THE VASCULAR FOUNDATION

Book II of The Confidence Reset

How Circulation, Sleep, and Hormones Shape Sexual Health

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Introduction: The Engine Under the Hood

Book I of The Confidence Reset showed you how the nervous system and neurochemistry sabotage performance from the top down. This book shows you how the vascular and hormonal system fails you from the bottom up.

These two failure modes often coexist in the same man. Performance anxiety creates oxidative stress that damages the endothelium. A damaged endothelium produces less nitric oxide. Less nitric oxide means weaker erections, which reinforces the anxiety loop. The two layers feed each other.

This book addresses the physical infrastructure: the endothelial lining of your blood vessels, the methylation pathway that keeps it intact, the mitochondrial function that powers hormone production, the prolactin axis that blocks it, the thyroid-DHT conversion pathway, the REM sleep cycle that maintains penile tissue, and the restoration stack that rebuilds all of it.

> *"If you have a circulation problem down there, you have a circulation problem everywhere. Erectile dysfunction is the smoke alarm for the cardiovascular system."*

The penile arteries are among the smallest in the body. They clog first, and they fail first. This is why vascular ED typically precedes cardiac events by three to five years in men who are not paying attention to the signals.

We are going to pay attention. And we are going to fix it.

Chapter 1: The Endothelial Engine

An erection is not a hormonal event. It is a neurovascular event. Understanding this distinction is the beginning of real recovery.

The process begins in the brain — a dopaminergic signal initiates the cascade. That signal travels via the parasympathetic nervous system to the pelvic nerves, which release nitric oxide into the penile smooth muscle. The nitric oxide activates an enzyme called guanylate cyclase, which produces cGMP — the molecule that actually relaxes the smooth muscle of the corpus cavernosum and allows it to fill with blood.

PDE5 inhibitors (Viagra, Cialis) work by blocking the enzyme that breaks down cGMP. They extend the signal. They do not create it. If your endothelium cannot produce nitric oxide in the first place, no PDE5 inhibitor will work. You are extending a signal that does not exist.

The Glycocalyx: The Layer Nobody Talks About

The endothelium is not a simple tube. Its inner surface is coated with a delicate gel layer called the glycocalyx — a mesh of proteoglycans and glycoproteins that acts as the primary mechanosensor of blood flow. When blood flows across the glycocalyx, it generates a shear stress signal that tells the endothelial cells to produce nitric oxide.

The glycocalyx is destroyed by: elevated blood sugar (even transient post-meal spikes), oxidative stress, systemic inflammation, elevated LPS endotoxins from gut dysbiosis, and — critically — elevated homocysteine. When the glycocalyx is stripped, the endothelial cells lose their primary stimulus for NO production. The machinery is intact but the sensor is gone.

Rebuilding the glycocalyx requires 4-7 months of consistent targeted support. The primary agents are sulodexide (a pharmaceutical glycosaminoglycan available in Europe and Asia, prescription-only in most markets) and Pycnogenol (French maritime pine bark extract), which has demonstrated glycocalyx-protective activity in multiple studies.

> **Pycnogenol:** 120-160mg daily. Protects the glycocalyx, reduces ADMA, supports NO production. Requires 8-12 weeks of consistent use before effects are measurable.
>
> **Sulodexide:** 500-1000 LRU daily if available in your jurisdiction. The most direct glycocalyx repair agent in clinical use.

The Endothelium as an Endocrine Organ

The endothelium is not a passive lining. It is an active endocrine organ that produces over 30 biologically active substances — including nitric oxide, prostacyclin, endothelin, and von Willebrand factor. Its health is central not just to erectile function but to cardiovascular risk, blood pressure regulation, inflammatory signalling, and immune function.

This is why fixing the endothelium matters beyond sexual health. The interventions in this book are cardiovascular medicine applied with precision to a specific functional outcome. The man who rebuilds his endothelial health for the sake of his erections is also dramatically reducing his cardiovascular risk, his inflammatory burden, and his cognitive decline trajectory.

> *"Fixing the endothelium for erectile function is fixing the cardiovascular system for everything else. The benefit is not compartmentalised."*

Chapter 2: The Homocysteine Poison

Standard medicine is not lying to you deliberately. But it is using the wrong reference range, and the consequences for vascular health are serious.

The standard laboratory reference for Homocysteine is typically listed as normal up to 15 µmol/L. This range was set based on population averages — it tells you what is typical for the population being measured, not what is optimal for endothelial health.

The clinical evidence from vascular medicine is clear: above 8 µmol/L, Homocysteine begins to actively injure the endothelial lining. The optimal range for longevity and vascular health is 5-8 µmol/L. A level of 15 is not "normal." It is a vascular disaster happening slowly, every hour, in every blood vessel in your body.

How Homocysteine Kills Erections

Homocysteine operates through four simultaneous damaging mechanisms:

- Direct endothelial injury: Homocysteine is a thiol amino acid that directly damages the endothelial cell membrane through oxidative stress and the production of hydrogen peroxide.
- The ADMA trap: Homocysteine increases levels of ADMA (Asymmetric Dimethylarginine) — the body's natural inhibitor of nitric oxide synthase. ADMA competes with arginine at the eNOS enzyme, blocking NO production at the source. This is why taking more arginine when Homocysteine is elevated does not work — ADMA wins the competition.
- Methylation failure: Elevated Homocysteine is the primary biochemical marker of impaired methylation. Poor methylation means impaired DNA repair, impaired neurotransmitter production, impaired estrogen detoxification, and impaired phosphatidylcholine synthesis — the last of which is required for maintaining cell membrane integrity throughout the vascular system.
- BH4 depletion: High Homocysteine generates peroxynitrite, which destroys BH4 — the essential cofactor for eNOS coupling. This creates a vicious cycle: elevated Homocysteine → BH4 destruction → eNOS uncoupling → superoxide instead of nitric oxide → more oxidative stress → more Homocysteine accumulation.

The Folate Trap

One of the most important clinical subtleties in this area is the "folate trap." A standard blood test may show normal serum folate and normal serum B12 — and yet the patient has functionally impaired methylation and elevated Homocysteine. This occurs when the folate cycle is blocked upstream, typically due to MTHFR gene variants (C677T being the most common) that impair the conversion of dietary folate to its active form, 5-MTHF.

The solution is not more folic acid. Folic acid is the synthetic, inactive form of folate. Supplementing with folic acid in a patient with MTHFR variants can actually worsen the functional deficiency by flooding the system with an unusable precursor that blocks the receptors for the active form.

The correct intervention is the active form directly:

- 5-MTHF (methylfolate) — the bioactive form of folate that bypasses the MTHFR bottleneck
- Methylcobalamin (methyl-B12) — not cyanocobalamin, which requires hepatic conversion
- P5P (pyridoxal-5-phosphate) — the active form of B6, required as a cofactor in the methylation cycle
- Riboflavin (B2) — cofactor for MTHFR enzyme activity itself; frequently overlooked
- TMG (Trimethylglycine / Betaine) — the alternative methylation donor that provides an MTHFR-independent route for recycling Homocysteine

> *"You do not just "take folate." You take the right form, at the right dose, with the right cofactors, in the right sequence. Blind methylation support can cause anxiety, insomnia, and dopamine dysregulation in sensitive individuals."*

The Homocysteine Testing Protocol

Request the following from your physician or through a private lab:

- Homocysteine (fasting) — your primary target marker. Aim < 8 µmol/L.
- Active B12 (holo-TC / holotranscobalamin) — not standard B12. Standard B12 measures total B12 including inactive forms; holo-TC measures only the biologically available fraction.
- RBC folate — not serum folate. RBC folate reflects intracellular stores over 3 months; serum folate reflects only recent dietary intake.
- MMA (Methylmalonic Acid) — functional marker of B12 sufficiency at the cellular level.
- hs-CRP — inflammatory marker; elevated CRP accelerates BH4 depletion and ADMA production.

Retest Homocysteine 8-12 weeks after beginning the methylation protocol. If it has not moved significantly, investigate MTHFR variant status and consider increasing TMG or adding creatine (which reduces the body's methylation demand by ~40-50%, freeing up SAMe for other methylation reactions).

Chapter 3: eNOS Uncoupling — The Internal Rust

Most men who discover they have low nitric oxide production do exactly the wrong thing: they buy L-arginine supplements and take them by the handful.

If your eNOS enzyme is uncoupled, adding arginine makes the situation measurably worse. Understanding why requires understanding the eNOS system in some detail.

The eNOS Mechanism

Endothelial Nitric Oxide Synthase (eNOS) is the enzyme responsible for converting L-arginine into nitric oxide and L-citrulline. This conversion requires several essential cofactors: BH4 (tetrahydrobiopterin), NADPH, FAD, FMN, haem, and calmodulin. When all cofactors are present and the enzyme is in its normal "coupled" state, it produces nitric oxide efficiently.

When BH4 is depleted — by oxidative stress, peroxynitrite, elevated Homocysteine, systemic inflammation, or elevated LPS endotoxins — the eNOS enzyme "uncouples." In the uncoupled state, the enzyme no longer produces nitric oxide. Instead, it produces superoxide — the same free radical generated by white blood cells to kill bacteria. Superoxide in the vascular wall reacts with any remaining nitric oxide to form peroxynitrite, which is itself a powerful oxidant that destroys more BH4, completing the vicious cycle.

> *"Adding arginine to an uncoupled eNOS system is like adding fuel to a broken engine. You are not producing power. You are producing heat, smoke, and internal damage."*

The Arginine-Infection Risk

There is an additional reason to be cautious with high-dose arginine supplementation that is almost never discussed in men's health circles: arginine is the primary amino acid required for replication by certain latent pathogens.

Herpes simplex virus (HSV-1 and HSV-2), Epstein-Barr virus (EBV), and several biofilm-forming bacteria (Ureaplasma urealyticum, Mycoplasma genitalium) all use arginine as a fuel source and for replication. High-dose arginine supplementation in a man with latent HSV or EBV infection can trigger viral reactivation and worsen the inflammatory burden that was already impairing eNOS function.

The alternative is L-citrulline, which the body converts to arginine endogenously at a controlled rate, avoiding the supraphysiological arginine spikes that can trigger viral reactivation. Citrulline also raises arginine plasma levels more effectively than arginine itself due to first-pass hepatic metabolism differences. But citrulline too must be used only after the eNOS system has been recoupled — adding citrulline to an uncoupled system is also counterproductive.

Recoupling the System

The sequence for recoupling eNOS and restoring nitric oxide production is not complicated, but the sequence matters. Do not skip to step 3:

- Step 1 — Reduce the oxidative burden: Eliminate inflammatory inputs (processed food, alcohol, seed oils, chronic sleep deprivation, unmanaged stress). Support glutathione production with NAC 600mg daily and glycine 3g daily. Vitamin C 1-2g daily protects BH4 from oxidative destruction.
- Step 2 — Restore BH4 cofactor support: Active methylfolate (5-MTHF), methylcobalamin, riboflavin (B2), and P5P together support BH4 regeneration via the DHFR and DHPR pathways. Without adequate B2, BH4 cannot be recycled from its oxidised form.
- Step 3 — Repair the glycocalyx: Pycnogenol 120-160mg daily and/or sulodexide (where available). This step restores the physical sensor mechanism that triggers NO production in response to blood flow.
- Step 4 — Reduce ADMA: Dietary nitrates (arugula, beetroot) provide an ADMA-independent route to NO production via the nitrate-nitrite-NO pathway, bypassing eNOS entirely while it recovers. Cacao flavonoids (theobromine + epicatechin) directly reduce ADMA and support eNOS expression.
- Step 5 — Only then consider L-citrulline: 3-6g daily once the above foundation is in place, typically after 6-8 weeks of steps 1-4.

Timeline: eNOS recoupling is not an overnight process. The endothelium turns over slowly. Meaningful functional improvement in NO production requires 8-16 weeks of consistent protocol adherence.

Chapter 4: The ATP Power Plant

Testosterone and DHT are manufactured products. The factory is your mitochondria. If the factory is broken, it does not matter how many raw materials are available — production stops.

Steroidogenesis and the Mitochondrial Link

Testosterone biosynthesis begins in the mitochondria of the Leydig cells in the testes. Cholesterol is transported into the inner mitochondrial membrane by the StAR protein (Steroidogenic Acute Regulatory protein). This transport step is the rate-limiting step in testosterone production — and it is entirely ATP-dependent.

Inside the mitochondria, cholesterol is converted to pregnenolone by the CYP11A1 enzyme. Pregnenolone then leaves the mitochondria and is converted through a series of cytosolic and endoplasmic reticulum enzymes to DHEA, androstenedione, and ultimately testosterone. The entire cascade requires NADPH — which is produced by mitochondrial function.

When mitochondrial ATP production is impaired — by SSRIs, finasteride, valproate, chronic LPS endotoxin exposure from gut dysbiosis, overtraining, long COVID, or age-related decline in Complex I and Complex III activity — the entire steroidogenic cascade slows or stops. The raw materials are present. The factory is offline.

> **Key insight:** A man with "low testosterone" may not have a testicular problem or a pituitary problem. He may have a mitochondrial problem. Standard endocrinology does not assess this.

The NAD+/NADPH Balance

One of the most important and most misunderstood relationships in biohacking is the NAD+/NADPH balance. The two molecules are related but functionally distinct:

- NAD+ is the oxidised form — the "energy currency" that drives the electron transport chain, ATP production, and sirtuin activation.
- NADPH is the reduced form — the "repair currency" that drives glutathione recycling, steroidogenesis (including DHT production via 5-alpha-reductase), and BH4 regeneration.

The popular biohacking practice of aggressively boosting NAD+ with NMN or NR without addressing NADPH risks shifting the redox balance toward oxidation — potentially depleting the very NADPH required for 5-alpha-reductase activity and DHT synthesis. This is a common cause of worsened hormonal symptoms in men who try high-dose NAD+ supplementation without proper cofactor support.

The correct approach is to support both sides of the balance simultaneously: B-vitamins (especially B1, B2, B3) as cofactors for the electron transport chain, magnesium as a universal cofactor for over 300 enzymatic reactions including those

in the mitochondrial matrix, and antioxidant support (glutathione precursors, CoQ10) to prevent electron leakage and NADPH depletion.

> *"Most people do not need NAD+ boosters. They need to fix what is burning through NAD+ in the first place — inflammation, gut dysbiosis, poor sleep, chronic stress."*

The Mitochondrial Reboot Stack

The following compounds support mitochondrial function in the sequence that matters:

- Magnesium glycinate or malate: 400mg daily. The single most important mitochondrial cofactor. Magnesium is required for ATP synthesis (ATP exists in the cell as Mg-ATP), for over 300 enzymatic reactions, and for NADPH production. Deficiency is nearly universal in Western populations.
- CoQ10 (as Ubiquinol, not Ubiquinone): 200mg daily with fat. Ubiquinol is the reduced, active form. It functions as an electron shuttle in the inner mitochondrial membrane (Complex I to Complex III) and as a membrane-bound antioxidant. Without adequate CoQ10, the electron transport chain cannot function efficiently regardless of substrate availability.
- Shilajit (purified Mumio, standardised to >60% fulvic acid): 300mg daily. Shilajit has been shown to enhance the efficiency of CoQ10 by supporting the NADH:CoQ10 electron transfer ratio. It also contains dibenzo-alpha-pyrones which directly support mitochondrial electron transport. The sourcing and purity are critical — most commercial shilajit products are contaminated with heavy metals. Only use pharmaceutical-grade, third-party tested sources.
- PQQ (Pyrroloquinoline Quinone): 20mg daily. PQQ supports mitochondrial biogenesis — the creation of new mitochondria — via activation of the PGC-1alpha pathway. This is distinct from optimising existing mitochondria; PQQ actually increases the number of mitochondria in the cell.
- Active B-Complex: Thiamine (B1, as TTFD or sulbutiamine for CNS penetration), riboflavin (B2 as R5P), niacinamide (B3, 250mg — not higher, to avoid sirtuin inhibition), and pantothenic acid (B5) are all required as cofactors at specific steps in the Krebs cycle and electron transport chain.

Note on NMN/NR: if you choose to use these NAD+ precursors, pair them with TMG or choline to offset the methyl-donor drain they create via the NNMT clearance pathway. Begin at low doses (125-250mg) and monitor for signs of methylation depletion: fatigue, low mood, elevated Homocysteine on retest.

Chapter 5: The Prolactin Blockade

Prolactin is one of the most important and most overlooked hormones in male sexual health. It is routinely ignored in standard men's health panels, listed as "normal" at levels that are functionally damaging, and never investigated as a root cause of the symptoms it directly creates.

What Prolactin Actually Does

Prolactin is a peptide hormone produced by the anterior pituitary. In women, it drives milk production after childbirth. In men, it serves as a post-orgasmic regulatory signal — it is part of the mechanism that creates the refractory period after ejaculation.

When chronically elevated in men, prolactin creates a permanent biochemical refractory state:

- It directly antagonises dopamine signalling at the D2 receptor — reducing libido, motivation, and the ability to initiate and sustain arousal.
- It blocks 5-alpha-reductase activity, reducing the conversion of testosterone to DHT. This is the pathway responsible for penile tissue health, erection quality, and sexual sensitivity.
- It suppresses LH (luteinising hormone) secretion from the pituitary, reducing the stimulus for testosterone production in the Leydig cells.
- It promotes the growth of estrogen-sensitive tissue (gynecomastia) and causes water retention via its action on renal tubular cells.
- It chronically elevates serotonin tone — creating a negative spiral with the 5-HT2C receptor overactivation described in Book I.

The Target Range

Standard laboratory "normal" for male prolactin is typically listed as up to 15-18 ng/mL. From the perspective of optimised male sexual function, this range is far too permissive. Clinical evidence and coaching data consistently show that:

- Above 10 ng/mL: measurable suppression of dopaminergic tone, beginning DHT blockade, noticeable libido reduction.
- Above 15 ng/mL: significant sexual dysfunction in most men, warranting investigation of underlying cause (microadenoma, medication, gut dysbiosis).
- 4-7 ng/mL: optimal range for male sexual function, dopaminergic tone, and DHT conversion.

If your prolactin is consistently above 15 ng/mL, request an MRI of the pituitary to rule out a prolactinoma (prolactin-secreting microadenoma) before pursuing natural intervention. This is a straightforward, treatable condition that is frequently missed.

The Root Cause: LPS Endotoxins and Gut Health

Chronically elevated prolactin in the absence of a pituitary tumour or medication cause almost always traces back to gut dysbiosis and LPS (lipopolysaccharide) endotoxin load. LPS is a component of the outer membrane of gram-negative bacteria in the gut. When gut barrier integrity is compromised — a condition known as intestinal hyperpermeability or "leaky gut" — LPS translocates into systemic circulation.

Systemic LPS drives chronic low-grade inflammation, suppresses mitochondrial function (directly impairing steroidogenesis), and elevates prolactin via its action on pituitary lactotroph cells. This is the root-cause chain: poor diet and gut dysbiosis → leaky gut → LPS endotoxaemia → elevated prolactin → blocked DHT → impaired erections.

Fixing the gut — through dietary reform (reducing processed food, alcohol, gluten in sensitive individuals), targeted probiotic support, butyrate-producing prebiotic fibres, and addressing SIBO where present — is therefore foundational to prolactin normalisation. No supplement protocol will fully lower prolactin against a backdrop of chronic LPS endotoxaemia.

Natural Prolactin-Lowering Strategy

- Fix the gut first: dietary reform, reduce alcohol and processed food, consider GI-MAP testing if symptoms of gut dysbiosis are present (bloating, irregular stool, food sensitivities).
- Vitamin B6 as P5P: 50mg twice daily. P5P is the active form of B6 and is a direct modulator of prolactin at the pituitary. Do not use standard pyridoxine hydrochloride at high doses — chronic high-dose pyridoxine HCl can cause peripheral neuropathy. P5P does not carry this risk at therapeutic doses.
- Tribulus terrestris (standardised to ≥50% protodioscin, Bulgarian-sourced): the protodioscin fraction has demonstrated prolactin-lowering activity of up to 70% in some research. Cycle: 5 weeks on, 2 weeks off. Most commercial Tribulus products are standardised to saponins from Indian or Chinese sources with very low protodioscin content — they are largely ineffective. Bulgarian standardised Tribulus is a meaningfully different product.
- Vitamin E (tocotrienol fraction, not tocopherol): 200-400 IU of mixed tocotrienols. Tocotrienols, unlike tocopherols, have demonstrated prolactin-modulating activity in clinical research.
- Improve sleep quality: prolactin has a circadian pattern with peaks during sleep. Disrupted REM sleep directly elevates daytime prolactin levels. Address sleep before adding pharmacological or supplement interventions.
- Reduce alcohol and wheat: both directly stimulate prolactin secretion via distinct mechanisms. Even moderate alcohol consumption produces measurable prolactin elevation within hours of ingestion.

Timeline: Expect 8-12 weeks to see meaningful prolactin reduction from natural interventions. Test at baseline and at 12 weeks.

Chapter 6: The T3/DHT Conversion Pathway

DHT — dihydrotestosterone — is the androgen that matters most for erectile quality, penile tissue health, and male sexual drive. It binds to the androgen receptor with three to ten times the affinity of testosterone. And most men optimising for testosterone are ignoring the one enzyme that converts testosterone into DHT.

The 5-Alpha-Reductase System

5-alpha-reductase (5-AR) converts testosterone to DHT and is present in two primary isoforms in humans: Type 1 (expressed primarily in the liver, skin, and brain) and Type 2 (expressed primarily in the prostate, seminal vesicles, and skin of the genitals). Both isoforms contribute to systemic DHT production, which is why finasteride (which blocks only Type 2) produces less severe side effects than dutasteride (which blocks both isoforms).

The activity of 5-AR is governed by several upstream regulators. In order of potency:

- Free T3 (triiodothyronine — active thyroid hormone): the single most important driver of 5-AR activity. Research consistently shows that hypothyroidism — even subclinical, with TSH in the "normal" range — dramatically reduces 5-AR activity and DHT conversion.
- IGF-1 (insulin-like growth factor 1): the second most potent driver. IGF-1 and DHT work synergistically — IGF-1 is required for 5-AR upregulation, and DHT feeds back to support IGF-1 production. Low IGF-1 creates a downward spiral that DHT supplementation alone cannot break.
- DHT itself: DHT feeds back positively to support 5-AR expression in a short-loop cycle. This is one reason why some men struggle to raise DHT: the enzyme is underexpressed because DHT is low, but DHT is low because the enzyme is underexpressed.

The Keto/Carnivore DHT Trap

Low-carbohydrate diets — ketogenic and carnivore diets in particular — are marketed extensively to men interested in testosterone optimisation. The evidence on this is more nuanced than the marketing suggests.

The liver requires carbohydrates for optimal T4-to-T3 conversion. The enzyme responsible for this conversion (deiodinase type 1) is glucose-sensitive — its activity decreases significantly under carbohydrate restriction. When T3 falls, 5-AR activity follows. The result is a man with adequate or even elevated total testosterone but severely impaired DHT conversion.

This pattern — high total testosterone, low DHT — is the classic presentation of a man on a strict low-carbohydrate diet. He may have strong muscle-building signals (testosterone is anabolic) but poor erectile quality, low sexual sensitivity, and declining penile tissue health (all DHT-dependent functions).

The minimum carbohydrate intake required to maintain T3 and 5-AR activity is approximately 100-150g per day from clean sources. The optimal range for most men pursuing hormonal health is 150-250g daily from quality sources: potatoes, rice, fruit, oats. This is not a license for processed carbohydrates — the glycaemic profile and insulin response matter.

> *"Stop competing with your own DHT. Clean carbohydrates are not the enemy. The thyroid-androgen pathway requires them. Penile tissue health requires DHT. DHT requires T3. T3 requires carbohydrates."*

Supporting 5-AR Activity Naturally

Beyond adequate carbohydrate intake and thyroid support, the following interventions directly or indirectly support 5-AR activity:

- Optimise thyroid function: test Free T3 and Free T4, not just TSH. Aim for Free T3 in the upper third of the reference range. If Free T3 is low despite normal TSH, address conversion blockers (selenium deficiency, excess stress, chronic inflammation).
- Optimise IGF-1: target ≥ 200 ng/mL. Key drivers of IGF-1: adequate sleep (80% of GH/IGF-1 is released during deep sleep), dietary protein ≥ 1.6g/kg bodyweight, resistance training, and adequate zinc.
- Zinc: a direct cofactor for 5-AR. Zinc deficiency — extremely common in Western men — dramatically reduces 5-AR activity. Target 15-25mg zinc daily (as glycinate or picolinate). Note: zinc repletion can take up to 6 months to restore full tissue-level sufficiency.
- Vitamin D (as D3 with K2): target serum 25(OH)D of 50-70 ng/mL. Vitamin D receptors are expressed on Leydig cells and in penile tissue. Deficiency impairs both testosterone production and 5-AR activity.
- Avoid all 5-AR inhibitors: finasteride, dutasteride, saw palmetto, pygeum, stinging nettle (high-dose continuous), rosemary oil (topical), pumpkin seed oil, curcumin (high-dose), quercetin (high-dose), DIM. This list includes several compounds marketed for "prostate health" or "testosterone support" that directly impair DHT production.

Penile Tissue Health and DHT Dependence

DHT is not just a libido molecule. It is the primary androgen responsible for maintaining the structural integrity and functional health of penile tissue. The smooth muscle of the corpus cavernosum is androgen-sensitive, and DHT (not testosterone) is the primary androgen maintaining its contractile properties, elasticity, and responsiveness.

Chronic DHT deficiency — whether from 5-AR inhibitor use, hypothyroidism, or dietary restriction of carbohydrates — leads to progressive changes in penile smooth

muscle: increased collagen deposition, reduced elasticity, and reduced vascular responsiveness. In men who have used finasteride or dutasteride for extended periods, these changes can become significant and partially irreversible.

Addressing DHT deficiency early — before structural changes have progressed — is therefore not merely a quality-of-life issue. It is tissue preservation.

Chapter 7: The REM Sleep Blueprint

Sleep is not a recovery state. It is an active biological process with specific hormonal, neurological, and vascular functions that cannot be substituted by any supplement or intervention during waking hours.

For male sexual health, REM sleep is the critical variable — not total sleep duration, not sleep efficiency as measured by wearables, but the quantity and quality of REM specifically.

Nocturnal Penile Tumescence: The 3am Workout

Nocturnal Penile Tumescence (NPT) — spontaneous erections during sleep — occurs during REM phases and serves a critical physiological function. These erections are not random. They are a maintenance programme for penile tissue.

During NPT, oxygen-rich blood is pumped through the corpus cavernosum, oxygenating the smooth muscle and preventing the hypoxia that would otherwise lead to progressive collagen deposition and fibrosis. Men who sleep normally experience 3-6 NPT episodes per night, each lasting 20-40 minutes, for a total of 1.5-3 hours of penile oxygenation every night.

Men with sleep apnoea, REM suppression (from alcohol, cannabis, SSRIs, or sedatives), or chronic sleep deprivation lose most or all of this nightly maintenance programme. Over months and years, this leads to progressive penile fibrosis and the vascular dysfunction that manifests as erectile difficulty.

> *"If you do not wake up with morning erections, you are not getting adequate REM. Your penile tissue is not being maintained. This is a medical issue, not a "getting older" issue."*

Morning erections are simply the last NPT episode of the night extending into wakefulness. Their absence is a reliable clinical sign of insufficient REM sleep or impaired NPT, and warrants investigation.

The Hormonal Cascade of REM

The hormonal events of REM sleep are not reproducible during waking hours:

- Testosterone: the majority of daily testosterone production occurs during sleep, with the highest pulse amplitude during the early hours of sleep. Acute sleep deprivation (one night of 5 hours or less) reduces testosterone by 10-15%. Chronic sleep restriction compounds this deficit significantly.
- Growth hormone / IGF-1: 80% of daily GH secretion occurs during slow-wave sleep (the stage immediately preceding REM). GH drives IGF-1 production, which as noted in Chapter 6, is required for optimal 5-AR activity and DHT conversion.

- DHEA: DHEA secretion is strongly linked to REM sleep quality and quantity. DHEA is an androgen precursor that feeds approximately 40% of total androgen synthesis.
- Cortisol/testosterone competition: cortisol and testosterone are both synthesised from cholesterol, and they compete for the same mitochondrial substrate (StAR protein transport). When sleep quality is poor, the cortisol circadian rhythm is disrupted, producing elevated morning and evening cortisol that suppresses testosterone production at the hypothalamic, pituitary, and testicular levels simultaneously.

Optimising REM Sleep for Vascular and Hormonal Health

The following interventions are organised in priority order — address them in sequence, not simultaneously:

- Rule out and treat sleep apnoea: sleep apnoea is the single most common and most under-diagnosed cause of impaired NPT and impaired testosterone production in men over 35. Symptoms: snoring, daytime fatigue, non-restorative sleep, morning headaches, frequent nocturnal urination. Diagnosis: overnight polysomnography or home sleep study. Treatment (CPAP or positional therapy) restores NPT and testosterone within weeks.
- Eliminate REM suppressants: alcohol (even 2 drinks suppresses REM for 4-6 hours), cannabis (particularly high-THC products), benzodiazepines, Z-drugs (zopiclone, zolpidem), antihistamines, and SSRIs all significantly impair REM. If medications cannot be changed, discuss this directly with your prescribing physician.
- Taurine 2-3g and Magnesium glycinate 400mg before bed: taurine is a GABA-A agonist that promotes delta wave sleep onset and cortisol reduction. Magnesium glycinate supports the conversion of cortisol to the less biologically active cortisone via 11-beta-HSD2 and reduces nocturnal cortisol generally.
- Circadian rhythm anchoring: consistent wake time (more important than bedtime), morning light exposure within 30 minutes of waking (10-20 minutes of outdoor light or a 10,000 lux light box), and dark/cool sleeping environment. Circadian disruption is one of the fastest ways to fragment REM.
- DHEA (7.5-25mg before bed): DHEA has an established relationship with REM sleep quality. Supplementation before bed has shown REM-enhancement in some research, particularly in men over 40 with documented low DHEA-S. Begin at the lowest dose and titrate up based on morning energy and mood. Test DHEA-S before and after.
- Melatonin (0.3-1mg): the physiological dose, not the 5-10mg mega-doses commonly sold. Melatonin does not sedate — it signals circadian phase. Physiological doses taken 90 minutes before desired sleep onset are more effective than high doses at bedtime for circadian rhythm support.

Chapter 8: The Vascular Restoration Stack

Everything in the preceding chapters has been building to this: a coherent, sequenced, 90-day restoration protocol that addresses the vascular foundation layer by layer.

The goal of this protocol is not a pump. It is not a single impressive erection. It is endothelial integrity — the systematic rebuilding of the biological infrastructure that makes erections reliable, sustainable, and independent of pharmaceutical assistance.

The Sequencing Principle

The single biggest mistake men make when approaching vascular restoration is starting with the wrong layer. They take citrulline without fixing eNOS uncoupling. They take testosterone without fixing mitochondrial function. They take DHT support without fixing the thyroid. Every intervention in this stack depends on the ones below it.

The sequence is non-negotiable:

- Foundation first: gut health, methylation, mitochondria, sleep. Nothing else works efficiently without these.
- Repair second: glycocalyx, eNOS recoupling, homocysteine lowering.
- Optimise third: prolactin reduction, DHT support, NO pathway enhancement.
- Amplify last: dopaminergic herbs, adaptogens, advanced peptides. These are signal amplifiers. Applied to a broken foundation, they amplify dysfunction, not function.

The 90-Day Protocol

Weeks 1-4: The Foundation

- Gut reform: eliminate alcohol, processed food, seed oils. Add 30g+ dietary fibre daily. Consider a GI-MAP stool test if gut symptoms are present.
- Methylation support: Active B-Complex (5-MTHF + methylcobalamin + P5P + riboflavin) — one dose morning with food. Add TMG 1g daily if Homocysteine is known to be elevated.
- Mitochondrial foundation: Magnesium glycinate 400mg (split: 200mg morning, 200mg before bed). CoQ10 as Ubiquinol 200mg with largest meal.
- Sleep: address REM suppression, establish consistent wake time, begin Taurine 2g + Magnesium glycinate 200mg at bedtime.
- Thyroid: test Free T3, Free T4, and Reverse T3. Add 150mcg selenium (as selenomethionine) daily to support T4-to-T3 conversion.

Weeks 5-8: The Repair

- Glycocalyx repair: add Pycnogenol 120mg daily (split: 60mg morning, 60mg afternoon with food).
- BH4 protection: Vitamin C 1g twice daily. Cacao (20-30g raw cacao or 90%+ dark chocolate) for epicatechin and theobromine.
- ADMA reduction: include arugula (rocket) 80-100g daily — the highest dietary source of nitrates for the nitrate-nitrite-NO pathway.
- Mitochondrial depth: add Shilajit (pharmaceutical grade, >60% fulvic acid) 300mg daily. Add PQQ 20mg daily.
- Prolactin: if tested and elevated, add P5P 50mg twice daily and begin Tribulus (Bulgarian, ≥50% protodioscin) at 500mg daily.
- Homocysteine retest at week 8: adjust methylation protocol based on results.

Weeks 9-12: The Optimisation

- NO pathway: add L-Citrulline 3g daily (only at this stage — not before eNOS recoupling is underway).
- DHT support: ensure carbohydrate intake is 150-250g daily from clean sources. Confirm zinc at 15-25mg daily.
- IGF-1 optimisation: resistance training 3-4x weekly (compound movements — squat, deadlift, press). Adequate protein ≥1.6g/kg. Review sleep quality for GH pulse optimisation.
- Vitamin D3 + K2: test 25(OH)D and aim for 50-70 ng/mL. Typical supplementation: 3000-5000 IU D3 with 100mcg MK-7 (K2) daily.
- Advanced (if plateaued): consider vacuum erection device (low pressure, 4-6 in Hg, 10 min daily) for mechanical penile oxygenation and fibrosis prevention. Consider acoustic wave therapy through a qualified provider if structural vascular remodelling is required.

The Maintenance Stack (Post-90 Days)

Once the restoration protocol is complete, the following forms the ongoing maintenance layer:

- Active B-Complex: daily, ongoing
- Magnesium glycinate: 400mg daily, ongoing
- CoQ10 as Ubiquinol: 100-200mg daily, ongoing
- Pycnogenol: 60-80mg daily for ongoing glycocalyx protection
- Vitamin D3 + K2: dose adjusted to maintain 25(OH)D at 50-70 ng/mL
- Arugula / dietary nitrates: daily dietary inclusion
- Tribulus (if prolactin management ongoing): cycled 5 weeks on / 2 weeks off
- Zinc: 15mg daily ongoing

Test every 6 months: Homocysteine, prolactin, DHT, Free T3, 25(OH)D, hs-CRP.

> *"The Vascular Foundation is not a 90-day programme. It is the permanent baseline of male biological health. Once established, it requires relatively modest maintenance. The cost of neglecting it compounds silently for years before it becomes impossible to ignore."*

Appendix A: Lab Targets Reference

Functional targets for vascular, hormonal, and sexual health — not standard lab "normal ranges."

Homocysteine (fasting): < 8 µmol/L (ideally 5-8)

Standard labs say <15. Above 8 is active endothelial injury. Test annually.

Prolactin: 4-7 ng/mL

Standard "normal" up to 15-18 ng/mL is functionally damaging. If >15, rule out pituitary adenoma.

DHT: ≥ 10% of Total Testosterone

Low DHT with normal T = impaired 5-AR activity. Check T3 and avoid 5-AR blockers.

Free T3: Upper third of reference range

The primary driver of 5-alpha-reductase activity. TSH alone is insufficient.

Free T4: Mid-range or above

Assess conversion to T3 via Reverse T3 if Free T3 is low with normal T4.

Total Testosterone: ≥ 500 ng/dL

Total T alone is not the key metric. DHT, SHBG, and prolactin are equally important.

SHBG: 20-40 nmol/L

High SHBG = low free T/DHT. Address via diet (adequate carbs), liver support, boron.

Estradiol (E2): 20-30 pg/mL

Excess estradiol causes venous leakage in the corpus cavernosum and suppresses LH.

IGF-1: ≥ 200 ng/mL

Required for 5-AR upregulation and DHT-IGF-1 feed-forward cycle. Optimise via sleep and training.

DHEA-S: ≥ 300 µg/dL (age-adjusted)

Feeds ~40% of androgen synthesis. Drops under chronic LPS and stress burden.

Fasting Insulin: < 10 µIU/mL

Insulin resistance drives SHBG elevation, impairs eNOS, and promotes LPS translocation.

25(OH) Vitamin D: 50-70 ng/mL

Below 30 ng/mL impairs testosterone production, 5-AR activity, and penile tissue health.

hs-CRP: < 1.0 mg/L

Primary inflammatory marker. Above 1 indicates active BH4 depletion and ADMA production.

Ferritin: 50-150 ng/mL

Iron is required for eNOS haem cofactor. High ferritin (>200) generates oxidative stress.

Active B12 (holo-TC): > 400 pmol/L

Standard B12 serum tests are unreliable. Test holotranscobalamin specifically.

RBC Folate: High normal

Reflects intracellular folate stores over 3 months. Serum folate is an unreliable proxy.

Appendix B: Supplements and Substances to Avoid

These compounds are commonly used or prescribed but have documented mechanisms that undermine vascular health, hormonal balance, or endothelial function in men.

Finasteride / Dutasteride

Pharmaceutical 5-AR inhibitors. Finasteride blocks Type 2; dutasteride blocks both isoforms. Post-finasteride syndrome (PFS) involves epigenetic silencing of the 5-AR gene, neurosteroid depletion, and GABA-A receptor changes. In susceptible men, these effects are persistent after discontinuation.

Saw Palmetto

Over-the-counter 5-AR inhibitor with comparable mechanism to finasteride. Widely included in "prostate support" supplements. Crashes have been reported that are indistinguishable from PFS. Avoid entirely.

Pygeum

Strong 5-AR inhibitory activity. Almost always included alongside saw palmetto in prostate-support blends. Avoid.

Rosemary oil (topical or high-dose oral)

Demonstrated 5-AR inhibitory activity comparable to topical finasteride in some studies. Avoid topical application to the scalp or skin if preserving DHT is a priority.

Pumpkin seed oil (high-dose)

Documented 5-AR inhibitory activity. Small culinary amounts are safe; supplemental doses (1g+ daily) are not.

SSRIs / SNRIs

Beyond their well-documented sexual side effects via serotonin excess and dopamine suppression, SSRIs have demonstrated direct mitochondrial toxicity, impair CoQ10-dependent electron transport, and can cause persistent sexual dysfunction (PSSD) after discontinuation in susceptible individuals. Any SSRI changes must be managed with your prescribing physician.

Statins (without CoQ10)

Statins deplete CoQ10 by blocking the mevalonate pathway — the same pathway used to synthesise both cholesterol and CoQ10. If statins are prescribed and cannot be discontinued, CoQ10 supplementation (200-400mg Ubiquinol daily) is non-negotiable.

Alcohol (regular use)

Raises prolactin, raises estradiol (via CYP19A1 aromatase in the liver), depletes B-vitamins, disrupts REM sleep, and directly impairs endothelial function. Even moderate regular consumption (>7 units/week) produces measurable hormonal damage.

High-dose Arginine (without BH4 support)

Counterproductive and potentially harmful in men with eNOS uncoupling, elevated Homocysteine, or latent viral infections. Use L-citrulline instead, and only after Weeks 1-8 of the restoration protocol.

IV NAD+ Drips

Cause heavy methyl-donor depletion via NNMT clearance, transiently raise Homocysteine, and produce a plasma spike with minimal cellular uptake compared to oral NAD+ precursors. The cost-benefit ratio does not support their use in this context.

Curcumin / Turmeric (high-dose)

Moderate 5-AR inhibitory and anti-androgenic activity at supplement doses (500mg+). Culinary turmeric in food is safe. High-dose curcumin supplements are not appropriate during vascular and hormonal restoration.

DIM (Diindolylmethane)

Despite being marketed as an "estrogen blocker," DIM at doses above 100mg acts as a testosterone blocker — an mTOR inhibitor and androgen receptor antagonist. Avoid entirely.

Ibuprofen / Paracetamol (chronic daily use)

Both reduce androgen receptor expression. Occasional use is acceptable. Chronic daily use during this protocol is counterproductive.

Appendix C: The WOAH Coaching Programme

This book is yours because you are a member of the WOAH coaching ecosystem. It is not available for purchase separately. It is designed to be used alongside the AI coach, not instead of it.

The three books of The Confidence Reset:

- Book I — Rewiring Confidence: The neurological and psychological layer. The nervous system, neurotransmitter architecture, performance anxiety loop, and the 30-day rewiring protocol.
- Book II — The Vascular Foundation (this book): The endothelial, circulatory, hormonal, and mitochondrial layer. The physical infrastructure of erections.
- Book III — Talking About It: The relationship and partner communication layer. Navigating intimacy and confidence together as a couple.

The WOAH AI coaching programme offers:

- Unlimited AI coach access — 24/7, voice-enabled, completely private
- Personalised protocol design based on your lab results and history
- Weekly signal tracking and protocol recalibration (Performance tier)
- Private consultation calls with the clinical team (Sovereign tier)

Access your coaching: **woah.love** Email: **office@woah.love**