLICLES**

~5× greater affinity than testosterone. Drives miniaturization in susceptible follicles.

→ **PROGRESSIVE FOLLICULAR MINIATURIZATION**

Shortened anagen phase, thinner caliber hair, eventual follicle dropout — clinical AGA.

GENETICS AND FOLLICULAR SENSITIVITY: WHY SOME PEOPLE LOSE HAIR AND OTHERS DO NOT

Androgenetic alopecia is highly heritable, but it is not driven by a single gene. The androgen receptor gene on the X chromosome has long drawn attention, which is why people often say baldness “comes from the mother’s side.” There is some truth to that — but not the whole story. Large genetic studies have identified more than 350 associated loci, many on autosomal chromosomes, so risk is inherited from **both parents**.

What these genes really influence is **follicular sensitivity to androgen signaling**. Two people can have similar DHT levels and very different hair outcomes if one person’s frontal and vertex follicles are more sensitive to androgens than the other’s. That is why family history is usually more informative than a hormone panel alone, and why scalp exam findings — miniaturization, caliber variation, and recession pattern — matter so much clinically.

Sensitivity also varies by scalp region. Occipital and temporal donor follicles are typically resistant to androgen-driven miniaturization. That is why transplanted hair usually keeps its growth behavior after relocation: it carries its donor-zone programming with it. By contrast, frontal, mid-scalp, and vertex follicles in susceptible people tend to express higher androgen receptor density and 5-alpha-reductase activity, making them more vulnerable to DHT.

Age of onset can still differ widely, even within the same family. Modifier genes, hormones, inflammation, and cumulative exposure all appear to affect when susceptibility becomes visible. So a father who receded at 22 does not guarantee the same timeline — but it does suggest a shared inherited risk.

AGE, EXPOSURE TIME, AND ANDROGEN LOAD ACROSS THE LIFESPAN

Understanding hair-loss risk requires more than a current androgen level. What matters is the **lifetime androgen exposure** a susceptible follicle has experienced since puberty — a cumulative burden that is often missed in cross-sectional blood tests.

1 PUBERTY (12–18)

First major window of androgen signaling. DHT rises sharply, and early temple recession or miniaturization may begin in highly susceptible individuals.

2 EARLY ADULTHOOD (20S)

Testosterone and DHT are often near peak in men. Early-onset AGA may accelerate here, while others with lower susceptibility remain stable.

3 MID ADULTHOOD (30–50S)

Cumulative follicular injury accumulates. As SHBG falls in some men, the free androgen fraction may rise even if total testosterone does not.

4 LATER LIFE (50+)

Total testosterone often declines, yet AGA may continue. Follicles that have already miniaturized do not fully recover, and prior exposure shapes the trajectory.

Practical takeaway: Lower testosterone now does not undo years of follicular miniaturization already in motion.

That is why a patient can say, “My testosterone is lower now than it was at 30 — why is my hair still thinning?” The answer is that miniaturization unfolds over years. Once the follicular papilla has been repeatedly exposed to androgenic signaling, the anagen phase shortens and structural changes can become self-reinforcing, even if hormone levels later soften.

Age-related SHBG changes add another layer. In some middle-aged men, falling SHBG increases the biologically active free androgen fraction even as total testosterone declines. For that reason, total testosterone alone is an imperfect proxy for follicular androgen activity across the lifespan.

ENDOGENOUS TESTOSTERONE IN MEN: WHAT IT CAN AND CANNOT TELL YOU

For men trying to estimate hair-loss risk through hormone testing, total testosterone is usually the first — and sometimes the only — number they see. On its own, it is a limited signal. It can help frame the endocrine picture, but it does not explain scalp-level androgen risk by itself.

Total testosterone includes three main fractions: testosterone tightly bound to SHBG, testosterone loosely bound to albumin, and free testosterone, which is unbound and available to tissues. In practical terms, two men can have the same total testosterone and very different biologic androgen exposure if their SHBG differs. SHBG varies with genetics, body composition, thyroid function, insulin sensitivity, age, and alcohol use. That is why calculated or measured free testosterone is often more useful than total testosterone alone — though still not the whole story.

> **Key practical point:** Blood testosterone can help describe hormone status, but it does not tell you how strongly the follicles on your scalp will respond.

The bigger limitation is that blood testosterone does not reveal follicular sensitivity to DHT, which is largely determined at the receptor level and influenced by genetics. A man with modest testosterone may still lose hair quickly if his follicles are highly sensitive to androgen signaling. Another man with higher testosterone may remain stable if his follicles are less reactive. For that reason, clinical pattern assessment often matters more than a hormone panel in isolation.

Real-world clues can be helpful when looking for higher androgen responsiveness. Oily scalp or skin, acne history, dense beard growth, early temple recession, rapid progression of thinning, and a strong family pattern of male hair loss all increase suspicion that the follicles are androgen-sensitive. These signs are not diagnostic on their own, but they often provide more context than a single testosterone value.

In practice, the most useful interpretation comes from combining hormone data with pattern recognition, family history, and scalp examination. That is the level of assessment that better reflects what is actually happening at the follicle.

TRT AND HAIR LOSS: WHERE THE REAL RISK SITS

Testosterone replacement therapy occupies a nuanced position in the hair-loss discussion. It is neither the guaranteed path to baldness that some fear nor the risk-free hormonal optimization that some advocates imply. The reality is more conditional: TRT can accelerate visible AGA in men who are already predisposed, while producing little or no discernible scalp change in men who lack significant genetic susceptibility.

The mechanism is straightforward. TRT — even when administered at physiological replacement doses — raises circulating testosterone, which in turn provides more substrate for 5-alpha-reductase to convert to DHT. In a man with high follicular androgen receptor sensitivity and active miniaturization already underway, that added DHT load can accelerate an existing process. In a man without that genetic background, the same hormonal shift may produce no meaningful scalp change whatsoever.

> **Key practical takeaway:** TRT is most likely to matter when there is already evidence of androgen-sensitive hair loss. The question is not whether TRT can cause baldness in everyone — it cannot — but whether it will speed up loss in someone already on that path.

STABLE HAIR BEFORE TRT

Lower risk if family history is absent. Some men notice nothing. Others may still see subtle change, so monitor closely in the first 6–12 months.

EARLY THINNING BEFORE TRT

Higher-risk category. Pre-existing miniaturization may accelerate, with faster temple recession, crown drop, or an oilier scalp within months.

FAMILY HISTORY + OLDER AGE

Meaningful cumulative exposure may already be present. TRT can unmask latent susceptibility or push an established pattern forward more quickly.

The distinction between physiological replacement and supraphysiological dosing also matters. Men whose TRT is carefully managed to bring testosterone into the normal physiological range are generally at lower risk than those whose treatment results in above-range levels. However, even within-range TRT can meaningfully increase hair-loss progression in the subset of men with high genetic susceptibility — particularly when combined with elevated 5-alpha-reductase activity.

For men thinking of starting TRT, the practical approach is to assess risk before treatment begins. That means reviewing family history on both sides, checking for early miniaturization, noting scalp oiliness, and considering age of onset. If loss is already visible, a conversation about DHT-targeting treatment before or alongside TRT is often more useful than waiting for the pattern to worsen. Early intervention is substantially more effective than trying to reverse advanced miniaturization later.

In other words: TRT should not be viewed as hair-safe or hair-hostile in the abstract. It should be evaluated in the context of the individual man, his baseline pattern, and the likelihood that his follicles are already androgen-sensitive.

STEROID CYCLES, BODYBUILDING, AND ACCELERATED ANDROGENIC PRESSURE

Anabolic-androgenic steroids (AAS) sit at a different level of androgenic exposure than medical TRT, and that distinction matters when assessing hair-loss risk in the context of gym culture and performance enhancement.

MEDICAL TRT

Aims to restore testosterone to a normal physiological range. Hair risk is usually conditional on genetic susceptibility, baseline miniaturization, and whether levels remain in-range.

AAS CYCLES

Often involve supraphysiological doses of testosterone, sometimes stacked with additional compounds. Total androgenic burden can exceed physiologic norms by a wide margin, but the impact varies by compound, dose, duration, and individual susceptibility.

Key practical point: Risk is rarely about one compound alone. Stacks differ, cycles differ, and a man with early AGA may notice faster recession, more scalp oiliness, worsening seborrhea, or a crown that seems to drop more quickly after a cycle.

MEDICAL TRT

Usually lower risk than AAS cycles, but not hair-neutral. Men with pre-existing thinning may still see progression if they are susceptible.

STACKED AAS CYCLES

Higher androgenic pressure, especially when multiple compounds are combined. The scalp response can be stronger when dose and duration rise together.

SUSCEPTIBILITY MATTERS

Family history, age, and visible miniaturization all change the equation. Two men can run similar cycles and have very different hair outcomes.

Several anabolic compounds can be more hair-aggressive than testosterone itself, or can produce metabolites with strong androgen-receptor activity. But the response is not uniform. Trenbolone, for example, does not convert to DHT, yet it binds the androgen receptor very strongly and may drive noticeable scalp effects in predisposed men. Other compounds that do convert to DHT-related metabolites may produce a different pattern of shedding or miniaturization.

In practice, the lived experience often sounds like this: faster temple recession, oilier scalp, seborrhea that becomes harder to control, or a crown that seems to thin more quickly after repeated cycles. Dose stacking and repeated exposure can create a sustained supraphysiological environment, and the cumulative effect may become visible only after several cycles rather than after the first one.

That is why “I tolerated one cycle fine” is not a reliable risk assessment. Some loss is gradual and only becomes obvious once miniaturization crosses a threshold, or later when age-related susceptibility catches up. Early changes may be subtle; advanced miniaturization is much less likely to reverse.

For men making decisions in real time, the useful questions are practical: What compounds are being used? How high is the total androgenic load? How long is the exposure? Is there a family history of AGA? Is thinning already visible at the temples or crown? Those factors usually matter more than any single headline about a specific drug.

GYM CULTURE, OPTIMIZATION CULTURE, AND THE HAIR TRADE-OFF

Testosterone optimization has become more visible over the past decade, driven by interest in performance, body composition, libido, and overall vitality. For many men, these are legitimate goals. The practical question is not whether those goals are valid, but how to weigh them against potential side effects — including hair loss risk in susceptible men.

That is why the hair conversation matters. A truly informed decision about hormone use should include a clear understanding of scalp hair as part of the trade-off, especially for men with a family history of androgenetic alopecia (AGA) or early signs of miniaturization. Men deserve that information up front so they can decide what matters most to them.

PERFORMANCE

Testosterone supports muscle protein synthesis, recovery, strength, and athletic capacity. These are common reasons men seek treatment.

VITALITY & LIBIDO

Low testosterone can affect energy, mood, and sexual function. Restoring physiological levels may improve quality of life for some men.

BODY COMPOSITION

Hormonal treatment can influence fat-to-muscle ratio, which is part of its appeal for men focused on physique and metabolic health.

HAIR PRESERVATION

For genetically susceptible men, androgen exposure can accelerate AGA. Hair risk should be discussed alongside other expected benefits.

In practical terms, the decision usually comes down to priorities and baseline risk. A man may reasonably decide that the benefits of TRT outweigh the possibility of faster thinning, especially if he is willing to use hair-directed treatment. Another man may place a higher value on preserving scalp hair and choose a more conservative hormonal approach. Neither choice is inherently right or wrong.

The key is clarity: what is the goal, what is the androgenic exposure, what is the family history, and what is already visible on the scalp? Those are the questions that make the trade-off real. When men have honest information, they can make decisions that are aligned with their own values rather than discovering the downside later.

WHY SOME MEN KEEP THEIR HAIR ON TRT AND OTHERS LOSE IT FAST

This is one of the most common questions in practical discussions of TRT and hair, and it deserves a direct answer. The short version: outcomes vary because the key biological variables vary. Here are the seven main reasons.

1

BASELINE MINIATURIZATION STATUS

Men who already have subclinical follicular miniaturization before starting TRT are at higher risk of visible acceleration. Microscopic trichoscopy can detect early changes before they are obvious.

2

FAMILY HISTORY AND GENETIC SUSCEPTIBILITY

The strongest predictor. Men with a strong family history of AGA tend to have more androgen-sensitive scalp follicles, independent of baseline testosterone.

3

ANDROGEN RECEPTOR SENSITIVITY

This is genetically determined. At the same DHT exposure, follicles with higher receptor sensitivity will miniaturize more quickly. Blood testing does not measure this.

4

5-ALPHA-REDUCTASE ACTIVITY

Variation in scalp 5-AR activity affects how much DHT is generated locally. Some men convert testosterone to DHT more efficiently at the follicular level.

5

INFLAMMATORY SCALP CONDITIONS

Scalp inflammation — including seborrheic dermatitis, folliculitis, or perifollicular inflammation — can amplify androgen-related miniaturization. Treating inflammation is an important modifier.

6

CUMULATIVE PRIOR EXPOSURE

Starting TRT at 50 after decades of endogenous androgen exposure is different from starting at 30. Timing and cumulative exposure both affect baseline follicular status.

7

CONCURRENT HAIR-LOSS TREATMENT

Finasteride, dutasteride, or minoxidil may offset or reduce the incremental hair-loss risk from TRT. This is a meaningful and often underused strategy.

WOMEN AND THE ANDROGEN CONVERSATION

Women remain underrepresented in discussions of androgen-related hair loss, and that gap can lead to missed or minimized diagnoses. Female-pattern hair loss (FPHL) most often presents as diffuse thinning over the crown with relative preservation of the frontal hairline, though some women also show bitemporal recession or frontal density loss. The mechanisms are variable, but in a meaningful subset of women, androgen sensitivity appears to play a clinically important role.

KEY POINT

Androgen-related hair loss in women is real, but it is variable and should not be reduced to a single lab value.

WHAT MATTERS CLINICALLY

Total testosterone may be normal even when the scalp is responding to a higher effective androgen signal.

IMPORTANT MODIFIERS

Free androgen fraction, follicular receptor sensitivity, SHBG context, and the broader endocrine picture all matter.

The challenge in female androgen assessment is that routine blood panels often look reassuring even when hair loss is androgen-sensitive. Standard testosterone reference ranges for women are broad, so a clinically relevant androgen signal may be missed if interpretation relies only on total testosterone. Low SHBG — seen in insulin resistance, PCOS, hypothyroidism, and sometimes with certain oral contraceptive patterns — can increase the free androgen fraction even when total testosterone remains in range.

Hyperandrogenic states offer a clearer signal. Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in reproductive-age women and is associated with elevated free androgens, reduced SHBG, and, in some patients, androgen-sensitive hair thinning. Associated features such as irregular cycles, acne, increased facial or body hair, and weight-management difficulty should prompt assessment rather than dismissal, especially when hair loss is part of the presentation.

POSTPARTUM

Telogen effluvium after delivery often resolves, but it may also unmask underlying FPHL in susceptible women.

PERIMENOPAUSE

Declining estrogen without a parallel fall in androgen exposure can shift the hormonal balance toward visible thinning.

MENOPAUSE

Further changes in estrogen-androgen balance may reveal or accelerate androgen-sensitive hair loss in some women.

In practice, the most useful approach is careful pattern recognition, attention to endocrine context, and avoidance of overreliance on one number. Women with androgen-related hair loss may have normal routine labs, but that does not make the condition any less real. It means the signal is often distributed across biology rather than captured by a single test.

ATHLETIC TRAITS, FEMALE ATHLETES, AND HYPERANDROGENIC PATTERNS

This section addresses a developing area of inquiry rather than a settled conclusion. Some peer-reviewed literature suggests that selected elite female athletes — especially in strength-dominant or power-based sports — may, at a population level, show higher endogenous androgen concentrations than the general female population. This possible overlap is relevant to sport-eligibility discussions and ongoing research, but it should be framed cautiously and without overgeneralization.

> **Important framing note:** The discussion below applies to possible population-level overlap in selected elite athletic contexts. It does not imply that women who exercise, participate in sport, or pursue athletic goals have elevated hair-loss risk. General exercise participation has no established association with androgen-sensitive hair loss in women.

The relevance to hair loss is limited and conditional. In selected populations, higher endogenous androgen exposure may overlap with greater likelihood of androgen-sensitive hair changes in women who are already predisposed by follicular receptor genetics. This is a possible biological association worth assessing when other clues are present — not a deterministic rule, and not a reason to pathologize athletic identity or physical traits.

In practice, this means that a woman with androgen-sensitive hair loss who also has a strong elite athletic background and additional hyperandrogenic features — such as acne, hirsutism, or irregular cycles — may merit a more careful endocrine review than a routine panel alone might suggest. The question is not whether sport caused the hair loss, but whether the broader endocrine picture includes features that are worth assessing as part of a complete evaluation.

ELITE STRENGTH/POWER ATHLETES

Selected populations may show a population-level overlap with higher endogenous androgens. Individual variation is substantial, so this is a clue to assess carefully rather than a rule.

GENERAL EXERCISE PARTICIPATION

No established association with elevated hair-loss risk. Routine exercise, sport participation, or athletic goals are not hair-loss risk factors in women by themselves.

WHEN TO INVESTIGATE FURTHER

Consider more endocrine evaluation only when hair concerns coexist with other hyperandrogenic features such as acne, hirsutism, or irregular cycles.

WHEN HAIR LOSS IS NOT JUST ABOUT ANDROGENS

One of the most important contributions a rigorous androgen-focused guide can make is to clearly delineate where androgen biology ends and other causes begin — and to acknowledge how often these causes co-exist.

Clinicians who approach every case of hair loss as a DHT story will miss diagnoses. Patients who self-diagnose androgen-sensitive loss and self-treat without ruling out other causes may delay resolution of something more straightforwardly correctable.

Telogen effluvium — diffuse shedding triggered by physiological stress — is among the most common causes of hair loss in both men and women, and one of the most frequently confused with AGA.

A significant physical stressor (surgery, severe illness, childbirth, crash dieting, significant weight loss) typically produces noticeable shedding two to four months later, as follicles precipitously exit the growth phase.

The shedding is usually diffuse, not patterned, and often resolves spontaneously once the triggering stressor is resolved. It can, however, be superimposed on underlying AGA, producing a mixed picture that is more difficult to interpret.

TELOGEN EFFLUVIUM

Diffuse stress-triggered shedding. Often resolves spontaneously. Can overlay AGA, complicating diagnosis.

THYROID DYSFUNCTION

Both hypo- and hyperthyroidism cause hair loss. TSH, free T3, and free T4 should be included in any comprehensive assessment.

LOW FERRITIN / IRON

Underdiagnosed contributor, especially in women. Serum ferritin (not just hemoglobin) is the relevant measure. Levels below 40–70 ng/mL may impair hair cycling.

INFLAMMATORY SCALP DISEASE

Seborrheic dermatitis, lichen planopilaris, FFA — each requires specific diagnosis and management distinct from AGA treatment pathways.

NUTRITIONAL COMPROMISE

Significant caloric restriction, protein deficiency, and micronutrient depletion can drive hair shedding independent of androgen status.

MEDICATIONS

Many commonly used drugs — including anticoagulants, retinoids, antidepressants, and antihypertensives — can trigger telogen effluvium as a recognized side effect.

WHAT BLOOD TESTS MAY HELP — AND WHAT THEY CANNOT TELL YOU

Blood work has a useful supporting role in hair-loss assessment, but it has clear limits. It can uncover treatable contributors and help rule out other causes of shedding, yet it cannot diagnose androgen-sensitive hair loss on its own.

The key reason is simple: the main driver of AGA is follicular receptor sensitivity, which is genetically determined and not measurable in peripheral blood. Blood tests can refine the clinical picture, but they do not replace pattern recognition, scalp examination, or trichoscopy.

Test	What It Can Tell You	What It Cannot Tell You
Total Testosterone	Helps screen for marked hypogonadism or significant hyperandrogenism	Does not reflect follicular androgen sensitivity and will not confirm AGA
Free Testosterone / SHBG	Gives a better sense of biologically active androgen exposure	Still cannot predict how sensitive the follicles are
DHEA-S	Can suggest adrenal androgen excess, especially in women with hyperandrogenic features	An isolated elevation rarely explains hair loss without the clinical context
Estradiol (women)	Useful in perimenopausal or cycle-related assessment	A single value is only a snapshot and does not diagnose AGA
Thyroid Panel (TSH, fT3, fT4)	Helps rule out thyroid dysfunction as a cause of diffuse shedding	Normal results do not exclude AGA or other common hair-loss causes
Ferritin / Iron Studies	Identifies iron deficiency or insufficiency that can impair hair cycling	Correcting iron does not necessarily reverse hair loss if AGA is also present
CRP (selected cases)	May point to systemic inflammation that could worsen a scalp inflammatory process	It is non-specific and not a routine hair-loss test
Prolactin (selected cases)	Can identify hyperprolactinemia, which may alter gonadal hormone production	Only useful when the history suggests an endocrine cause, such as irregular cycles

For most patients with suspected AGA, the most practical blood panel includes total testosterone, free testosterone or SHBG, thyroid function, ferritin, and — in women when clinically indicated — DHEA-S and estradiol. This panel does not diagnose AGA, but it can identify modifiable factors that should be addressed alongside the main hair-loss diagnosis.

SIGNS YOUR PATTERN MAY BE ANDROGEN-DRIVEN

Formal diagnosis still requires clinical or trichoscopic assessment, but certain patterns and associated features can prompt a more targeted evaluation. These signs do not confirm androgen-sensitive loss on their own, but when they cluster — especially with a supportive family history — they make the picture more convincing.

IN MEN

- Temples start to recede in the late teens or 20s, and the hairline never fully recovers after a shed
- Frontal hairs become finer and the front band looks less dense, even if the hairline has not moved much
- The crown starts to look thinner in photos before it is obvious in the mirror
- The scalp becomes suddenly oilier, sometimes with persistent seborrheic dermatitis
- There is a significant acne history, especially if it involved the face, back, chest, or scalp
- Beard growth is dense and body hair develops quickly, suggesting strong androgen responsiveness at non-scalp follicles
- Family history of pattern hair loss on either the maternal or paternal side, especially if it appears on both sides

IN WOMEN

- The central part widens gradually, even when blood tests are reported as “normal”
- The scalp looks more see-through under bright light, especially at the crown and midline
- There is diffuse frontal density reduction or a “thin veil” appearance over the top of the scalp
- Temples thin or stay sparse after a shed, rather than returning to their prior density
- Thinning accelerates around perimenopause, often alongside a change in the estrogen-to-androgen balance
- PCOS features appear alongside hair changes — irregular cycles, acne, increased facial or body hair, or difficulty with weight
- Low SHBG on testing, even with normal total testosterone, suggests a higher free androgen fraction

A particularly important clinical observation is the simultaneous presence of miniaturizing scalp hair and increased body or facial hair in the same person. This apparent paradox — losing scalp hair while gaining body hair — is one of the most reliable markers of androgen sensitivity and reflects the region-specific receptor programming discussed earlier in this guide. In women, this pattern, especially when combined with other hyperandrogenic features, should prompt a thorough endocrine assessment rather than reassurance that “your testosterone is normal.”

WHAT ACTUALLY HELPS IF ANDROGENS ARE PART OF THE PICTURE

Identifying androgen sensitivity matters most when it changes the treatment plan. The right approach depends on the patient profile, the likely driver of hair loss, and the degree to which androgen exposure can be modified. In practice, this is less about one universal regimen and more about choosing the least-wrong strategy for the situation in front of you.

1

MEN WITH CLASSIC ANDROGEN-DRIVEN AGA

Core approach: 5-alpha-reductase inhibition with finasteride or dutasteride, usually alongside minoxidil if additional density support is needed.

Clinical emphasis: These are the most evidence-supported options for slowing miniaturization in typical male pattern hair loss. Early treatment generally works better than waiting for obvious recession or crown loss.

Practical note: Expectations should focus on stabilization and partial improvement, not full restoration.

2

MEN ON TRT WHO WANT TO PRESERVE HAIR

Core approach: Treat the hair problem directly rather than assuming TRT must simply be stopped. Finasteride or dutasteride are often the main hair-preserving tools, with minoxidil added when needed.

Clinical emphasis: TRT can unmask or accelerate thinning in predisposed men, but response is highly individual. Hair preservation requires monitoring the scalp over time, not just adjusting serum hormones.

Practical note: Decisions should be coordinated with the clinician managing testosterone, because hair goals and systemic goals may need to be balanced.

3

MEN USING OR CONSIDERING AAS

Core approach: The most effective strategy is reducing or discontinuing the androgen exposure if that is feasible. Hair-directed treatment can be layered on, but it is rarely enough to fully offset a strong AAS effect.

Clinical emphasis: AAS-related hair loss can progress quickly in genetically susceptible men, and the degree of risk varies by compound and dose. Finasteride is not a universal solution here, especially when the androgenic load is not DHT-mediated.

Practical note: Minoxidil may support growth, but it should be framed as adjunctive rather than protective enough to neutralize ongoing exposure.

4

WOMEN WITH SUSPECTED ANDROGEN-SENSITIVE THINNING

Core approach: Minoxidil remains the most broadly applicable growth-supportive option. When androgen sensitivity is convincing, spironolactone and selected hormonal strategies can be appropriate, and finasteride may be considered in carefully selected cases with strict contraception.

Clinical emphasis: The pattern is often diffuse rather than dramatic recession, and normal total testosterone does not exclude androgen sensitivity. Treatment choices should be guided by the clinical pattern, reproductive considerations, and whether there are signs of hyperandrogenism.

Practical note: The goal is usually slower progression and thicker appearance, not a guarantee of reversal.

5

MIXED CASES WHERE ANDROGENS ARE ONLY PART OF THE PICTURE

Core approach: Combine androgen-focused therapy with measures that address other contributors, especially minoxidil, scalp inflammation control, and correction of any identifiable triggers or deficiencies.

Clinical emphasis: Not every patient with shedding or thinning is predominantly androgen-driven. Telogen effluvium, inflammatory scalp disease, traction, low iron, thyroid disease, and other factors can coexist with AGA and change the treatment response.

Practical note: In these patients, the best results usually come from treating both the pattern loss and the overlay of modifiable non-androgen causes.

Across all profiles, the same caution applies: treatment should match the biology, not just the label. Hair transplantation can be useful in selected patients, but it should sit inside a long-term plan if active androgenetic progression is still ongoing. When the diagnosis is uncertain or the phenotype is mixed, the safest approach is usually stepwise treatment with close follow-up rather than aggressive one-shot intervention.

WHAT DOES NOT HELP ENOUGH ON ITS OWN

The hair-loss market is full of products that sound reassuring but have limited evidence or only modest effects. That matters because relying on weak strategies can delay treatment during the period when earlier intervention would be most useful. These are worth a clear-eyed look.

GENERIC “TESTOSTERONE BOOSTERS”

Why it is not enough: Most over-the-counter “testosterone boosters” do not produce clinically meaningful hormonal changes. And even if they did, androgen-sensitive hair loss is driven more by follicular sensitivity and DHT activity than by a low-normal testosterone number.

HAIR VITAMINS WITHOUT A PROVEN DEFICIENCY

Why it is not enough: Biotin, collagen, and broad “hair health” blends are unlikely to help if iron, zinc, and vitamin D are already adequate. Correcting a documented deficiency can matter; supplementing someone who is replete usually does little.

ASSUMING NORMAL TESTOSTERONE MEANS NO ANDROGEN RISK

Why it is not enough: A normal total testosterone level does not rule out androgen-sensitive hair loss. Follicular receptor sensitivity and local DHT conversion can still drive thinning even when standard labs look unremarkable.

RELYING ON SHAMPOO ALONE

Why it is not enough: Ketoconazole and zinc pyrithione shampoos may help inflammation and can be useful adjuncts, but their effect is modest. They are not sufficient as stand-alone treatment for active androgenetic alopecia with meaningful miniaturization.

SCALP MASSAGE ALONE

Why it is not enough: Scalp massage may improve comfort and hair care awareness, but evidence for meaningful regrowth is limited. It does not address the main biologic drivers of androgenetic alopecia.

CONTINUING HIGH-ANDROGEN EXPOSURE

Why it is not enough: In men using supraphysiologic AAS, treating the hair while maintaining the exposure creates a moving target. Hair-directed therapy may help at the margins, but it often cannot overcome ongoing androgen stress in genetically susceptible patients.

DECISION FRAMEWORKS

One of the highest-value contributions this guide can make is to translate complex androgen biology into practical, actionable decision-making frameworks. The four frameworks below are designed for the real-world scenarios most commonly encountered when androgen-related hair loss intersects with clinical decisions about hormones, performance goals, and treatment strategies.

FRAMEWORK 1: MAN WITH SUSPECTED ANDROGEN-DRIVEN AGA — NO TRT

CONFIRM TRUE MINIATURIZATION

Do not treat pattern change alone as definitive. Look for bitemporal recession, crown thinning, caliber variability, and progressive shortening of terminal hairs on trichoscopy.

ASSESS ANDROGENIC SUSCEPTIBILITY

Family history, early onset, oily scalp, and visible density loss help identify men most likely to progress. Normal labs do not rule out androgen-sensitive follicles.

START DHT-DIRECTED TREATMENT EARLY

For men with confirmed miniaturization, the decision should usually be framed around how aggressively to suppress DHT, not whether hair loss is "real."

TRACK RESPONSE OBJECTIVELY

Use standardized photos and trichoscopy, then reassess at 3 to 6 months. The goal is stabilization first, then regrowth if possible.

In practice, the key decision is whether the clinical picture justifies early intervention rather than watchful waiting. Men with high-susceptibility AGA generally benefit most when treatment begins before loss becomes visually obvious at every angle.

FRAMEWORK 2: MAN ON TRT NOTICING HAIR CHANGES

SEPARATE TRT BENEFIT FROM HAIR COST

TRT may be clinically appropriate even if it accelerates AGA, but that tradeoff should be explicit rather than accidental. Hair changes after TRT deserve active review, not reassurance alone.

DETERMINE WHETHER TRT UNMASKED SUSCEPTIBILITY

Ask whether recession, crown thinning, or increased shedding started after TRT initiation or dose escalation. That pattern strongly suggests hormone-sensitive follicles.

CONSIDER CONCURRENT HAIR PROTECTION

For men who want to remain on TRT, DHT-targeted therapy is often the most logical way to preserve hair while maintaining androgen replacement goals.

MONITOR THE SCALP LIKE A TREATMENT OUTCOME

Serial photos, trichoscopy, and a clear follow-up interval are essential. If loss progresses despite intervention, the next step is usually to revisit TRT dose, formulation, or hair strategy.

For men on TRT, the decision is rarely "hair treatment or testosterone." It is usually a three-part question: is TRT necessary, is the scalp showing androgen sensitivity, and what combination best preserves both goals?

FRAMEWORK 3: WOMAN WITH SUSPECTED ANDROGEN-SENSITIVE THINNING

1. LOOK FOR THE CLINICAL PATTERN

Common clues include widening of the central part, reduced frontal or crown density, miniaturized hairs on dermoscopy, and preserved hairline with diffuse thinning rather than classic male-pattern recession.

2. LOOK FOR ANDROGEN CLUES OUTSIDE THE SCALP

Persistent acne, oily skin or scalp, increased facial/body hair, irregular cycles, infertility history, or prior PCOS diagnosis all increase suspicion for androgen-sensitive thinning.

3. CHECK FOR COMMON ENDOCRINE CONTRIBUTORS

Useful testing often includes total testosterone, free testosterone or calculated free androgen index, SHBG, DHEAS, prolactin when indicated, and thyroid studies. Consider ferritin, vitamin D, zinc, and CBC if diffuse shedding is also present.

4. INTERPRET LABS IN CONTEXT

Normal androgen labs do not exclude female pattern hair loss or androgen sensitivity. The diagnosis is usually clinical, supported by pattern recognition and trichoscopy rather than a single abnormal value.

5. MATCH TREATMENT TO THE UNDERLYING DRIVER

If the pattern fits androgen-sensitive thinning, consider hair-directed therapy and, when appropriate, gynecologic or endocrine management for PCOS, hyperandrogenism, or perimenopausal transition.

The practical question in women is not simply whether androgens are "high," but whether the scalp is behaving like an androgen-sensitive organ. That distinction determines whether the workup should stay cosmetic or expand into endocrine evaluation and targeted treatment.

FRAMEWORK 4: YOUNGER ADULT / EARLY-ONSET RISK

RECOGNIZE WHY EARLY ONSET MATTERS

Hair loss in the late teens or 20s often signals heavier genetic loading and a longer time horizon for progression. Even modest thinning can become major loss if it is left unaddressed for years.

CONFIRM THAT THIS IS TRUE AGA

Young adults are frequently told to "wait and see." A better approach is early trichoscopy to document miniaturization and distinguish AGA from shedding, hair styling damage, or temporary stress-related loss.

TREAT THE WINDOW, NOT JUST THE SYMPTOM

The most important advantage of early treatment is preserving follicles before they become severely miniaturized. For high-risk young adults, stabilization now is often more valuable than trying to recover lost density later.

PLAN FOR LONG-TERM ADHERENCE

Young patients need a strategy they can sustain for years. That means discussing expectations, side effects, follow-up timing, and the difference between maintenance, partial recovery, and ongoing progression.

Early-onset AGA is one of the clearest situations where delay has a cost. If miniaturization is present, the decision should be framed around long-term follicular preservation, not around whether the loss is severe enough to justify action today.

CLINICAL TAKEAWAY

The most useful framework across all four scenarios is the same: identify the pattern early, define the androgen context, decide whether the scalp is showing true susceptibility, and then match treatment intensity to the patient's goals and risk profile. The earlier that decision is made, the more hair can usually be preserved.

KEY TAKEAWAYS

The core arguments of this guide can be distilled into six evidence-based principles. These are not simplifications — they are the conclusions that a careful reading of the androgen biology and clinical literature supports, stated clearly enough to be immediately actionable.

AGA REFLECTS SENSITIVITY PLUS EXPOSURE

Androgenetic alopecia is driven by inherited follicular sensitivity and cumulative androgen exposure over time, not testosterone level alone.

DHT IS THE CENTRAL DRIVER IN CLASSIC PATTERN LOSS

Scalp DHT, generated through 5-alpha-reductase, is the key mediator of follicular miniaturization in classic AGA.

TRT CAN ACCELERATE LOSS IN PREDISPOSED MEN

Testosterone replacement increases androgen substrate and can speed AGA in men with clear genetic susceptibility.

ANABOLIC STEROID EXPOSURE RAISES RISK SHARPLY

Supraphysiologic androgen cycles create far greater hair-loss pressure than physiologic TRT, and some loss may be irreversible.

WOMEN CAN HAVE ANDROGEN-SENSITIVE HAIR LOSS

A meaningful subset of women develop androgen-sensitive thinning, especially with PCOS, low SHBG, perimenopause, or hyperandrogenic features.

MIXED PICTURES ARE COMMON AND REQUIRE FULL ASSESSMENT

Telogen effluvium, thyroid disease, iron deficiency, inflammatory scalp disease, and medication effects often coexist with or mimic AGA.

Each principle stands on its own, but the practical message is the same: identify androgen sensitivity early, distinguish it from other causes of shedding, and match treatment intensity to the biology and the patient's goals.

FREQUENTLY ASKED QUESTIONS

DOES HIGH TESTOSTERONE CAUSE HAIR LOSS?

Not by itself. High testosterone provides more substrate for DHT conversion, but whether that becomes hair loss depends much more on follicular androgen receptor sensitivity — which is genetically encoded — and 5-alpha-reductase activity in scalp tissue. Many men with high-normal or elevated testosterone keep their hair for life because they do not have the susceptibility that makes follicles respond strongly to the androgenic signal. Conversely, some men with entirely normal or even below-average testosterone develop clear androgenetic alopecia because their follicles are highly sensitive to modest DHT exposure.

CAN I DO TRT WITHOUT WRECKING MY HAIR?

Sometimes, yes — but not with a guarantee. TRT raises circulating testosterone and can increase DHT production, which may accelerate miniaturization in men who are already genetically predisposed. The real-world risk depends on how much miniaturization is already present, family history, follicular sensitivity, 5-alpha-reductase activity, dose, and whether hair-loss treatment is also being used. TRT does not universally cause hair loss; in some men it unmasks or speeds up AGA, while in others it produces little or no visible scalp change.

WHAT'S THE DIFFERENCE BETWEEN TRT AND STEROID USE FOR HAIR RISK?

TRT and anabolic steroid use are not the same exposure level. TRT aims to restore physiologic testosterone levels, while anabolic steroid cycles often create supraphysiologic androgen exposure. For hair, that usually means TRT may accelerate loss in susceptible men, but steroid use can place far greater pressure on the follicles and may trigger more rapid, more extensive, and sometimes less reversible loss. The difference matters because dose, duration, and androgen burden all influence scalp risk.

IF STEROIDS TRIGGERED MY HAIR LOSS, IS ANY OF IT REVERSIBLE?

Sometimes partially, but not always completely. If the shedding was mainly a temporary stress response or early miniaturization that has not become fixed, some recovery may occur after stopping the androgen exposure. If a follicle has already undergone significant miniaturization, regrowth is less predictable and some loss may be permanent. The earlier the exposure is recognized and the faster it is addressed, the better the odds of preserving or recovering density.

IS DHT REALLY MORE IMPORTANT THAN TESTOSTERONE?

At the follicular level, yes. DHT binds the androgen receptor with substantially higher affinity than testosterone, making it the main androgenic mediator of classic pattern hair loss. That is why 5-alpha-reductase inhibitors, which lower DHT without dramatically lowering testosterone, can slow or halt AGA progression. Testosterone still matters, but mostly as the upstream precursor to DHT and as a contributor in some androgen-sensitive contexts.

WHY DID MY HAIR GET WORSE EVEN THOUGH MY BLOODS LOOKED NORMAL?

Because standard bloodwork can miss the biology that actually drives hair loss. Total testosterone can look normal while free androgen exposure is effectively higher because of low SHBG, or because the follicles are unusually sensitive to androgen signaling. In women in particular, many people with androgen-sensitive hair loss remain within “normal” lab ranges. Hair loss is a tissue-level problem, not just a serum-number problem, so clinical pattern, family history, and trichoscopy matter as much as blood tests.

CAN WOMEN HAVE ANDROGEN-RELATED HAIR LOSS WITHOUT OBVIOUS HORMONAL SYMPTOMS?

Yes. This is common and often underrecognized. Women can develop androgen-sensitive hair thinning even without acne, hirsutism, cycle changes, or clearly abnormal total testosterone. What matters is the free androgen fraction, often influenced by SHBG, plus follicular receptor sensitivity. That means a woman can have androgen-related hair loss while her standard labs look unimpressive and her symptoms outside the scalp are minimal.

WHICH BLOOD TESTS ARE ACTUALLY WORTH CHECKING?

For most patients with suspected hair loss, a practical starting panel includes total testosterone, SHBG so free testosterone can be estimated, thyroid function (TSH and free T4), and ferritin. In women with hyperandrogenic features, DHEA-S and estradiol are often useful additions. CRP and prolactin are best reserved for selected clinical scenarios rather than routine screening. No blood panel alone can confirm or exclude androgen-sensitive AGA, so results should always be interpreted alongside the hair-loss pattern, family history, and trichoscopic findings.

Across all of these questions, the same principle applies: hair loss risk is shaped by androgen exposure, follicular sensitivity, and the presence of other shedding triggers — not by one lab value alone.

STILL UNSURE WHETHER ANDROGENS ARE PART OF YOUR HAIR LOSS PICTURE?

> A structured assessment can help distinguish androgen-driven miniaturization from diffuse shedding, inflammatory scalp conditions, or a mixed pattern — and clarify which evidence-based next steps are most appropriate for your biology and goals.

Hair loss is not a single condition with a single mechanism, and androgens — while central in many cases — are only one part of a thorough evaluation. Determining whether DHT-mediated miniaturization is driving your presentation, or whether telogen effluvium, scalp inflammation, nutritional factors, or a genuinely mixed picture is contributing, changes what is most likely to help and what is not.

The androgen index framework in this guide is designed as a structured starting point: integrating family history, pattern characteristics, scalp behavior, endocrine context, and exposure history into a coherent clinical picture. Applying it well to your individual situation, however, requires the kind of personalized assessment that no article — however detailed — can replace. Pattern recognition, trichoscopic evaluation, and targeted investigation are where the biology becomes a clear diagnosis and a practical plan.

PATTERN ASSESSMENT

Differentiate true miniaturization from diffuse shedding, map distribution, and assess caliber variation — the clinical foundation for any treatment decision.

ANDROGEN INDEX REVIEW

Review the full androgen-relevant context — not just a hormone panel, but the broader clinical picture that shapes risk and response.

TARGETED INVESTIGATION

Use a focused blood workup to answer the specific questions raised by your presentation — rather than relying on a generic screen.

EVIDENCE-BASED PLAN

Translate the findings into treatment recommendations aligned with the evidence, your pattern, and your goals, with clear expectations and monitoring.

Whether you are a man considering TRT, a woman trying to understand whether androgens are relevant to your hair changes, or a clinician looking for a more structured framework for the androgen-hair conversation, our aim at Hair Longevity Institute is the same: to help you leave with a more complete, nuanced, and actionable understanding than most resources provide. The biology is complex, but it is understandable — and when it is understood well, it can be addressed with far greater precision.