THE STUB CLUB PROTOCOL

TIER 3

THE ANDROGENIC FINISHER

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CRITICAL PREREQUISITE: Tier 3 is not a standalone protocol. It is the third layer of a three-part system. If you have not completed Tier 1 (minimum 90 days) and Tier 2 (minimum 4-6 months), stop here and complete those first. The compounds in Tier 3 are androgenic and dopaminergic signal amplifiers. They amplify whatever signal the system is currently producing. On a rebuilt, functional vascular and hormonal foundation, they produce the intended effects. On an uncorrected, inflamed, uncoupled system, they amplify dysfunction. Men who have used Tier 3 compounds without the foundational tiers report some of the worst outcomes in the men's health coaching literature.

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Introduction: Signal Mastery — The Final Layer

Tier 1 stopped the internal rust. Tier 2 rebuilt the pipes. Tier 3 masters the signal.

The analogy that runs through this entire protocol is engineering. You cannot install a high-performance engine in a rusted car with collapsed fuel lines. Tier 1 cleared the rust. Tier 2 rebuilt the fuel delivery system. Tier 3 is the engine upgrade — but it only works because the fuel lines are now capable of supplying it.

Signal Mastery means two things simultaneously: maximising the androgenic signal that the now-functional vascular and hormonal system can produce and respond to, and ensuring that the signal is received correctly — by an androgen receptor that is sensitive, by a dopamine receptor that is responding, by a 5-alpha-reductase enzyme that is converting, and by a nervous system that is no longer suppressing the entire cascade with chronic cortisol and sympathetic overdrive.

> *"Total testosterone is not the signal. DHT is the signal. And DHT is only the signal if the androgen receptor is listening. Tier 3 addresses all three: the production, the conversion, and the reception."*

This book is the most complex of the three Stub Club volumes. It covers the androgen receptor framework in depth — including the critical distinction between AR polymorphism and AR silencing, which determines the entire treatment direction. It covers the DHT ecosystem: 5-alpha-reductase upregulation, the T3 thyroid connection, the IGF-1 feed-forward loop. It covers the dopaminergic amplification layer — Tribulus, Mucuna, the cycling protocol. It covers neurosteroids and the GABA-allopregnanolone axis that determines whether the brain can receive the androgenic signal without sympathetic override. And it covers the advanced options: Vitamin D3 optimisation, zinc, boron, the sauna-cold plunge protocol, and the specific cases where medical intervention (HCG, TRT, Proviron) becomes the appropriate next step.

Read this entire book before beginning the protocol. The sequencing and the cycling rules in Tier 3 are more nuanced than in Tiers 1 and 2. Getting them wrong produces receptor downregulation and hormonal instability. Getting them right produces the compound effect of all three tiers operating together.

Chapter 1: The Androgen Receptor — The Missing Variable

The most common error in men's hormonal optimisation is treating testosterone as the endpoint. It is not. Testosterone is the raw material. DHT is the signal. The androgen receptor is the receiver. And the receiver can be broken in ways that no amount of testosterone — or DHT — can fix if the underlying receptor problem is not diagnosed correctly.

What the Androgen Receptor Actually Does

The androgen receptor (AR) is a nuclear transcription factor — not a cell-surface receptor, not a hormone, but a protein that binds androgens (primarily DHT, then testosterone) and then translocates into the cell nucleus to directly regulate gene expression. This is the genomic pathway: AR + DHT → nuclear translocation → DNA binding → gene transcription changes.

There is also a faster non-genomic pathway: DHT activating the AR at the cell membrane triggers PI3K/Akt signalling, which activates eNOS and produces cGMP — the immediate vasodilatory signal for erection. This is why DHT (not testosterone, not estrogen) is the primary androgen for erectile function: it operates on both the genomic timescale (weeks to months of structural changes in penile tissue) and the non-genomic timescale (minutes, for the acute eNOS-cGMP vascular response).

A man can have total testosterone of 1000 ng/dL and DHT of 80 ng/dL and experience the symptoms of a man with testosterone of 200 ng/dL — if his androgen receptor is not functioning correctly. This is "Type 3" hypogonadism: normal or high hormones, broken signal reception. It is extremely common, rarely diagnosed, and the primary target of Tier 3.

AR Density, CAG Repeats, and Sensitivity — The Variables You Can and Cannot Change

The androgen receptor gene contains a polymorphic region of CAG (cytosine-adenine-guanine) trinucleotide repeats in exon 1. The number of CAG repeats is inversely correlated with AR transcriptional activity — more repeats means less AR activity per unit of DHT bound. CAG repeat length is genetically fixed, maternally inherited, and cannot be changed.

What can be changed is AR sensitivity and signalling efficiency — the modifiable overlay on top of the genetic baseline:

- Inflammation and LPS endotoxin: directly reduce AR expression at the transcriptional level via NF-kB activation, which suppresses AR gene transcription. This is why Tier 1 (gut health, LPS reduction) is prerequisite to Tier 3 — without it, the AR is being continuously suppressed by the same inflammatory inputs that were addressed in the foundational tiers.

- Cortisol: glucocorticoid receptor and androgen receptor compete for shared coactivator proteins (SRC-1, SRC-2). Chronic cortisol elevation reduces the availability of these coactivators for the AR, functionally reducing AR transcriptional output even when DHT levels are adequate.
- Estradiol excess: excess estradiol promotes a shift from AR-mediated to ER-mediated gene regulation in androgen-sensitive tissues, reducing the biological impact of DHT in those tissues. Managing estradiol to the 20-30 pg/mL range is therefore as important for AR sensitivity as managing DHT itself.
- Prolactin: as covered in Tier 1, prolactin blocks D2 receptors and 5-AR activity. It also directly reduces AR sensitivity in penile smooth muscle — the most DHT-sensitive tissue in the male body. Prolactin normalisation is foundational for AR sensitivity.
- Mitochondrial ATP: the AR genomic pathway requires the cell to be in an anabolic, energy-replete state to activate the gene expression programmes that DHT drives. Mitochondrial dysfunction reduces ATP availability, reducing AR transcriptional output even when the receptor is structurally normal. This is the Tier 1 mitochondrial connection to Tier 3 function.

The Critical Distinction: AR Polymorphism vs. AR Silencing

This distinction determines the entire treatment direction in Tier 3. Getting it wrong means years of misapplied interventions.

AR polymorphism (CAG-repeat-driven reduced sensitivity): the AR gene is structurally intact and transcriptionally active, but the CAG repeat length produces lower-than-average transcriptional output per unit of DHT. The AR responds to androgen — it just responds less efficiently. The treatment direction is increasing DHT and DHT-like androgen signalling to compensate for the reduced per-unit efficiency.

AR silencing (epigenetic gene suppression): the AR gene is structurally intact but epigenetically silenced — typically by DNMT1-mediated DNA methylation of the AR gene promoter region. The AR does not respond to androgen at any dose. Adding more DHT, taking Proviron, or using TRT will not produce the expected response — and in some cases, additional androgen loading into a silenced-AR system causes downstream negative feedback effects that worsen the situation.

> *"You cannot supplement your way past a silenced receptor. If you are applying androgens to a silenced AR, you are not enhancing the signal. You are creating hormonal noise that the body then has to process without the normal AR-mediated feedback regulation."*

How to distinguish them:

- CAG-repeat polymorphism: responds to DHT-type interventions (Proviron, DHT cream, high-protodioscin Tribulus). Symptoms improve with androgen optimisation. DUTCH test shows normal or high DHT with normal to low 3-

alpha-androstanediol-glucuronide (3-alpha-diol-G), suggesting reduced DHT conversion through the 3-HSD pathway rather than AR activation failure.

- AR silencing: does not respond to androgen interventions. Men who have tried multiple DHT-boosting approaches, TRT, or even exogenous DHT cream without benefit. DUTCH test shows low 3-alpha-diol-G despite normal DHT, suggesting the DHT is not being used — not converted by the AR-mediated 3-alpha-HSD pathway because the AR is not driving the downstream gene expression.
- The epigenetic fix: HDAC inhibitors de-methylate and reactivate silenced genes. The preferred natural HDAC inhibitor for AR reactivation is sodium butyrate (300-600mg daily) — the same compound that supports gut barrier integrity. A clinical coaching case cited months of sodium butyrate use before meaningful AR reactivation became measurable. Niacinamide is a second-line option. Sodium valproate is explicitly not recommended — it is a mitochondrial toxin and is difficult to discontinue.

Levers for AR Sensitivity That Are Available to Every Man

Regardless of whether AR polymorphism or AR silencing is the primary issue, the following interventions improve AR sensitivity and signalling efficiency through mechanisms available to every man in Tier 3:

- L-carnitine (food-sourced): carnitine increases AR density in muscle and reproductive tissue via a mechanism involving acetyl-CoA and chromatin remodelling near the AR gene. The most bioavailable source is dietary, not supplemental — lamb contains the highest carnitine concentration of commonly available meats, followed by beef, then chicken. The TMAO cardiovascular risk from supplemental carnitine is not present with food-sourced carnitine when gut microbiome health is addressed (Tier 1). Supplemental L-carnitine is less preferred specifically because the TMAO risk is mediated by gut dysbiosis converting supplemental carnitine to TMAO; food sources do not produce the same conversion pattern.
- Sauna + cold plunge: heat shock proteins (HSP70, HSP90) are AR chaperone proteins — they maintain the AR in its correctly folded, ligand-competent conformation. Sauna exposure strongly induces HSP expression. Immediate cold plunge after sauna prevents the prolactin spike that sauna alone can cause. The sequence is critical: sauna first (15-20 minutes at 80-100°C), then cold plunge immediately (not delayed). Cold plunge alone does not produce the prolactin spike; the heat-induced prolactin spike occurs in the recovery period, which the cold plunge interrupts.
- Tribulus terrestris (Bulgarian, ≥50% protodioscin): the primary natural AR sensitiser in Tier 3. The protodioscin fraction of Tribulus does not raise total testosterone significantly, but it raises DHT and DHEA, lowers prolactin (by up to 70% in some research), and directly supports AR sensitivity via coactivator

recruitment at the N-terminal domain. Sourcing is the dominant variable — Bulgarian or Slovak-sourced Tribulus with ≥50% protodioscin standardisation is a categorically different product from Chinese or Indian-sourced Tribulus with generic saponin standardisation. Most commercial products are the latter.

- Vitamin D3 with K2: Vitamin D receptors (VDR) and androgen receptors interact at the nuclear level — VDR and AR share coactivator proteins, and adequate Vitamin D status supports AR transcriptional output via this shared coactivator pool. Target serum 25(OH)D of 50-70 ng/mL. Vitamin K2 (as MK-7, 100mcg daily) is required to direct calcium mobilised by Vitamin D3 away from arteries and toward bone — without K2, high-dose D3 supplementation can promote arterial calcification.
- Zinc: a direct 5-AR cofactor and an indirect AR sensitiser via reduction of aromatase activity (which lowers estradiol, improving the DHT:E2 ratio and the AR:ER competition described above). Target 15-25mg daily as zinc glycinate or picolinate. Zinc repletion from a deficient state can take up to 6 months to reach full tissue-level sufficiency — this is why zinc is introduced in Tier 1 and continued in Tier 3.
- Boron: 10mg daily. Boron inhibits sex hormone-binding globulin (SHBG), increasing free testosterone and free DHT available for AR binding. It also reduces aromatase activity and has demonstrated prolactin-lowering effects. Under-studied and underutilised relative to its clinical utility.

Chapter 2: DHT Mastery — The 5-AR Upregulation Protocol

DHT is the king androgen. It binds the androgen receptor with 3-10 times the affinity of testosterone. It is the primary driver of penile tissue health, erection quality, sexual sensitivity, and the neurosteroid pathway that produces the GABAergic molecules that reduce anxiety and support desire. And most men optimising for testosterone are completely ignoring it.

The 5-Alpha-Reductase System — Full Mechanistic Picture

5-alpha-reductase converts testosterone to DHT in a NADPH-dependent reaction. There are two primary isoforms:

- Type 1 (SRD5A1): expressed primarily in the liver, skin, brain, and scalp. Contributes to systemic DHT production, neurosteroid synthesis (via allopregnanolone production from progesterone), and sebaceous gland DHT. Less sensitive to finasteride.
- Type 2 (SRD5A2): expressed primarily in the prostate, seminal vesicles, epididymis, and genital skin. This is the primary isoform for penile tissue DHT production and is the target of finasteride. Dutasteride blocks both isoforms.

The drivers of 5-AR activity, in order of potency:

- Free T3 (active thyroid hormone): the single most potent driver of 5-AR activity. T3 upregulates SRD5A1 and SRD5A2 gene expression directly. This is why the keto/carnivore-induced T3 crash produces low DHT despite normal testosterone — the enzyme is underexpressed because T3 is inadequate. Hepatic T4-to-T3 conversion requires glucose (carbohydrates). The minimum carbohydrate threshold for optimal T3 is approximately 100-150g per day.
- IGF-1: the second most potent driver. IGF-1 and DHT form a feed-forward loop — IGF-1 upregulates 5-AR, which increases DHT, which feeds back to support IGF-1 production. Low IGF-1 is therefore both a cause and a consequence of low DHT. Breaking this cycle requires IGF-1 optimisation independently — via sleep (80% of GH/IGF-1 is released during deep sleep), resistance training, and adequate dietary protein.
- DHT itself: positive feedback loop. Higher DHT drives higher 5-AR expression. Lower DHT reduces 5-AR expression. This is the self-reinforcing nature of the low-DHT state — once established, it tends to persist without deliberate intervention.

What blocks 5-AR (in addition to the pharmaceutical inhibitors):

- Epigenetic silencing via DNMT1: finasteride has been shown to cause DNMT1-mediated methylation of the SRD5A2 promoter region, silencing the gene itself. This is the mechanism by which some men who used finasteride continue to have impaired DHT conversion years after stopping — the enzyme is not just

blocked, it is epigenetically turned off. The fix is HDAC inhibitor support (sodium butyrate, niacinamide) to de-methylate the promoter. DHT itself has been shown to reduce DNMT1 activity, creating a partial self-correcting mechanism in non-silenced individuals.

- Serotonin via 5-HT2A: serotonin binding to 5-HT2A receptors has been shown to reduce 5-AR mRNA expression. This is one mechanism by which SSRIs produce DHT reduction and contribute to post-SSRI sexual dysfunction. Interestingly, 5-HT2A activation (not serotonin generally) is one of the rare serotonin effects that can raise 5-AR mRNA — making serotonin's net effect on 5-AR contextual and receptor-specific.
- Chronic sympathetic overdrive: chronic cortisol elevation reduces 5-AR activity via the glucocorticoid-androgen receptor competition mechanism described in Chapter 1.

The IGF-1 Optimisation Protocol

IGF-1 is the most overlooked variable in DHT optimisation and one of the most impactful levers available in Tier 3. Target is ≥ 200 ng/mL. The natural tools for raising IGF-1 from the ground up:

- Sleep: 80% of daily GH secretion occurs during slow-wave sleep (stages 3 and 4). GH drives IGF-1 production in the liver. A man who is not getting adequate REM and slow-wave sleep is not producing adequate GH, is not driving adequate IGF-1, and is not supporting the 5-AR activity that IGF-1 enables. Sleep is the single most impactful IGF-1 intervention and the most commonly neglected.
- Resistance training — compound movements: heavy compound lifts (squat, deadlift, press, row) produce the most significant acute GH and IGF-1 pulses of any exercise modality. The stimulus must be sufficient — sub-maximal, low-load, high-repetition training does not produce the same GH pulse as high-load, low-to-moderate repetition training. Training frequency: 3-4 sessions per week, with at least 48 hours between training sessions for the same muscle groups.
- Fasting: even 16-24 hours of fasting produces a 200-300% increase in GH pulsatility, which drives a subsequent IGF-1 rise on re-feeding. This is the biological basis for the popular intermittent fasting practice. However, extended fasting (beyond 48 hours) begins to reduce IGF-1 as the body prioritises catabolism over anabolic signalling.
- Dietary protein: IGF-1 production is protein-dependent. Leucine is the primary amino acid that stimulates hepatic IGF-1 production via mTOR. Target protein intake of ≥ 1.6g/kg bodyweight daily, with leucine-rich sources (whey protein, eggs, meat) at each meal.

- Magnesium: magnesium deficiency reduces GH pulse amplitude. This is one more reason why magnesium malate from Tier 1 is foundational to Tier 3 function — not just for cortisol and ATP, but for GH-IGF-1 axis support.
- Vitamin D3: Vitamin D directly stimulates IGF-1 production and upregulates the IGF-1 receptor in multiple tissue types. Men with Vitamin D deficiency consistently show lower IGF-1, which normalises with Vitamin D repletion.

The T3-DHT Connection: Managing the Thyroid Variable

Free T3 is the primary upstream driver of 5-AR activity and is the variable most often unaddressed in DHT optimisation protocols. The connection operates via thyroid hormone response elements in the promoters of both SRD5A1 and SRD5A2 — T3 directly upregulates the transcription of both 5-AR isoforms.

Subclinical hypothyroidism — normal TSH, normal T4, low-normal or low Free T3 — is the pattern most frequently missed. TSH alone is an inadequate assessment. Free T3 must be measured directly. The target is the upper third of the reference range.

Factors that impair T4-to-T3 conversion:

- Carbohydrate restriction: the hepatic deiodinase type 1 enzyme that converts T4 to T3 requires glucose. Low-carbohydrate diets consistently reduce Free T3 in studies. The minimum safe carbohydrate threshold is 100-150g per day.
- Selenium deficiency: selenocysteine is the active site of deiodinase enzymes. Selenium deficiency directly impairs T4-T3 conversion. Target 150-200mcg selenium daily as selenomethionine.
- Chronic inflammation: IL-6 and TNF-alpha reduce deiodinase activity. This is why Tier 1 anti-inflammatory work is prerequisite to Tier 3 thyroid optimisation — the inflammation addressed in Tier 1 is the same inflammation impairing T3 production.
- Elevated Reverse T3 (RT3): in high-stress states, the body preferentially converts T4 to Reverse T3 (an inactive isomer of T3) rather than active T3. RT3 competes with T3 for receptor binding, functionally reducing thyroid signalling even when Free T3 appears adequate. Test RT3 if Free T3 is low-normal despite adequate T4.

  Practical action: Test Free T3, Free T4, Reverse T3, and selenium at the start of Tier 3. If Free T3 is in the lower half of the reference range, address selenium (150-200mcg/day), carbohydrate intake (minimum 150g/day from clean sources), and inflammation burden before attempting androgenic amplification. Raising testosterone without Free T3 in the upper range is working against the 5-AR enzyme's primary driver.

Chapter 3: The Dopaminergic Amplification Layer

Erection begins in the brain. Specifically, it begins with a dopaminergic signal from the mesolimbic pathway that initiates the parasympathetic cascade leading to penile smooth muscle relaxation and blood flow. No matter how well-rebuilt the endothelium is or how optimal the DHT levels are, if the dopaminergic initiation signal is absent or blunted, the cascade does not start.

Tier 3's dopaminergic layer addresses this signal initiation — the compounds that sensitise D2 receptors, lower prolactin, support dopamine precursor availability, and cycle the botanical androgens in the rotation that prevents receptor downregulation.

Prolactin: The Master Dopamine Brake (Final Reset)

By Tier 3, Prolactin should be in the 4-7 ng/mL range from the Tier 1 interventions (P5P, Tribulus, sleep improvement, alcohol elimination, gut repair). If it is not, Tier 3 will work against a continuous D2 blockade that limits the effectiveness of every dopaminergic compound in this chapter.

Verify Prolactin before beginning Tier 3. If it is above 10 ng/mL, Tier 1 is incomplete — either the gut LPS source has not been adequately addressed, alcohol is still being consumed regularly, or the P5P and Tribulus protocol has not been run for sufficient duration (8-12 weeks minimum for meaningful prolactin reduction from natural interventions).

If Prolactin is confirmed below 10 ng/mL (ideally 4-7), proceed. The D2 receptors are now capable of responding to the dopaminergic compounds in this chapter.

Tribulus Terrestris — The Cornerstone of Tier 3

Tribulus has been mentioned in previous chapters, but Tier 3 is where it becomes the primary cycling botanical. Its role here is not merely supplementary — it is the central AR-sensitising, prolactin-lowering, DHT-raising compound around which the Tier 3 cycle is structured.

The mechanism of protodioscin in Tribulus:

- DHT elevation via DHEA pathway: protodioscin stimulates the conversion of DHEA to androstenedione and then to DHT via the DHEA pathway — the backdoor route to DHT that does not require testosterone as an intermediate. This is why Tribulus raises DHT and DHEA without proportionally raising total testosterone in most research.
- Prolactin reduction: the protodioscin fraction has demonstrated direct prolactin-lowering effects of up to 70% in some clinical observations. The mechanism involves stimulation of dopaminergic tone in the tuberoinfundibular pathway — the same pathway where P5P exerts its prolactin-lowering effect. The two compounds are synergistic in prolactin reduction when cycled correctly.

- AR coactivator recruitment: protodioscin acts as an AR coactivator via the N-terminal domain of the receptor — it does not bind the AR directly, but enhances the transcriptional efficiency of DHT already bound to the receptor. This is the "amplifier" function: the same amount of DHT produces greater transcriptional output when protodioscin is present.
- Adaptogenic cortisol modulation: high-quality standardised Tribulus demonstrates cortisol-balancing activity in some research — not just reducing cortisol, but normalising the cortisol circadian rhythm. This indirectly supports AR sensitivity via reduced glucocorticoid-coactivator competition.

The sourcing requirement cannot be overstated. Bulgarian Tribulus standardised to ≥50% protodioscin (ideally 60-90%) from Eastern European sources (Bulgaria, Slovakia) is a fundamentally different product from the vast majority of commercial Tribulus products, which are standardised to total saponins from Chinese or Indian sources with very low protodioscin content. In independent testing, many "Bulgarian Tribulus" products labelled on the high street do not meet the protodioscin content claimed.

> **Cycling rule:** Tribulus must be cycled: 5 weeks on, 2 weeks off minimum. Extended continuous use without cycling produces receptor desensitisation to the prolactin-lowering and DHT-supporting effects. The cycling break is not optional — it is when receptor sensitivity is restored for the next cycle.

Mucuna Pruriens — The Dopamine Precursor

Mucuna pruriens is a leguminous plant whose seeds contain L-DOPA (levodopa) at concentrations of 3-6% by weight in raw form, or up to 15-20% in standardised extracts. L-DOPA is the immediate metabolic precursor to dopamine — it is what the body makes from tyrosine before converting it to dopamine in the dopaminergic neurons.

Mucuna is the only significant dietary/supplemental source of direct dopamine precursor. Unlike tyrosine supplementation (which provides a further-upstream precursor), L-DOPA from Mucuna bypasses the tyrosine hydroxylase step — the rate-limiting, BH4-dependent step in dopamine synthesis — and enters the dopamine synthesis pathway directly.

Relevant effects for Tier 3:

- Dopamine elevation: Mucuna reliably increases dopamine synthesis and turnover, producing effects on mood, motivation, and libido that are more rapid than tyrosine or phenylalanine supplementation.
- Prolactin suppression: dopamine is the primary physiological inhibitor of prolactin secretion. By raising dopamine tone, Mucuna produces additional prolactin-lowering effects that complement the P5P and Tribulus mechanisms.

- LH and testosterone support: elevated dopamine tone stimulates GnRH pulsatility from the hypothalamus, increasing LH secretion and downstream testosterone production.
- Sperm quality: human research with Mucuna has demonstrated significant improvements in sperm count, motility, and morphology — reflecting the broad androgenic and dopaminergic restoration effects of the compound.

Dosing and cycling: Mucuna is a powerful dopaminergic compound and must be cycled more aggressively than Tribulus. The dopamine precursor load can produce receptor sensitisation followed by desensitisation if used continuously. Cycling protocol: 3 weeks on, 2 weeks off. Dose: 300-500mg of a standardised extract (15% L-DOPA content) daily, taken in the morning.

Critical caution: Mucuna contains L-DOPA, which is absolutely contraindicated in combination with MAO inhibitors (including pharmaceutical MAOIs, and to a lesser degree with St. John's Wort, Rhodiola rosea, and selegiline). The combination risks serotonin syndrome or hypertensive crisis. Do not combine Mucuna with any MAO-inhibiting compound.

A second caution: in men with a history of SSRI use (PSSD-adjacent profiles), the rapid dopamine precursor load from Mucuna can trigger the 5-HT2C brake described in The Confidence Reset Book I — serotonin and dopamine are in dynamic balance, and a sharp dopamine increase can trigger a counter-regulatory serotonergic response. Start at a low dose (150mg) and titrate up over two weeks if this is your history.

Catuaba and Muira Puama — The Synergistic Pair

These two Amazonian botanicals are among the most consistently reported natural aphrodisiacs in the clinical coaching literature and in the ethnomedical tradition that preceded modern research. They are presented together because their mechanisms are complementary and because they are most effectively used as a pair.

Catuaba (Trichilia catigua) mechanisms:

- Dopamine and norepinephrine reuptake inhibition: the yohimbine-related alkaloids in Catuaba inhibit the reuptake of both dopamine and norepinephrine at the synapse, producing a mild DAT (dopamine transporter) inhibitory effect. Unlike pharmaceutical DAT inhibitors, the effect is mild and does not produce the tolerance or addiction risk of stimulant drugs.
- 5-HT2 receptor modulation: Catuaba alkaloids have demonstrated 5-HT2 receptor antagonist activity — directly addressing the serotonin-dopamine imbalance that suppresses sexual response in men with elevated 5-HT2C tone.
- Neuroprotective and anti-neuroinflammatory effects: Catuaba extracts reduce microglial activation and neuroinflammation, which is particularly relevant for men with post-SSRI or post-finasteride dopaminergic blunting.

Muira Puama (Ptychopetalum olacoides) mechanisms:

- Acetylcholinesterase inhibition: Muira Puama inhibits the enzyme that breaks down acetylcholine, raising cholinergic tone. As established in The Confidence Reset Book I, acetylcholine is the primary driver of the parasympathetic response required for erection — and the primary driver of mesolimbic dopamine release. Raising acetylcholine via Muira Puama supports both the neurological and vascular components of sexual function.
- Dopamine upregulation: Muira Puama has demonstrated effects on striatal dopamine turnover, supporting dopaminergic activity through a distinct mechanism from Catuaba's reuptake inhibition.
- Stress-protective adaptogenic activity: Muira Puama reduces cortisol response in animal models and has demonstrated memory and cognitive support consistent with its cholinergic mechanism.

Combined dose: Catuaba bark extract 500mg + Muira Puama 500mg, morning. Both should be from standardised bark extracts. Cycling: 5 weeks on, 2 weeks off — the same cycle as Tribulus, allowing all three to be cycled together.

The Dopaminergic Cycling Schedule

The cycling structure for the Tier 3 dopaminergic and androgenic botanicals is the most important operational rule in this volume. Receptor downregulation from continuous use of dopaminergic compounds is not a theoretical risk — it is the documented mechanism by which men who "take Tribulus every day for years" eventually report that "it stopped working." It stopped working because the receptors adapted to continuous stimulation.

The Tier 3 rotation:

Weeks 1-5 (ON): Full Tier 3 botanical stack active

- Tribulus (Bulgarian, ≥50% protodioscin): 500-750mg daily, morning
- Mucuna pruriens (15% L-DOPA standardised): 300-500mg daily, morning
- Catuaba bark extract: 500mg daily, morning
- Muira Puama bark extract: 500mg daily, morning

Weeks 6-7 (OFF): All four botanical compounds stopped

- Continue all Tier 1 and Tier 2 compounds unchanged
- No botanical androgens or dopaminergic herbs
- This is the receptor reset period. Do not shortcut it.

Week 8 onwards: Repeat the 5-week ON cycle

The two-week OFF period is when receptor sensitivity is restored. Men who experience their best results with Tier 3 compounds typically report that the second and third cycles are more potent than the first — because the receptor reset has occurred between cycles.

During the OFF weeks, the foundation (Tiers 1 and 2) continues to work. The methylation repair, the endothelial rebuild, the BH4 protection, the glycocalyx restoration — none of these require cycling. The ON/OFF applies only to the botanical androgens and dopaminergics of Tier 3.

Chapter 4: The Neurosteroid Layer — GABA, Allopregnanolone, and the Calm Signal

There is a layer of sexual function that is almost never discussed in men's health content and is critically important for men who have done everything right metabolically and hormonally but still find that the system does not fire reliably under pressure: the neurosteroid layer.

Neurosteroids are metabolites of sex hormones that act directly on GABA-A receptors in the brain, producing the calm, non-anxious, parasympathetically-dominated state that is the neurological prerequisite for sexual response. The primary neurosteroid relevant here is allopregnanolone — a metabolite of progesterone produced via the 5-alpha-reductase Type 1 enzyme in the brain.

The Allopregnanolone-GABA-A Axis

Allopregnanolone (3-alpha-hydroxy-5-alpha-pregnan-20-one) is a positive allosteric modulator of GABA-A receptors — meaning it binds to GABA-A receptors at a site separate from GABA itself and enhances the receptor's response to GABA. The net effect is increased GABAergic inhibitory tone: reduced anxiety, reduced neuronal excitability, reduced sympathetic activation.

This matters for sexual function because erection is a parasympathetic event that is actively suppressed by sympathetic activation (anxiety, cortisol, adrenaline). Allopregnanolone is the endogenous neurochemical mechanism for calming the sympathetic override and enabling the parasympathetic state. Men with low allopregnanolone — due to impaired 5-AR Type 1 activity in the brain, due to finasteride-related 5-AR silencing, or due to chronic stress-driven progesterone shunting toward cortisol — are neurochemically incapable of achieving the GABAergic state required for reliable sexual response under pressure, regardless of their hormonal status.

A second neurosteroid relevant to Tier 3 is 3-alpha-androstanediol (3-alpha-diol) — the product of DHT conversion via 3-alpha-HSD. 3-alpha-diol also acts on GABA-A receptors (particularly in the limbic system) and is described clinically as having "prosexual" and anti-anxiety effects distinct from and complementary to allopregnanolone. Low 3-alpha-diol despite adequate DHT is the DUTCH test marker for the AR-silencing pattern described in Chapter 1.

Supporting Neurosteroid Production Naturally

The primary lever for natural allopregnanolone support is 5-AR Type 1 activity in the brain, which is supported by:

- Adequate progesterone availability: progesterone is the precursor to allopregnanolone. Men produce small amounts of progesterone in the adrenal glands and testes. Adequate dietary cholesterol (eggs, meat) provides the sterol substrate. Chronic cortisol elevation shunts progesterone toward cortisol

production via the adrenal stress response — another mechanism by which chronic stress directly impairs neurosteroid production.

- Glycine: glycine directly upregulates 5-AR Type 1 expression in the prefrontal cortex and hippocampus. It is the most accessible natural lever for increasing central allopregnanolone production. Glycine is already present in the Tier 2 GlyNAC protocol — continuing it in Tier 3 serves the neurosteroid function as well as the glutathione function. Caution: in men with PFS-related neurosteroid receptor sensitisation, glycine can paradoxically worsen anxiety through its NMDA co-agonist activity. If this occurs, pair with taurine (which counteracts the excitatory NMDA component) or reduce the dose.
- TSPO agonism: the translocator protein (TSPO) on the outer mitochondrial membrane is the rate-limiting step for neurosteroidogenesis — it transports cholesterol into the mitochondria where the steroidogenic enzyme cascade begins. Natural TSPO support includes adequate dietary cholesterol, Vitamin D (which upregulates TSPO expression), and moderate aerobic exercise. Pharmaceutical TSPO agonists (etifoxine) exist but require prescription and risk GABA-A receptor downregulation with continuous use.
- Pregnenolone (low-dose, pulsed): pregnenolone is the first steroid produced from cholesterol in the mitochondria — the "mother steroid" from which all other steroids branch. Low-dose pregnenolone (10-25mg) taken on alternating days can support both allopregnanolone and DHEA production. Critical warning: pregnenolone supplementation in men with impaired 5-AR activity can shunt toward estrogen and cortisol production rather than allopregnanolone, causing gynecomastia, water retention, and mood worsening. This risk is highest in men with history of finasteride or dutasteride use. Do not use daily — pulse dosing (alternating days) is the maximum frequency, and the response must be monitored closely.

Agmatine Sulfate — The NMDA Modulator for Tier 3

Agmatine sulfate was introduced in The Confidence Reset Book I as the in-the-moment NMDA circuit breaker for performance anxiety. In Tier 3, it earns a place as a daily complement to the botanical androgenic stack because of its dual function: it reduces the glutamate-driven anxiety that suppresses sexual response, and it modulates nitric oxide synthase toward the pro-erectile eNOS pathway.

Agmatine mechanisms relevant to Tier 3:

- NMDA receptor antagonism: reduces glutamate excitotoxicity and the racing-brain sympathetic activation that prevents the GABAergic state required for sexual response. Unlike pharmaceutical NMDA antagonists (memantine, ketamine), agmatine's modulatory mechanism does not produce dissociative effects or cognitive impairment.
- NOS modulation: agmatine selectively inhibits iNOS (the inflammatory nitric oxide synthase that produces excess NO during inflammation) while favouring

eNOS activity. This is the opposite profile from L-arginine alone, which feeds both iNOS and eNOS without selectivity. Agmatine therefore reduces inflammatory NO overproduction while supporting the pro-erectile eNOS pathway.

- D2 receptor sensitivity: agmatine has demonstrated D2 receptor sensitising effects in animal models, complementing the Tribulus and Mucuna mechanisms for dopaminergic tone.
- IL-6 reduction: directly reduces the inflammatory cytokine that suppresses testosterone synthesis at the Leydig cell level and that drives the LPS-Prolactin chain that Tiers 1 and 2 addressed.

Dosing: 500-1000mg agmatine sulfate daily, morning with Tier 3 botanical stack. Note European availability: agmatine is not approved as a food supplement in all EU member states. Verify jurisdiction-specific status before sourcing.

Cycling: agmatine does not require the strict 5-week cycling of the androgenic botanicals, but a 4-weeks-on / 1-week-off rotation is recommended to maintain receptor sensitivity.

Coffee: The Overlooked Dopaminergic Tool

Caffeine in coffee — at the correct dose — is one of the most evidence-backed dopaminergic interventions available. The mechanism relevant to Tier 3 is adenosine receptor antagonism: caffeine blocks adenosine A2A receptors, which are co-localised with D2 receptors in the striatum. A2A blockade potentiates D2 receptor signalling — effectively sensitising D2 receptors to dopamine without directly increasing dopamine levels.

Research has demonstrated that 1-2 cups of coffee daily increases D2 and D3 receptor density in the striatum. In men recovering from dopaminergic blunting from ashwagandha or SSRIs, coffee at this dose can meaningfully accelerate D2 receptor density restoration.

Additionally, coffee has been shown to act as a natural PDE5 inhibitor (at high doses), reduce estradiol (hepatic clearance support), and support eNOS activity.

The dose matters critically: 1-2 cups per day is the therapeutic range. Beyond this, the sympathetic activation from excess caffeine works directly against the parasympathetic state required for sexual function. An anxious, tachycardic, caffeine-overstimulated man has counteracted every other Tier 3 intervention with his third and fourth cups.

> **Practical rule:** Two cups of black coffee in the morning. No caffeine after 2pm (to protect REM sleep and nocturnal GH pulsatility). No energy drinks, no pre-workouts with high-dose caffeine during the Tier 3 protocol.

Chapter 5: Vitamin D3, Zinc, Boron — The Hormonal Infrastructure

Three compounds that have not been individually addressed in depth in previous tiers deserve detailed treatment in Tier 3: Vitamin D3, zinc, and boron. Together they form the hormonal infrastructure layer — the micronutrient foundation that determines how efficiently the androgenic signals produced by Tier 3 compounds can be translated into biological outputs.

Vitamin D3 — The Steroid Prohormone

Vitamin D is not a vitamin in the traditional sense. It is a seco-steroid prohormone that, after hepatic and renal hydroxylation to its active form 1,25(OH)2D (calcitriol), acts on Vitamin D receptors (VDRs) in virtually every tissue in the body.

Its relevance to Tier 3 is broad and specific:

- Testosterone production: VDRs are expressed on Leydig cells. Vitamin D directly stimulates testosterone biosynthesis via upregulation of StAR protein expression and CYP11A1 enzyme activity. Human trials have demonstrated 25% increases in testosterone with Vitamin D supplementation in deficient men over 12 months.
- 5-AR activity: Vitamin D upregulates 5-AR expression, directly supporting DHT conversion. Men with low Vitamin D consistently show lower DHT:T ratios, which normalise with repletion.
- AR-VDR crosstalk: in the cell nucleus, VDR and AR share coactivator proteins (particularly SRC-1 and SRC-2). Adequate Vitamin D status ensures that these coactivators are available for AR transcriptional activation rather than being monopolised by VDR in a Vitamin D-deficient state.
- Penile tissue health: penile smooth muscle and corpus cavernosum contain VDRs. Vitamin D directly supports the tissue health of erectile structures, independent of its systemic hormonal effects.
- IGF-1 elevation: Vitamin D upregulates IGF-1 production and receptor expression, feeding the 5-AR-DHT-IGF-1 feed-forward loop described in Chapter 2.
- Hair follicle support: VDRs in hair follicles drive the anagen (growth) phase. Vitamin D deficiency is an independent cause of hair loss that is distinct from DHT-driven alopecia.

Target: serum 25(OH)D of 50-70 ng/mL. Most men in temperate climates with indoor jobs are at 20-30 ng/mL — functionally deficient from a hormonal perspective.

Dosing: 3000-5000 IU Vitamin D3 daily with the largest meal (fat-soluble, requires dietary fat for absorption). Must be paired with Vitamin K2 (MK-7, 100-200mcg daily)

to direct calcium mobilised by D3 away from arterial walls and toward bone. D3 without K2 at higher doses risks arterial calcification over time.

Testing caveat: the standard 25(OH)D test measures storage form. Some men show normal 25(OH)D but poor conversion to the active 1,25(OH)2D form due to CYP27B1 enzyme impairment — caused by chronic inflammation, kidney dysfunction, or (reported in some clinical discussions) COVID-related enzyme inhibition. If symptoms of Vitamin D deficiency persist despite adequate 25(OH)D, request testing of 1,25(OH)2D through a specialist.

Zinc — The Androgenic Mineral

Zinc is required as a direct cofactor for over 300 enzymatic reactions, but its specific relevance to Tier 3 is concentrated in four androgenic mechanisms:

- 5-alpha-reductase cofactor: the zinc ion is a structural requirement for 5-AR enzyme function. Zinc deficiency reduces 5-AR activity directly — this is why zinc deficiency produces low DHT despite normal testosterone. It is also why some pharmaceutical 5-AR inhibitor research has used zinc depletion as an experimental control.
- Aromatase inhibition: zinc inhibits aromatase (the enzyme that converts testosterone to estradiol) at the enzyme level. This is a mild to moderate effect at nutritional doses — not as potent as pharmaceutical aromatase inhibitors — but consistent and meaningful in the context of optimising the testosterone:estradiol ratio.
- LH stimulation: zinc is required for normal pituitary LH pulsatility. Zinc deficiency reduces LH pulse amplitude, which reduces testicular testosterone stimulation.
- Prolactin modulation: zinc deficiency increases prolactin sensitivity and may elevate basal prolactin. Zinc repletion has demonstrated prolactin-normalising effects independent of the P5P and Tribulus mechanisms.

Target: 15-25mg elemental zinc daily. Forms: zinc glycinate or zinc picolinate are the most bioavailable. Zinc oxide (the form in most cheap supplements) is poorly absorbed.

Critical note on zinc repletion timeline: tissue-level zinc repletion from a deficient state takes up to 6 months of consistent supplementation. Men who have been zinc-deficient for years cannot replete in weeks. This is why zinc appears in Tier 1 (begin repletion early) and continues in Tier 3 (maintain and optimise).

Copper balance: long-term zinc supplementation above 25mg daily without copper supplementation can deplete copper. Add 1-2mg copper glycinate if using zinc above 15mg for extended periods.

Boron — The SHBG Suppressor

Boron is a trace mineral with a clinical story that is disproportionately underappreciated relative to the clinical evidence supporting it. In a single dose-ranging human trial, 10mg boron daily for 7 days produced the following changes:

- Free testosterone: increased by approximately 28% via SHBG reduction.
- Estradiol: decreased by approximately 39% via aromatase inhibition.
- DHT: increased (secondary to the testosterone and estradiol changes).
- SHBG: decreased significantly (the primary mechanism for free testosterone increase).

SHBG (Sex Hormone-Binding Globulin) is the protein that binds testosterone and DHT in the bloodstream, making them biologically inactive. Only unbound (free) testosterone and DHT can enter target cells and activate the androgen receptor. A man with total testosterone of 700 ng/dL but very high SHBG may have less biologically active androgen than a man with 400 ng/dL total testosterone and normal SHBG.

Boron is the most accessible and evidence-backed natural SHBG-lowering intervention available. Its mechanism operates via inhibition of SHBG synthesis in the liver (boron interferes with the glucocorticoid-induced SHBG upregulation pathway) and possibly via direct SHBG-androgen binding competition at the molecular level.

Dose: 10mg boron daily, taken with food. Boron is toxic at very high doses (above 20mg/day chronically) but the 10mg therapeutic dose has a wide safety margin. Available as boron glycinate or as part of calcium-magnesium-boron mineral formulas.

> **The SHBG-boron connection in context:** Men following ketogenic or very low-carbohydrate diets are at particular risk of high SHBG — carbohydrate restriction raises SHBG via insulin suppression (insulin lowers SHBG). In these men, boron's SHBG-suppressing effect is especially clinically significant.

Chapter 6: The Advanced Options — When to Consider Medical Intervention

Tier 3 is the natural ceiling of the Stub Club Protocol. The compounds and practices in Tiers 1-3 represent the most comprehensive evidence-based natural protocol available for hormonal restoration and androgenic optimisation. For a significant proportion of men who complete all three tiers correctly, this is sufficient.

For some men — those with primary hypogonadism, those with significant PFS-related epigenetic damage, those who are beyond the natural capacity for hormonal recovery based on age or medical history — the medical options described in this chapter become the appropriate next step.

This chapter is not a prescription. It is an education. Decisions about pharmaceutical hormonal intervention require a physician who will order the appropriate labs, interpret them in the correct functional context (not just against standard lab ranges), and prescribe and monitor accordingly.

HCG — The LH Mimic

Human chorionic gonadotropin (HCG) mimics LH (luteinising hormone) — the pituitary signal that directly stimulates testicular testosterone and DHT production. It is the preferred first-line medical intervention for secondary hypogonadism (where the problem is insufficient LH signalling from the pituitary, not a testicular failure) and for men who want to maintain testicular size and function while supporting hormonal output.

Who it is appropriate for: men with confirmed secondary hypogonadism on lab work (low LH, low testosterone), men with testosterone in the lower third of the reference range despite completing Tiers 1-3, and men for whom TRT is under consideration but who want to attempt a less suppressive approach first.

The critical dosing error to avoid: the most common clinical mistake is overdosing. Protocols recommending 2000-4000 IU three times per week are in the literature, but they consistently produce estradiol spikes to 100+ pg/mL, driving the water retention, gynecomastia, and mood disruption that then require aggressive aromatase inhibitor use — a further hormonal destabilisation. The clinical coaching evidence strongly favours a low-dose approach:

- Starting protocol: 500-800 IU, 2-3 times per week, subcutaneous injection.
- Vitamin E synergy: research has demonstrated that pairing HCG with Vitamin E (tocotrienol form, 200-400 IU daily) increases the effectiveness of HCG stimulation without requiring higher HCG doses. This allows the testosterone-stimulating benefit with a lower estradiol side effect burden.
- Monitoring: total testosterone, free testosterone, DHT, estradiol, prolactin, and LH/FSH at baseline and 6 weeks into the protocol. Estradiol should remain in

the 20-30 pg/mL range. If it rises above 40 pg/mL, reduce HCG dose before considering aromatase inhibitors.

- Long-term use: HCG can be used long-term (years) unlike enclomiphene/clomid, which are not recommended as permanent solutions. HCG also has the specific benefit of maintaining 5-AR activity in the testes over 4-6+ months — meaning it supports not just testosterone but DHT conversion more durably than testosterone-only TRT.

Topical testosterone to the scrotum: the scrotal skin is the highest concentration of 5-AR Type 1 enzyme in accessible external skin — DHT conversion from topically applied testosterone is significantly higher at the scrotum than at any other topical application site. For men who choose a TRT route, scrotal testosterone cream produces more local and systemic DHT relative to estradiol than injectable testosterone, making it the preferred route for men who are sensitive to estradiol or who are prioritising DHT optimisation.

Enclomiphene — The SERM Route

Enclomiphene (brand name Androxal) is a selective estrogen receptor modulator (SERM) that works via a fundamentally different mechanism from HCG: it blocks estrogen receptors in the hypothalamus and pituitary, removing the negative feedback signal that estrogen provides to GnRH and LH secretion. The result is increased GnRH pulsatility → increased LH → increased testicular testosterone.

In men with secondary hypogonadism and adequate testicular function, enclomiphene can double or triple testosterone, producing levels in the 800-1000+ ng/dL range without exogenous testosterone.

The critical downside: enclomiphene consistently lowers IGF-1. The mechanism involves estrogenic signalling in the liver, where estrogen drives IGF-1 production — blocking pituitary estrogen receptors also reduces the estrogenic stimulus for hepatic IGF-1 synthesis. For a protocol that depends on IGF-1 as the second-most-important driver of 5-AR activity, this is a significant trade-off.

Enclomiphene is best positioned as a short-term intervention (3-6 months) rather than a permanent solution, and is most appropriate for men with clearly confirmed secondary hypogonadism who want a non-suppressive option that preserves testicular function and fertility. It is not appropriate as a long-term permanent testosterone support strategy.

TRT — The Decision Framework

Testosterone replacement therapy is the most effective hormonal intervention available for confirmed hypogonadism. It is also the most frequently prescribed prematurely — before natural options have been genuinely exhausted, before the causes of low testosterone have been investigated and addressed, and before the patient understands the implications of permanent hormonal dependency.

The cases where TRT becomes the appropriate conclusion of the Stub Club Protocol:

- Total testosterone consistently below 300 ng/dL on multiple morning tests after completing Tiers 1-3 and addressing all modifiable factors (gut health, sleep, body composition, thyroid, Vitamin D, zinc, cortisol).
- Primary hypogonadism (high LH, low testosterone) — the testes are not responding to adequate pituitary stimulation, and no natural intervention or HCG will produce meaningful testosterone restoration because the testicular cells themselves are impaired.
- Confirmed significant hormonal deficiency with clinical symptoms that have not responded to the complete Tier 1-3 protocol after adequate duration.

The TRT pre-checklist (from clinical coaching source data):

- Fix gut and inflammation first — TRT in the context of chronic LPS-driven inflammation will produce more aromatisation to estradiol and more prolactin-driven side effects.
- Reach target body composition — adipose tissue is a significant source of aromatase; men above approximately 90kg or with high body fat will aromatise more testosterone to estradiol from TRT.
- Review all medications — antidepressants, painkillers, antifungals, and many other medications impair testosterone production or AR function. Remove what can be removed first.
- Understand the SHBG dynamic — TRT via injection typically raises estradiol and SHBG via aromatisation. Managing injection frequency (more frequent lower-dose injections produce more stable levels and less estradiol spike) and considering topical routes (scrotal for DHT, non-scrotal transdermal for more estrogen-balanced profiles) determines whether TRT produces the intended outcome.

The PFS-Specific Pathway — What Is Different

Men who have used finasteride, dutasteride, saw palmetto at high doses, or other 5-AR inhibitors for extended periods and experienced persistent post-discontinuation symptoms face a different clinical challenge than men with uncomplicated hormonal decline. The PFS pathway requires specific modifications to the Tier 3 approach:

- AR silencing assessment first: before beginning the androgenic amplification of Tier 3, confirm whether AR silencing is contributing to the symptom picture via DUTCH test (low 3-alpha-diol despite normal DHT). If AR silencing is present, begin sodium butyrate (300-600mg daily) and allow 2-4 months before introducing the full Tier 3 botanical stack.
- Neurosteroid priority: allopregnanolone depletion is a central PFS mechanism. Support the glycine + taurine protocol, Vitamin D, and dietary cholesterol for

TSPO substrate before attempting androgenic amplification. Men with significant anxiety, brain fog, or emotional blunting from PFS need the GABAergic foundation restored before the androgenic layer will be tolerable.

- Avoid progesterone/pregnenolone without confirmed 5-AR activity: as described in the neurosteroid chapter, these can shunt toward estradiol and cortisol in the presence of impaired 5-AR, worsening rather than improving symptoms.
- Proviron (mesterolone) — the medical option for CAG-repeat AR polymorphism: Proviron is an oral, non-hepatotoxic (at low doses) synthetic DHT derivative that binds the androgen receptor with high affinity. For men with CAG-repeat driven AR insensitivity — not AR silencing — who are not responding to natural DHT optimisation, Proviron at 25-50mg daily (under medical supervision) provides direct AR activation. It does not aromatise to estradiol, does not suppress natural testosterone production significantly at low doses, and has the longest clinical safety record of any oral androgen.

Chapter 7: The Sauna-Cold Plunge Protocol — Heat Shock and Prolactin Management

The sauna-cold plunge sequence is the only accessible physical practice that simultaneously achieves three Tier 3 objectives: heat shock protein induction (for AR chaperone support), cardiovascular endothelial conditioning (for Tier 2 glycocalyx maintenance), and prolactin management.

Why the Sequence Is Non-Negotiable

Sauna exposure produces a significant, dose-dependent release of GH (growth hormone) — up to 5-fold increases in GH concentration have been documented in research. It also produces heat shock proteins (HSP70, HSP90), which are the chaperone proteins that maintain AR in its ligand-competent conformation. Both of these are highly desirable for Tier 3 objectives.

However, sauna also produces a prolactin spike in the recovery period — specifically, a rise in prolactin that peaks 20-40 minutes after leaving the sauna. For men who have worked through Tier 1 to normalise prolactin, this heat-induced prolactin spike is a recovery-period interference that undermines the prolactin management that Tier 3 depends on.

Immediate cold plunge (within 2 minutes of leaving the sauna) interrupts the prolactin recovery spike. The mechanism is twofold: cold exposure activates the sympathoadrenal system in a way that does not elevate prolactin (unlike the heat recovery period's parasympathetic rebound) and the dopaminergic effect of cold exposure (norepinephrine release in the brain's dopaminergic circuits) inhibits prolactin secretion via the tuberoinfundibular pathway.

The sequence is: sauna → immediate cold plunge → no sauna again without cold plunge. If you use sauna without the cold plunge, you are producing GH and HSPs and then undoing the prolactin work of Tier 1. Both or neither.

Practical Protocol

Frequency and structure:

- Frequency: 3-4 sessions per week. Research by Dr. Rhonda Patrick and Finnish population cohort data associate 4+ sauna sessions per week with the most significant cardiovascular, hormonal, and longevity benefits.
- Sauna parameters: 80-100°C (176-212°F). Traditional Finnish dry sauna is the most studied modality. Infrared saunas operate at lower temperatures and produce less cardiovascular conditioning and less GH response, though they provide infrared-specific benefits (mitochondrial and tissue recovery).
- Duration: 15-25 minutes per session. GH response is dose-dependent on heat and duration up to approximately 25-30 minutes, after which the marginal response diminishes.

- Cold plunge parameters: water temperature 5-15°C (40-59°F). Duration: 2-5 minutes. Immersion (full body) is more effective than showering for the vagal and dopaminergic responses.
- Timing: not within 3 hours of a heavy training session (the heat and cold will impair some of the muscle adaptation signals from resistance training). Morning or afternoon is preferred over immediately post-workout.
- Hydration: replace fluids and electrolytes (particularly sodium and magnesium) after sauna. A 400mg magnesium malate dose after sauna replaces the magnesium lost through sweat and supports the post-sauna recovery period.

Cardiovascular Effects: The Tier 2-Tier 3 Bridge

Sauna exposure produces cardiovascular effects that directly support the endothelial work of Tier 2:

- Shear stress on the endothelium: during sauna, cardiac output increases significantly (heart rate rises to 100-150 bpm in most subjects), generating increased blood flow velocity and shear stress across the vascular endothelium. This shear stress is the primary physiological stimulus for eNOS activation and NO production. Regular sauna use therefore provides repeated endothelial eNOS-stimulating shear stress events beyond those produced by exercise alone.
- NO bioavailability improvement: multiple studies have demonstrated that regular sauna use increases plasma nitric oxide metabolites (NOx) and improves flow-mediated dilation — the clinical measure of endothelial function. This is the Tier 2 glycocalyx repair and eNOS recoupling work being exercised and maintained by the sauna protocol.
- Mitochondrial biogenesis: heat stress activates PGC-1alpha, the master regulator of mitochondrial biogenesis — growing new mitochondria. Combined with the PQQ from Tier 2 (also a PGC-1alpha activator), regular sauna use supports ongoing mitochondrial health beyond what supplementation alone achieves.

Chapter 8: The Complete Tier 3 Reference

The Full Three-Tier Stack Summary

All Tier 1 and Tier 2 compounds continue unchanged throughout Tier 3. The stack below represents the additions:

Tribulus terrestris (Bulgarian ≥50% protodioscin) — 500-750mg | Morning, weeks 1-5 of each cycle | *DHT elevation via DHEA pathway. Prolactin reduction. AR coactivator recruitment. Must cycle.*

Mucuna pruriens (15% L-DOPA standardised) — 300-500mg | Morning, weeks 1-3 then off 2 weeks (nested within 5-week cycle) | *Dopamine precursor. Prolactin suppression. LH support. Do NOT combine with MAO inhibitors.*

Catuaba bark extract — 500mg | Morning, weeks 1-5 of each cycle | *Dopamine + norepinephrine reuptake inhibition. 5-HT2 antagonism. Anti-neuroinflammatory.*

Muira Puama bark extract — 500mg | Morning, weeks 1-5 of each cycle | *Acetylcholinesterase inhibition. Parasympathetic tone. Dopamine turnover. Adaptogenic.*

Agmatine sulfate — 500-1000mg | Morning, 4 weeks on / 1 week off | *NMDA modulation. eNOS favouring. D2 sensitisation. IL-6 reduction.*

Vitamin D3 — 3000-5000 IU | Daily with largest meal | *Testosterone biosynthesis. 5-AR upregulation. AR-VDR coactivator sharing. Must pair with K2.*

Vitamin K2 (MK-7) — 100-200mcg | Daily with D3 | *Calcium direction away from arteries. D3 synergist. Arterial calcification prevention.*

Zinc glycinate — 15-25mg | Daily, with food (not within 2hr of iron) | *5-AR cofactor. Aromatase inhibition. LH support. Prolactin modulation. Long repletion timeline.*

Boron glycinate — 10mg | Daily with food | *SHBG suppression. Free testosterone increase. Estradiol reduction. DHT support.*

Selenium (selenomethionine) — 150-200mcg | Daily | *T4-to-T3 deiodinase cofactor. 5-AR activity support via T3 pathway.*

Sodium butyrate (if AR silencing suspected) — 300-600mg | Daily, 2-4 months before botanical stack if indicated | *HDAC inhibitor. AR gene de-methylation. Gut barrier dual function.*

Coffee (black) — 1-2 cups | Morning only, no caffeine after 2pm | *D2/D3 receptor density upregulation via A2A antagonism. Natural PDE5 inhibition. Estradiol clearance.*

The Cycling Schedule

Weeks 1-5: Botanical stack active

- Tribulus 500-750mg, Catuaba 500mg, Muira Puama 500mg: all daily, morning.

- Mucuna 300-500mg: take weeks 1-3, then stop for weeks 4-5 (nested shorter cycle within the 5-week botanical window).
- Agmatine: continue from 4-weeks-on / 1-week-off cycle regardless of botanical week.
- All other Tier 3 support compounds (D3+K2, zinc, boron, selenium): daily, no cycling required.

Weeks 6-7: ALL botanical compounds off

- Tribulus, Mucuna, Catuaba, Muira Puama: stop completely for 2 weeks.
- Continue all Tier 1, Tier 2, and non-botanical Tier 3 compounds.
- This is the receptor reset. Do not supplement with other dopaminergic or androgenic herbs to fill the gap.

Week 8: Begin next cycle

- Restart from Week 1 of the botanical cycle.
- Most men report second and third cycles producing the strongest effects — receptor reset has occurred.

The Lab Targets for Tier 3 Completion

Total Testosterone: ≥ 500 ng/dL. But total T is not the endpoint — free T and DHT are.

DHT: ≥ 10% of total testosterone (T:DHT ratio ~10:1, DHT ~60-100 ng/dL). Rising DHT with stable or rising T confirms 5-AR function is improving.

Free Testosterone: > 2-3% of total T. Boron and SHBG management are the primary levers.

SHBG: 20-40 nmol/L. If above 45, boron + carbohydrate intake + liver support. If below 15, review insulin resistance and thyroid.

Prolactin: 4-7 ng/mL. If still above 10 entering Tier 3, Tier 1 is incomplete.

Estradiol: 20-30 pg/mL. Above 40 with normal testosterone indicates aromatisation excess — zinc, boron, body composition, review dietary inputs.

Free T3: Upper third of reference range. If not here, 5-AR is running at sub-optimal capacity regardless of androgenic support.

IGF-1: ≥ 200 ng/mL. Below this, the 5-AR-DHT feed-forward loop is impaired. Sleep and resistance training before supplemental interventions.

25(OH)D (Vitamin D): 50-70 ng/mL. The most commonly under-corrected hormonal variable.

3-alpha-diol-G (DUTCH): Low despite normal DHT = AR utilisation failure. Assess for AR silencing, initiate sodium butyrate protocol.

hs-CRP: < 1.0 mg/L. If elevated at Tier 3, return to gut and dietary reform. Inflammation at Tier 3 undermines every androgenic intervention.

The Self-Monitoring Signals at Tier 3

- Morning erections: should be reliably present, with increasing quality, by Week 3-4 of the first botanical cycle if the Tier 1 and Tier 2 foundation is solid. If absent, the foundation is the issue, not the Tier 3 botanicals.
- Libido: should show clear improvement in drive, motivation, and spontaneous sexual interest within the first 2-3 weeks of the botanical stack. The dopaminergic layer (Tribulus-Mucuna-Catuaba-Muira Puama) produces the most subjectively noticeable effects in this category.
- Mood and motivation: the D2 receptor sensitisation from Tier 3, combined with the prolactin reduction, should produce improved baseline mood, reduced anhedonia, and increased goal-directed motivation beyond the sexual domain. These are systemic dopaminergic improvements, not just sexual ones.
- Sleep quality: should be maintained from Tier 2. If sleep deteriorates in Tier 3, the dopaminergic compounds (particularly Mucuna at higher doses) may be overstimulating the system. Reduce Mucuna dose or move it earlier in the morning.
- Energy and cognitive clarity: improving mitochondrial function, thyroid optimisation, and dopaminergic tone should produce measurable improvements in sustained mental energy and focus.

What Comes After Tier 3

Tier 3 is the natural ceiling of the Stub Club Protocol. It is not the end of the work — it is the beginning of maintenance.

After completing two full botanical cycles (approximately 4-5 months of Tier 3) with confirmed lab improvements, the protocol shifts to a sustainable long-term maintenance mode:

- Tiers 1 and 2 foundation: permanent ongoing baseline, with the post-6-month reduced maintenance doses described in the Tier 2 reference.
- Tier 3 cycling: continue the 5-week ON / 2-week OFF botanical rotation indefinitely. The cycling is not a temporary measure — it is the mechanism that keeps the system responding to the compounds.
- Labs: test every 6 months. Track the primary androgenic and vascular markers. If any marker drifts out of range, the protocol gives you the specific lever for that specific marker.
- Lifestyle infrastructure: the sauna-cold plunge protocol, resistance training, sleep quality maintenance, arugula and dietary nitrates, the egg protocol, carbohydrate adequacy for T3 — these are not temporary interventions. They are the biological environment in which the three-tier protocol operates. Remove them and the protocol gradually reverts.

"The Stub Club Protocol is not a supplement programme. It is a biological operating system. Tiers 1, 2, and 3 together — when implemented correctly, in sequence, with patience — represent a comprehensive restoration of the hormonal, vascular, neurological, and androgenic systems that male sexual health depends on. The work is real. The biology is real. The results follow the work."

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Appendix A: Compounds to Avoid in Tier 3

These compounds are commonly encountered in men's health circles but are directly counterproductive to the Tier 3 goals.

Ashwagandha

Serotonergic (MAO-inhibiting, tryptophan-elevating). Possible 5-AR inhibitory activity. Directly opposes the dopaminergic amplification of Tier 3. Remove entirely.

Saw palmetto, pygeum, stinging nettle (high-dose)

5-AR inhibitors. Directly counteract DHT amplification. Incompatible with Tier 3 objectives.

DIM above 100mg

Androgen receptor antagonist and mTOR inhibitor at supplement doses. Blocks the AR signalling that Tier 3 is attempting to amplify.

High-dose curcumin supplements

Moderate 5-AR inhibitory and AR antagonist activity at supplement doses. Dietary turmeric is acceptable.

MAO inhibitors with Mucuna

Absolute contraindication. Mucuna contains L-DOPA; combined with MAO inhibitors (pharmaceutical MAOIs, high-dose St. John's Wort, selegiline) risks hypertensive crisis or serotonin syndrome.

Alcohol

Depletes B-vitamins, elevates prolactin acutely, impairs REM sleep and GH pulsatility, increases aromatisation to estradiol. Incompatible with Tier 3 at any regular frequency.

MK-677 (Ibutamoren)

Multiple clients have experienced cortisol/adrenaline spikes, water retention, appetite dysregulation, and raised prolactin with MK-677. CJC-1295 + ipamorelin is preferred if peptide secretagogue GH support is desired.

IV NAD+ drips

Heavy methyl-donor drain, raises Homocysteine, undermines the Tier 1 methylation work that the entire stack depends on.

Ibuprofen/paracetamol (chronic daily use)

Reduces androgen receptor expression. Occasional use acceptable; chronic daily use during Tier 3 is counterproductive.

Appendix B: The PFS/PSSD-Specific Tier 3 Pathway

A specific reference for men with post-finasteride syndrome (PFS) or post-SSRI sexual dysfunction (PSSD) who are beginning Tier 3. Standard Tier 3 sequencing applies to most men; PFS/PSSD cases require the modifications below.

- Step 0 (before Tier 3 botanicals): obtain DUTCH test. Confirm 3-alpha-diol-G level relative to DHT. If 3-alpha-diol-G is low with normal or elevated DHT, AR silencing is likely contributing. Begin sodium butyrate 300-600mg daily for 2-4 months before introducing the full botanical stack.
- Neurosteroid priority: ensure glycine (3g daily) + taurine (3g daily) + Vitamin D3 (5000 IU) are fully established from Tiers 1 and 2 before beginning androgenic amplification. The GABAergic foundation must be in place for the androgenic signal to be received without triggering sympathetic override.
- Mucuna dose: begin at 150mg (not 300-500mg) and titrate up over 4 weeks. PFS/PSSD-affected dopaminergic systems may be hyper-responsive to L-DOPA initially.
- Avoid progesterone and pregnenolone without confirmed 5-AR activity. In PFS, these will shunt toward estradiol and cortisol rather than allopregnanolone in the presence of impaired 5-AR.
- Proviron consideration: if after 2 months of sodium butyrate + full botanical cycle there is no meaningful improvement in 3-alpha-diol-G on repeat DUTCH, Proviron 25mg daily under medical supervision may be appropriate for CAG-repeat AR polymorphism cases.
- AR silencing confirmation: if Proviron produces no response, true epigenetic AR silencing is likely. Continue sodium butyrate for extended period (6+ months) with niacinamide 250mg daily as a second HDAC inhibitor. Assess progress at 6-month DUTCH retest.
- Do not use TRT or HCG as a substitute for AR repair in silencing cases: androgenic load into a silenced AR system does not produce the intended response and creates negative feedback effects that worsen the hormonal environment.