THE STUB CLUB PROTOCOL

TIER 2

THE ENDOTHELIAL OVERHAUL

Pipe & Flow Restoration

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PREREQUISITE: This is Tier 2. If you have not completed 90 days of Tier 1, completed the 30-day check, and confirmed that your Homocysteine is trending below 8 µmol/L and your Prolactin is trending toward the 4-7 ng/mL range, go back to Tier 1. The compounds in Tier 2 are signal amplifiers. They amplify whatever state the vascular system is currently in. Applied to a broken, inflamed, uncoupled system, they make the damage worse.

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Introduction: The Pipes Are the Problem

Tier 1 stopped the internal rust. It lowered Homocysteine, began the Prolactin reset, restored mitochondrial ATP production, protected the testes from BPA, and repaired the methylation cycle that underlies every other biological function in this protocol.

Tier 2 rebuilds the pipes.

The endothelium — the single-cell-thick lining of every blood vessel in your body — is the most important organ you have never heard of. It is the site of nitric oxide production, the regulator of vascular tone, the primary determinant of how blood flows through the penile arteries, and the structure that has been under sustained attack from every inflammatory, dietary, and toxic insult your body has absorbed.

When the endothelium is damaged, no amount of testosterone, no PDE5 inhibitor, no dopaminergic herb, and no amount of willpower produces reliable erectile function. You are trying to pump water through rusted, narrowed, unresponsive pipes. The pressure does not matter. The pipes are the problem.

> *"Standard medicine treats erectile dysfunction as a symptom to suppress. The Stub Club Protocol treats it as a pipe problem to solve. There is a difference between forcing flow through a broken system and rebuilding the system so flow becomes natural."*

Tier 2 is the pipe rebuild. It is more complex than Tier 1, it requires more patience — the glycocalyx takes 4-7 months to meaningfully restore — and it requires the Tier 1 foundation to be in place or it will not fully work. But when it does work, the effects are not a temporary chemical override. They are a restored biological system.

This book covers the full endothelial mechanism, the five-step sequenced protocol, six specific compounds with full sourcing and timing rationale, the dietary interventions that accelerate the repair, the devices that support tissue maintenance during the rebuild, and the lab markers that tell you whether it is working.

Chapter 1: The Endothelium — What It Is and Why It Failed

Before you spend money on Tier 2 compounds, you need to understand exactly what they are repairing. The endothelium is not a passive tube. It is an active endocrine organ. Understanding it at that level is what allows you to use the compounds correctly.

The Endothelium as an Endocrine Organ

The endothelium is a monolayer of cells — one cell thick — lining the interior surface of every blood vessel in the body. Its total surface area in an adult male is approximately 700 square metres. It produces over 30 biologically active substances, including nitric oxide, prostacyclin, endothelin-1, von Willebrand factor, and tissue plasminogen activator.

For male sexual function, the most critical of these is nitric oxide — specifically, the nitric oxide produced by the eNOS (endothelial nitric oxide synthase) enzyme in response to blood flow shear stress across the endothelial surface. This NO diffuses into the smooth muscle of the corpus cavernosum, activates guanylate cyclase, produces cGMP, and causes the smooth muscle to relax — allowing blood to fill the erectile chambers.

PDE5 inhibitors work by blocking the enzyme that breaks down cGMP. They extend the signal. They cannot create the signal if eNOS is not producing nitric oxide. If the endothelium is damaged, the primary source of the signal is gone.

The Glycocalyx: The Layer That Does the Sensing

The inner surface of the endothelium is not bare. It is coated with a complex gel-like layer called the glycocalyx — a mesh of proteoglycans, glycoproteins, and glycolipids that extends 0.5-4.5 micrometres into the vascular lumen. This layer is the primary mechanosensor of blood flow.

When blood flows across the glycocalyx, the resulting shear stress deforms the glycocalyx structure. This mechanical deformation is transduced into a biochemical signal that tells the endothelial cell to activate eNOS and produce nitric oxide. The glycocalyx is therefore the sensory organ that initiates the entire NO production cascade. Without an intact glycocalyx, the endothelial cell does not receive the shear stress signal correctly, eNOS is not activated efficiently, and NO production falls regardless of how much arginine or citrulline is being supplemented.

The glycocalyx is destroyed by:

- Elevated blood glucose — even transient post-meal spikes above 140 mg/dL cause rapid, measurable glycocalyx shedding via enzymatic cleavage of heparan sulphate chains.

- Systemic inflammation — TNF-alpha, IL-6, and IL-1beta all promote glycocalyx degradation via matrix metalloproteinase (MMP) activation.
- Elevated homocysteine — as covered in Tier 1, Homocysteine generates reactive oxygen species directly at the endothelial membrane and promotes glycocalyx breakdown.
- LPS endotoxins — bacterial LPS activates endothelial toll-like receptor 4 (TLR4), triggering an inflammatory response that includes glycocalyx shedding.
- Ischaemia/reperfusion — brief periods of reduced blood flow followed by restoration produce reactive oxygen species that damage the glycocalyx.
- Oxidised LDL — promotes MMP activation and glycocalyx degradation.

Critical point: You cannot supplement your way to NO production if the glycocalyx is stripped. The sensor must be rebuilt before the signal can be generated. This is why the sequencing in Tier 2 starts with glycocalyx repair, not NO boosters.

eNOS: Coupled vs. Uncoupled — The Difference Between Building and Burning

eNOS in its normal, "coupled" state converts L-arginine to nitric oxide and L-citrulline. This conversion requires several cofactors in the correct stoichiometry: BH4 (tetrahydrobiopterin), NADPH, FAD, FMN, haem, and calmodulin.

When BH4 is depleted — by oxidative stress, peroxynitrite, elevated Homocysteine, inflammation, and LPS — the eNOS enzyme undergoes a conformational change that uncouples the electron transfer pathway. In the uncoupled state, eNOS no longer converts arginine to NO. Instead, it transfers electrons directly to molecular oxygen, producing superoxide — the same free radical used by neutrophils to kill bacteria.

Superoxide in the vascular wall then reacts with any remaining nitric oxide to form peroxynitrite — one of the most destructive reactive nitrogen species in biology. Peroxynitrite: destroys more BH4 (completing the vicious cycle), oxidises BH2 (the spent form of BH4, which cannot be recycled without adequate B2), nitrates tyrosine residues in proteins, damages mtDNA, and promotes further glycocalyx degradation.

> *"Adding more arginine or citrulline to an uncoupled eNOS system does not produce more nitric oxide. It produces more superoxide. You are feeding the fire, not the engine."*

This is the single most important concept in Tier 2. The sequencing rule — recouple before you boost — is not arbitrary. It is the difference between the protocol producing results and the protocol making the underlying condition worse.

Why the Penile Arteries Fail First

The internal pudendal artery and its branches that supply the corpus cavernosum are among the smallest calibre arteries in the body — 1-2mm in internal diameter. By

comparison, the coronary arteries are 2-4mm in diameter and the carotid arteries are 6-8mm.

Endothelial dysfunction follows a predictable pattern: smallest vessels first. When the glycocalyx is damaged and eNOS is uncoupled systemically, the functional deficit becomes clinically apparent first in the vessels with the smallest margin for reduced NO production — the penile arteries. The same endothelial dysfunction exists in the coronary arteries at this stage, but the margin is larger and the symptoms have not yet appeared.

This is the clinical basis for the well-documented finding that erectile dysfunction in men under 60 is a significant independent predictor of future major cardiovascular events, typically preceding cardiac symptoms by 3-5 years. The endothelial dysfunction is the same. The penile arteries just show it first.

Fixing endothelial function in Tier 2 is therefore not just about erectile performance. It is comprehensive cardiovascular preventive medicine. The two outcomes are biologically inseparable.

Chapter 2: The Arginine Trap — And What to Use Instead

The standard internet recommendation for erectile dysfunction is L-arginine. It is on every "natural ED supplement" label. It is the active ingredient or the claimed mechanism in dozens of commercial products. It is, in many cases, exactly the wrong intervention.

Why Arginine Fails — and When It Makes Things Worse

Arginine supplementation fails for two independent reasons in men with endothelial dysfunction:

First: the ADMA blockade. ADMA (Asymmetric Dimethylarginine) is the endogenous inhibitor of eNOS, produced in excess when Homocysteine is elevated and when endothelial cells are under inflammatory stress. ADMA and arginine compete for the same binding site on eNOS. When ADMA levels are high — as they are in any man with elevated Homocysteine and endothelial inflammation — adding more arginine does not overcome the competition. ADMA wins because it binds eNOS with higher affinity under inflammatory conditions.

Second: the uncoupled eNOS problem. As described in Chapter 1, when BH4 is depleted, eNOS converts arginine to superoxide rather than nitric oxide. High-dose arginine supplementation in a man with uncoupled eNOS accelerates superoxide production. The result is increased oxidative stress, further BH4 depletion, further eNOS uncoupling, and more peroxynitrite damage to the endothelium. The man is paying to make his vascular health worse.

The Additional Risk: Viral Reactivation

There is a third reason to be cautious with high-dose arginine that is almost completely absent from men's health discussions: arginine is the primary amino acid substrate required for replication by several latent pathogens that have high seroprevalence in the adult male population.

Herpes simplex virus (HSV-1, HSV-2), Epstein-Barr virus (EBV), and several biofilm-forming bacteria including Ureaplasma urealyticum, Mycoplasma genitalium, and Pseudomonas aeruginosa all preferentially utilise arginine as a fuel and replication substrate. A man with latent HSV-1 (estimated 67% of adults globally) who begins high-dose arginine supplementation may experience increased viral reactivation — cold sores, nerve pain, or subclinical viral activity that adds to the systemic inflammatory burden at exactly the time the Tier 2 protocol is attempting to reduce it.

L-lysine competes with arginine for intestinal absorption and for cellular uptake. If you choose to use citrulline in the later stages of Tier 2, pairing it with 1-2g of L-lysine daily provides partial protection against arginine-driven viral reactivation.

The Correct NO Strategy: The Nitrate-Nitrite-NO Pathway

When eNOS is uncoupled, the correct approach to NO support is to bypass eNOS entirely using the dietary nitrate-nitrite-NO pathway. This pathway does not require eNOS. It does not require BH4. It does not require any cofactor that might be depleted by the inflammatory state you are trying to repair.

The pathway works as follows: dietary nitrate (NO3-) from food is reduced to nitrite (NO2-) by commensal bacteria on the tongue and in the gut. Nitrite is then further reduced to nitric oxide (NO) in the blood and tissues, particularly in hypoxic conditions — which is exactly the condition in ischaemic or poorly perfused penile tissue.

The highest dietary nitrate sources, in order:

- Arugula (rocket): the highest nitrate concentration of any commonly available food — approximately 480mg nitrate per 100g. A 100g serving of arugula provides a clinically relevant nitrate dose. This is why arugula specifically — not beetroot, not beet juice, not beetroot powder — is the dietary intervention specified in Tier 2. Beetroot is a distant second.
- Raw cacao / dark chocolate (90%+): provides epicatechin and theobromine, which directly reduce ADMA levels, support eNOS expression, and provide flavonol-based antioxidant protection for BH4. 20-30g of raw cacao or a 20-30g serving of 90%+ dark chocolate provides a meaningful dose of these compounds daily.
- Pomegranate juice (100%, no added sugar): contains punicalagins and ellagic acid, which have demonstrated eNOS-upregulating and ADMA-lowering activity in multiple human trials. 200ml daily is the relevant dose in the research.

Dietary protocol: Arugula daily (80-100g), raw cacao 20-30g daily, pomegranate juice 200ml daily. These three together address ADMA, support eNOS expression, and provide the nitrate-nitrite-NO bypass during the eNOS recoupling period.

When Citrulline Becomes Appropriate

L-citrulline — the preferred alternative to arginine for NO-pathway support — is appropriate in Tier 2 only after Weeks 5-8, when the eNOS recoupling process is underway. The reasons for the delayed introduction are identical to the arginine problems above: citrulline converts to arginine endogenously, and that arginine then feeds the eNOS enzyme. If eNOS is still uncoupled when citrulline is introduced, the arginine it generates contributes to superoxide production.

L-citrulline has two significant advantages over L-arginine: it avoids first-pass hepatic metabolism (arginine is significantly metabolised in the gut and liver before reaching systemic circulation, which limits its effectiveness), and it avoids the supraphysiological arginine plasma spikes that drive viral reactivation risk.

Dose when introduced: 3g L-citrulline daily, with meals. If stacking with Pycnogenol (which it pairs extremely well with), the combination of citrulline 3g + Pycnogenol

120-160mg produces synergistic NO-pathway effects that neither compound achieves independently at these doses.

Chapter 3: The Six Tier 2 Compounds

Six compounds. Each one targeted at a specific step in the endothelial repair sequence. They are presented in the order in which they are introduced in the protocol — because the sequence matters as much as the compounds themselves.

Compound 1: Pycnogenol (French Maritime Pine Bark Extract) — 120mg daily

Pycnogenol is the proprietary standardised extract of French maritime pine bark (Pinus pinaster), standardised to a defined procyanidin profile. It is not interchangeable with generic "pine bark extract" — the standardisation of the procyanidin content and the specific geographic source are what determine the biological activity of the extract.

In Tier 2, Pycnogenol is the primary glycocalyx repair and ADMA-reduction agent. It works through three simultaneous mechanisms:

- Glycocalyx protection and restoration: Pycnogenol oligomeric proanthocyanidins inhibit the MMP enzymes (particularly MMP-2 and MMP-9) that degrade the heparan sulphate and chondroitin sulphate chains of the glycocalyx. By blocking the degradation pathway, Pycnogenol allows the glycocalyx to restore from its own biosynthetic capacity. It also directly stimulates glycosaminoglycan synthesis in endothelial cells, accelerating the repair.
- ADMA reduction: Pycnogenol has demonstrated direct ADMA-lowering activity in clinical trials, operating through mechanisms that include reduced symmetric vs asymmetric dimethylarginine ratio and support for DDAH (dimethylarginine dimethylaminohydrolase) enzyme activity — the enzyme that clears ADMA from the system.
- eNOS upregulation: Pycnogenol activates the NF-E2-related factor 2 (Nrf2) pathway, which upregulates eNOS gene expression and increases the cellular capacity to produce NO once the BH4 cofactor is available and eNOS is recoupled.

Clinical trial data: multiple human trials have demonstrated erectile function improvements with Pycnogenol at 120-160mg daily, particularly when combined with L-arginine (in healthy endothelium) or L-citrulline. The Pycnogenol + citrulline combination in this protocol uses these two compounds together from Week 5 onwards.

Timing: split dosing — 60mg morning, 60mg afternoon — is preferred over a single daily dose because the procyanidin compounds have a relatively short plasma half-life. Consistent dosing throughout the day maintains more stable plasma concentrations.

Duration for glycocalyx repair: 4-7 months. This is non-negotiable. The glycocalyx is a biological structure that requires time to regenerate. Pycnogenol accelerates the process; it does not shortcut the biology.

> **Sourcing note:** Pycnogenol is a registered trademark. Products labelled "Pycnogenol" are licensed to use the proprietary extract and must meet standardisation criteria. Generic "French maritime pine bark extract" products may or may not meet the same criteria. For this protocol, use the registered Pycnogenol extract specifically.
>
> **Mild 5-AR caveat:** Pycnogenol contains taxifolin, which has a mild 5-alpha-reductase inhibitory effect — significantly milder than saw palmetto but worth noting. At 120mg daily the effect is clinically minor. The endothelial and ADMA benefits far outweigh this concern at the protocol dose.

Compound 2: Vitamin C — 1-2g daily (split dose)

Vitamin C in Tier 2 is not a general antioxidant supplement. It is a BH4 protector. This is a specific and targeted mechanism that makes Vitamin C the most important BH4-preservation tool available without prescription.

BH4 (tetrahydrobiopterin) is the essential cofactor that keeps eNOS coupled. It is destroyed by oxidative stress — specifically by the peroxynitrite generated by uncoupled eNOS in a vicious cycle. Vitamin C regenerates BH4 from its oxidised form, BH2 (7,8-dihydrobiopterin), via a non-enzymatic ascorbate-dependent reduction. This reaction does not require any enzyme, does not depend on NADPH availability, and is not rate-limited by inflammatory state. It requires only adequate ascorbate (Vitamin C) in the correct cellular compartment.

Research has demonstrated that high-dose Vitamin C supplementation measurably increases the BH4:BH2 ratio in endothelial cells, increases eNOS coupling efficiency, and increases measurable NO bioavailability — without adding any arginine substrate. The mechanism is entirely at the cofactor level: more BH4 available means more coupled eNOS, means more NO from existing arginine, means improved endothelial function.

Dose: 1g twice daily (morning and evening) for the first 8 weeks. After Week 8, can reduce to 1g once daily if desired, while maintaining the dietary Vitamin C from arugula and other sources. Split dosing is preferred because Vitamin C is water-soluble with a plasma half-life of approximately 1.7 hours at doses of 1g.

Form: ascorbic acid is the most cost-effective and well-studied form. Buffered forms (sodium ascorbate, calcium ascorbate) are preferred for men with gastrointestinal sensitivity at higher doses. Liposomal Vitamin C provides superior cellular uptake but is not necessary at these doses for the BH4 protection mechanism.

Compound 3: Active B-Complex (Tier 2 Continuation)

The Active B-Complex from Tier 1 continues into Tier 2 without modification. Its role expands in Tier 2:

- Riboflavin (B2): as BH4 is being protected by Vitamin C, riboflavin (as riboflavin-5-phosphate) becomes the primary cofactor for DHPR (dihydropteridine reductase), the enzyme responsible for enzymatic BH4 recycling from BH2. Vitamin C handles the non-enzymatic recycling; DHPR + riboflavin handles the enzymatic recycling. Both pathways are required.
- Methylation continuation: Homocysteine lowering continues to be the most important upstream intervention for ADMA reduction and BH4 preservation. The B-Complex remains the tool for this.
- Thiamine (B1) for NADPH: NADPH is the electron donor for the eNOS electron transfer chain in its coupled state. Thiamine (as a cofactor for pyruvate dehydrogenase and alpha-ketoglutarate dehydrogenase) supports NADPH production via the Krebs cycle. Without adequate NADPH, even fully coupled eNOS cannot sustain NO production under demand.

If you are still using the same Active B-Complex from Tier 1, continue. If you have changed products, re-verify that all active forms are present.

Compound 4: CoQ10 as Ubiquinol — 200mg daily with food

CoQ10 enters Tier 2 as the endothelial-specific mitochondrial support compound. While Shilajit in Tier 1 optimised overall mitochondrial efficiency, CoQ10 as Ubiquinol in Tier 2 specifically targets the eNOS mitochondrial electron transfer mechanism and endothelial cell ATP production.

Endothelial cells are highly metabolically active and depend on adequate mitochondrial ATP for: maintaining tight junction integrity (which is ATP-dependent), synthesising glycocalyx components (a biosynthetically expensive process), and producing the NADPH required for eNOS coupling.

CoQ10 additionally serves as a membrane-bound antioxidant specifically within the inner mitochondrial membrane — the location where BH4 is most vulnerable to oxidative destruction. By maintaining mitochondrial membrane redox state, CoQ10 Ubiquinol provides an additional layer of BH4 protection beyond the cytosolic Vitamin C mechanism.

The Ubiquinol form (reduced CoQ10) is specified because it does not require the reduction step required by Ubiquinone (standard CoQ10). In men over 35, and in men with significant oxidative stress (which is the target population for Tier 2), the conversion of Ubiquinone to Ubiquinol is compromised. Using Ubiquinol directly bypasses this limitation.

Dose: 200mg daily with the largest meal of the day. CoQ10 is fat-soluble and absorption is dramatically improved when taken with dietary fat. Taking it with a fat-containing meal rather than separately is not optional — it is a bioavailability requirement.

> **Statin interaction:** Statins block the mevalonate pathway, which is also required for endogenous CoQ10 synthesis. Men taking statins are typically severely

CoQ10-depleted. If you are on a statin, CoQ10 supplementation at 200-400mg Ubiquinol daily is not optional — it is correction of a drug-induced deficiency.

Compound 5: GlyNAC (Glycine + NAC) — Glycine 3g / NAC 600mg daily

GlyNAC — the combination of glycine and N-acetylcysteine — is the Tier 2 glutathione restoration tool. It earns its place in this protocol through a specific mechanism: endothelial glutathione is the last line of defence for BH4 against peroxynitrite.

Glutathione (GSH) is the most abundant intracellular antioxidant in endothelial cells. It directly scavenges peroxynitrite before it can react with BH4. When endothelial glutathione is depleted — by chronic oxidative stress, inflammation, age, and the same LPS-driven inflammatory state that damaged the glycocalyx — BH4 becomes unprotected against peroxynitrite attack, and the eNOS uncoupling cycle cannot be broken regardless of what else is supplemented.

GlyNAC rather than direct glutathione supplementation is specified because:

- NAC provides cysteine, the rate-limiting amino acid precursor for glutathione synthesis — specifically the tripeptide gamma-glutamylcysteinylglycine. Direct liposomal glutathione supplementation is less efficient at raising intracellular (as opposed to plasma) glutathione than precursor-based approaches.
- Glycine completes the tripeptide. Glycine availability often limits glutathione synthesis in men with high cysteine turnover. The combination of both precursors, at the doses specified, has demonstrated superior glutathione-raising efficacy compared to either compound alone.
- Both compounds have independent vascular benefits: NAC reduces oxidised LDL, lowers ADMA, and reduces platelet aggregation. Glycine directly supports glycocalyx biosynthesis (glycine is a required substrate for the synthesis of heparan sulphate proteoglycans — the primary structural components of the glycocalyx).

Important caution: some men experience worsening libido or anhedonia on NAC, particularly those with a history of SSRI use, ashwagandha-induced dopamine blunting, or who are already in dopamine recovery. The mechanism is that NAC reduces extracellular glutamate via the cystine-glutamate antiporter, which can reduce dopaminergic tone via the nucleus accumbens glutamate-dopamine interaction. If you experience mood flattening or libido reduction on NAC, reduce the dose to 300mg or discontinue and substitute with dietary glycine alone (found at highest concentration in gelatin, bone broth, and skin-on poultry).

Dosing note: NAC: 600mg daily, with food (reduces gastrointestinal side effects). Glycine: 3g daily, can be taken separately as a powder mixed in water — flavourless and readily soluble. Both morning or split morning/evening.

Compound 6: L-Citrulline — 3g daily (from Week 5 onwards)

L-citrulline is the final compound introduced in Tier 2 and is added only after the first four weeks of glycocalyx repair and BH4 protection are underway. It is an amino acid found at highest concentration in watermelon rind, and it is the preferred arginine-delivery mechanism for eNOS support for the reasons described in Chapter 2.

At Week 5, with Pycnogenol, Vitamin C, riboflavin, and CoQ10 all in play:

- ADMA is beginning to fall (Pycnogenol + Homocysteine correction).
- BH4 is being protected (Vitamin C + riboflavin + CoQ10).
- Glutathione is being restored (GlyNAC).
- The eNOS enzyme is moving from uncoupled toward coupled state.

At this point, introducing arginine substrate (via citrulline) begins to produce more NO rather than more superoxide. The window has opened.

The Pycnogenol + citrulline combination is specifically synergistic. Pycnogenol upregulates eNOS gene expression while citrulline increases the arginine substrate available to the now-recoupling enzyme. The combination in human trials produces significantly greater erectile function improvements than either compound at equivalent doses alone.

Dose: 3g L-citrulline malate (the malate form provides superior dissolution and slightly better absorption) or pure L-citrulline, taken 30-60 minutes before expected activity or with morning supplements. Can be taken as powder mixed in water.

Chapter 4: The 5-Step Sequenced Protocol

The sequence is the protocol. The compounds are the tools. Using the tools in the wrong order — or all simultaneously from day one — produces suboptimal results at best and accelerates damage at worst.

This chapter gives you the exact sequence and the biological rationale for each step.

The Master Rule: Sequence Before Stack

The five steps below are not interchangeable. Each step creates the biological conditions that allow the next step to work. Skipping Step 1 to get to Step 3 is not aggressive optimisation. It is misunderstanding the mechanism.

To use an engineering analogy: you do not install new pipes while the building is still on fire. You extinguish the fire (Tier 1), you clear the debris (Step 1), you inspect and repair the infrastructure (Steps 2-3), you test under low pressure (Step 4), and then you restore full operational flow (Step 5).

Step 1 — Reduce the Oxidative Burden (Weeks 1-4)

Before any repair compounds are introduced, the sources of ongoing oxidative damage must be addressed. Applying glycocalyx-repair compounds to an endothelium that is still being continuously damaged by the same inputs that caused the damage is like painting over active rust.

- Dietary: eliminate alcohol (direct eNOS inhibitor and BH4 destroyer), seed oils (PUFAs oxidise to 4-HNE and acrolein, potent vascular toxins), refined sugar (the fastest driver of glycocalyx glycation and shedding), and ultra-processed food generally.
- Begin: arugula daily, raw cacao daily, pomegranate juice daily — the dietary nitrate and ADMA-lowering baseline.
- Begin: Vitamin C 1g twice daily — BH4 protection from Day 1.
- Begin: GlyNAC (glycine 3g + NAC 600mg) — glutathione restoration and glycocalyx substrate.
- Continue: all Tier 1 compounds without modification.

The goal of Weeks 1-4 is to stop the active destruction before attempting the repair. Homocysteine should be falling from Tier 1. Prolactin should be declining. Gut LPS load should be reduced from Tier 1 dietary interventions. With these inputs reduced, the endothelium has its first opportunity to begin net repair.

Step 2 — Glycocalyx Repair (Weeks 1-4 ongoing)

Pycnogenol is introduced from Day 1 of Tier 2 and continues for the full 4-7 months of the protocol. The glycocalyx repair work runs parallel to and in support of every subsequent step.

- Add: Pycnogenol 60mg morning + 60mg afternoon from Day 1.

- Add: CoQ10 Ubiquinol 200mg with largest meal from Day 1.

Pycnogenol simultaneously inhibits glycocalyx degradation enzymes (MMP-2, MMP-9), stimulates glycosaminoglycan synthesis, reduces ADMA, and upregulates Nrf2 — all of which contribute to the endothelial repair environment.

The 4-7 month timeline is not an estimate. It is the biological rate at which endothelial cells regenerate and glycocalyx components are biosynthesised. Individual cells turn over at varying rates depending on vascular location and shear stress history. The turnover of the glycocalyx in penile vascular beds is believed to require a minimum of 4 months of sustained repair conditions.

This is where patience becomes a protocol requirement. At 4 weeks, you will likely not see the full effect. At 8-12 weeks, you should begin to notice improvements in morning erections, vascular sensation, and responsiveness. At 4-6 months, the endothelial function is approaching a genuinely rebuilt state.

Step 3 — BH4 Coupling Restoration (Weeks 1-8 intensive)

The BH4 restoration work runs in parallel with glycocalyx repair. Vitamin C is the primary tool; the Active B-Complex provides the riboflavin for enzymatic BH4 recycling.

- Vitamin C 1g morning + 1g evening: maintains ascorbate concentrations sufficient for non-enzymatic BH4 regeneration throughout the day.
- Riboflavin (B2) in active form: powers DHPR-mediated enzymatic BH4 recycling from the B-Complex.
- GlyNAC: glutathione protects BH4 from peroxynitrite attack, providing the third layer of BH4 defence.

The three layers of BH4 protection in Tier 2:

- Layer 1 — prevention: reducing the oxidative inputs that generate peroxynitrite (dietary reform, Homocysteine lowering from Tier 1).
- Layer 2 — scavenging: glutathione (via GlyNAC) and Vitamin C scavenge peroxynitrite before it reaches BH4.
- Layer 3 — recycling: Vitamin C (non-enzymatic) and riboflavin/DHPR (enzymatic) regenerate BH4 from its oxidised form BH2.

When all three layers are operating simultaneously, the BH4 pool begins to recover. As BH4 rises, eNOS begins to shift from uncoupled (superoxide-producing) toward coupled (NO-producing) state. This is not binary — it is a gradual shift that mirrors the BH4:BH2 ratio improvement.

Step 4 — ADMA Clearance and NO Pathway Preparation (Weeks 2-8)

ADMA — the eNOS inhibitor — must be reduced before the NO pathway can be opened. Three interventions converge on this:

- Homocysteine correction from Tier 1: Homocysteine drives ADMA production by inhibiting DDAH (the ADMA-clearing enzyme) via hydrogen sulphide competition. As Homocysteine falls, DDAH activity recovers and ADMA clearance improves.
- Pycnogenol: directly lowers ADMA via DDAH support and reduced symmetric/asymmetric dimethylarginine ratio.
- Arugula dietary nitrates + raw cacao: provide an ADMA-independent NO source via the nitrate-nitrite-NO pathway, maintaining some NO bioavailability during the period when eNOS is still uncoupled and ADMA is still elevated.

By Week 5-6, with Homocysteine falling, Pycnogenol active, and the dietary interventions in place, ADMA levels should be trending down. This is when the eNOS enzyme has a meaningful chance of producing more NO than superoxide when arginine substrate is provided.

Step 5 — NO Amplification (From Week 5)

The final step introduces the citrulline substrate to the now partially-recoupled eNOS system:

- Add: L-citrulline 3g daily from Week 5.
- The Pycnogenol + citrulline combination is now fully active.
- Optional at Week 5: if using the vacuum erection device protocol (Chapter 6), this is when it becomes most effective — the mechanical stimulus to the penile vasculature now has a biochemically-prepared endothelium to respond to it.

The NO amplification phase is where most men first notice the significant functional improvements that the preceding weeks of foundational work made possible. The glycocalyx sensor is beginning to function again. The eNOS enzyme is beginning to couple. BH4 is protected. ADMA is falling. The arginine substrate is now available. The system is producing NO.

Chapter 5: The Dietary Protocol

The dietary interventions in Tier 2 are not optional additions. They are integral components of the protocol that no supplement can replicate. The compounds in Chapter 3 work within a biological environment that diet determines. A poor dietary environment actively counteracts the supplements.

The Three Daily Non-Negotiables

Three foods must be present daily throughout the Tier 2 protocol:

- Arugula (rocket): 80-100g daily. The highest dietary nitrate source available. Used as a salad base, in smoothies, or consumed directly. Does not require cooking — heat destroys the nitrate-to-nitrite conversion capacity in the oral bacteria that initiate the nitrate-nitrite-NO pathway. Do not use mouthwash during this protocol — antiseptic mouthwash kills the commensal bacteria responsible for nitrate reduction.
- Raw cacao or 90%+ dark chocolate: 20-30g daily. Provides epicatechin (a flavonol that directly activates eNOS via the PI3K/Akt pathway), theobromine (a PDE inhibitor with vasodilatory activity), and a significant ADMA-lowering effect. Raw cacao powder mixed into coffee or yogurt is the easiest delivery method. The 90%+ dark chocolate threshold is specified because below this, the sugar content begins to undo the glycocalyx-protective benefits via post-meal glucose-driven glycocalyx shedding.
- Pomegranate juice (100%, unsweetened): 200ml daily. The ellagitannins in pomegranate (punicalagins) convert to urolithins in the gut, which have demonstrated mitochondrial biogenesis and eNOS-upregulating activity. Human trials have shown measurable ADMA reduction and NO bioavailability improvement with daily 200ml consumption. Check the label: it must be 100% pomegranate juice with no added sugar or juice dilution.

The DHT-Preserving Carbohydrate Strategy

As covered in The Confidence Reset Book II, the T3/DHT conversion pathway requires adequate carbohydrate intake for optimal hepatic T4-to-T3 conversion. This continues to apply in Tier 2. Do not pursue a very low-carbohydrate or ketogenic diet during this protocol.

Target carbohydrate intake: 150-250g daily from clean sources. The specific sources that best support the Tier 2 protocol:

- White rice: high-glycaemic but low in gut-irritating compounds. Rapid glucose delivery for hepatic T4-T3 conversion. Pairs well with arugula salads.
- Potatoes (cooked and cooled): cooking and cooling generates resistant starch, which is a prebiotic fibre that feeds butyrate-producing gut bacteria. Butyrate maintains gut barrier integrity and reduces LPS translocation. Cold potato salad

(with olive oil and vinegar) is the highest-value carbohydrate source for the combined Tier 2 goals.

- Fruit: particularly berries (high in anthocyanins that support eNOS and reduce oxidative stress) and watermelon (contains L-citrulline natively, contributing to NO pathway support).
- Avoid: refined sugar in processed food (directly glycates and sheds the glycocalyx), high-fructose corn syrup, and seed oil-containing processed foods.

The Egg Protocol — Non-Negotiable

Two to three whole eggs daily continue from the Tier 1 protocol. Their role in Tier 2 expands:

- Choline: the primary choline source in the diet. Choline is the precursor to phosphatidylcholine — the dominant phospholipid in endothelial cell membranes. Endothelial membrane integrity requires ongoing phosphatidylcholine synthesis. Choline deficiency directly impairs this.
- Cholesterol: testosterone and all steroid hormones are synthesised from cholesterol. The dietary cholesterol phobia of the 1980s is clinically irrelevant for healthy men — dietary cholesterol does not meaningfully raise cardiovascular risk in the context of a low-inflammatory diet and is essential for steroid hormone production.
- Sulphur amino acids: eggs are the highest sulphur-containing food in the typical diet. Sulphur amino acids (methionine, cysteine) are required for glutathione synthesis. In combination with the GlyNAC supplementation in Tier 2, dietary sulphur from eggs significantly boosts the glutathione-restoration protocol.

Foods to Eliminate During Tier 2

- Alcohol: the only acceptable quantity during the active Tier 2 protocol is zero. Alcohol generates acetaldehyde in the liver, which is a direct BH4 destroyer via carbonyl-induced modification of dihydropteridine reductase. A single heavy drinking night can set back BH4 recovery by days. Weekly drinking maintains the BH4-depleted state that Tier 2 is attempting to reverse.
- Seed oils (canola, soybean, corn, sunflower, safflower): high in linoleic acid (LA), which oxidises to 4-hydroxynonenal (4-HNE) and other lipid peroxidation products that are potent eNOS inhibitors and glycocalyx damaging agents. Replace with olive oil, butter, ghee, and coconut oil for cooking.
- Refined sugar and high-fructose foods: as noted, post-meal glucose spikes above 140 mg/dL cause immediate glycocalyx shedding. The dietary goal is to maintain stable blood glucose throughout the day. If you currently eat a high-sugar diet, reducing it is the single most impactful dietary change for glycocalyx protection.

- Soy products (tofu, soya milk, tempeh, soy protein isolate): contain phytoestrogens (genistein, daidzein) that bind oestrogen receptors and modulate sex hormone signalling. In the context of an endothelial restoration protocol that depends on DHT and testosterone, additional estrogenic input is counterproductive.

The Raw Carrot Salad (Tier 2 Version)

Continuing from Tier 1, the raw carrot salad is upgraded in Tier 2:

1-2 medium raw carrots, grated. Add 1 tablespoon apple cider vinegar (ADMA-lowering acetic acid, plus microbiome support). Add 1 tablespoon olive oil (oleocanthal, anti-inflammatory). Add 1 small clove of raw garlic, minced (allicin, the most potent natural DDAH activator — allicin directly increases ADMA clearance enzyme activity and reduces cardiovascular risk via this mechanism). Optional: fresh parsley (highest dietary nitrate source after arugula, and contains apigenin — but note apigenin in very high concentrations has mild AR-antagonist activity; parsley at culinary doses is safe and beneficial).

Eat it daily. It is not glamorous. It is functional.

Chapter 6: Devices — The Mechanical Protocol

Tier 2 is the first Tier in which mechanical devices become relevant. This is not about performance aids. It is about tissue maintenance during the repair period.

The Vacuum Erection Device — Daily Oxygenation

The vacuum erection device (VED) — a cylinder placed over the penis with a pump to create negative pressure — has a clinical application beyond its use as a performance aid. At low pressure settings, daily VED use produces mechanical penile tumescence that oxygenates the smooth muscle of the corpus cavernosum via the same mechanism as nocturnal penile tumescence during REM sleep.

The penile smooth muscle is androgen-sensitive tissue that requires regular oxygenation to maintain its structure, elasticity, and vascular responsiveness. In men with significant endothelial dysfunction, the natural oxygenation from NPT may be insufficient. Daily low-pressure VED use supplements this oxygenation, prevents the progressive collagen deposition and fibrosis that occurs in chronically hypoxic penile tissue, and maintains the mechanical stretch that stimulates endothelial shear stress — which, as the Tier 2 compounds restore glycocalyx and eNOS function, begins to generate increasing NO production.

Protocol specifics:

- Pressure: 4-6 inches of mercury (low negative pressure). Do not exceed this. High negative pressure VED use — above 8-10 inches of mercury — causes microtrauma to the penile vasculature, petechial haemorrhages, and can cause fibrosis. The device must have a pressure gauge or safety valve.
- Duration: 10 minutes daily. This is not a performance use — do not maintain high pressure for longer periods. The goal is repeated low-pressure tumescence cycles, not sustained erection.
- Timing: from Week 5 of Tier 2, when the endothelial repair work is underway. Beginning before this — in a heavily inflamed, glycocalyx-stripped system — provides less benefit and more mechanical stress.
- Medical-grade devices only: genuine VED devices marketed for erectile dysfunction rehabilitation have safety valves and standardised pressure gauges. Novelty or non-medical devices should not be used for this protocol.

Acoustic Wave Therapy — For Structural Vascular Remodelling

Acoustic wave therapy (AWT) — also marketed as low-intensity shockwave therapy (Li-ESWT) — applies low-intensity acoustic pressure waves to penile tissue. It is the only non-invasive treatment that has demonstrated the ability to stimulate neovascularisation (the growth of new blood vessels) and structural vascular remodelling in the penile vasculature.

The mechanism: acoustic waves at the intensity and frequency used in Li-ESWT create microtrauma in the penile tissue that triggers a healing response. This healing response includes release of vascular endothelial growth factor (VEGF), recruitment of endothelial progenitor cells, and the formation of new vascular branches. In men with structural vascular disease (arteriogenic ED), this creates new vascular pathways where existing pathways are narrowed or occluded.

Li-ESWT is a medical procedure administered by a clinician. It requires proper equipment (the energy levels are calibrated to specific protocols) and a qualified practitioner. It should not be confused with commercial devices sold for home use, which do not operate at the therapeutic parameters used in clinical protocols.

Who benefits: men who have completed Weeks 5-8 of the biochemical Tier 2 protocol and are experiencing persistent difficulty despite measurable improvements in other markers. Men with documented arteriogenic ED on penile Doppler ultrasound. Men who have used finasteride or dutasteride and have significant penile smooth muscle fibrosis.

Typical protocol: 6-12 sessions over 6-12 weeks, 2000-3000 pulses per session at 0.09-0.25 mJ/mm² energy flux density. Response rates in the research literature range from 70-85% in appropriately selected patients with vascular ED.

Pelvic Floor Relaxation and Strengthening

Pelvic floor dysfunction is an underappreciated contributor to vascular ED in modern men. The ischiocavernosus and bulbocavernosus muscles of the pelvic floor compress the base of the penis during erection, increasing intracavernosal pressure. If these muscles are weak, pressure is lost. If they are chronically hypertonic (in spasm from chronic sitting, chronic stress, or chronic sympathetic activation), they can restrict venous outflow during tumescence and impair the mechanical filling of the erectile chambers.

Two components of pelvic floor exercise are relevant:

- Kegel contractions (strengthening): slow, sustained contractions of the pelvic floor held for 5-10 seconds, repeated 10-15 times, 2-3 times daily. These target the ischiocavernosus and bulbocavernosus. There is randomised controlled trial evidence that pelvic floor rehabilitation produces significant erectile function improvements in men with mild-moderate vascular ED.
- Pelvic floor release (relaxation): diaphragmatic breathing in the supine position with deliberate relaxation of the pelvic floor on each exhale. Paradoxically, hypertonic pelvic floor is more common than weak pelvic floor in men with performance anxiety and chronic sympathetic activation. A tight, spasming pelvic floor can produce similar symptoms to a weak one. The release component is as important as the strengthening.
- Reverse Kegels: bearing down and opening the pelvic floor as if initiating urination or defecation (without actually doing so), held for 3-5 seconds. These

stretch and relax hypertonic pelvic floor muscles and are particularly useful for men who have been doing Kegels exclusively and may have created additional hypertonicity.

Grip-strength and calf-raise exercises are also relevant: research has identified grip strength as a proxy marker for overall androgen receptor sensitivity and vascular health in men. Calf raises improve the calf muscle pump, which supports systemic venous return and reduces the peripheral venous pooling that can reduce pelvic blood pressure. Neither is a primary erectile intervention but both contribute to the vascular environment.

Chapter 7: Lab Markers — How You Know It Is Working

The Tier 2 protocol is 4-7 months of consistent work. Lab markers are the objective measure of progress that self-reporting cannot provide with reliability.

The Primary Tier 2 Markers

Test at baseline (before starting Tier 2) and at 12 weeks and 6 months:

- Homocysteine: should be below 8 µmol/L from Tier 1. If not, Tier 1 is not complete. In Tier 2, continued Homocysteine reduction reflects ongoing methylation improvement and ADMA reduction.
- hs-CRP (high-sensitivity C-reactive protein): the primary systemic inflammation marker. Should be trending toward < 1.0 mg/L. If hs-CRP is still above 2.0 mg/L at 12 weeks of Tier 2, the inflammatory source has not been adequately addressed — typically gut LPS or a dietary input that has not been removed.
- ADMA (Asymmetric Dimethylarginine): not universally available but measurable through specialist cardiovascular or functional medicine labs. Target < 0.6 µmol/L. This is the most direct marker of the primary Tier 2 intervention. Its decline confirms that eNOS uncoupling is resolving.
- Fasting glucose and post-meal glucose (2-hour post-meal test): target fasting < 90 mg/dL, 2-hour post-meal < 120 mg/dL. Values above these thresholds drive glycocalyx shedding continuously. If post-meal glucose is above 140 mg/dL, the dietary reform is insufficient.
- Fasting insulin: target < 10 µIU/mL. Insulin resistance drives SHBG elevation, impairs eNOS, and worsens LPS-driven inflammation. This should improve as diet is reformed.

Secondary Markers

- DHT: should be rising as Prolactin falls and 5-AR activity is restored. Target DHT at ≥ 10% of total testosterone. If DHT is not rising despite Prolactin normalisation, check Free T3 (thyroid is the primary driver of 5-AR activity after prolactin is addressed).
- Free T3: upper third of the reference range. If below this, the T3-DHT conversion pathway is impaired regardless of what Tier 2 compounds are doing to the endothelium.
- Prolactin: should be trending toward 4-7 ng/mL from Tier 1. If still above 10 at the start of Tier 2, Tier 1 is incomplete.
- CoQ10 plasma level: available through specialist labs. If CoQ10 is severely depleted despite 200mg Ubiquinol supplementation, consider statins or other CoQ10-depleting medications as the cause.

- Ferritin: 50-150 ng/mL. Ferritin is required for the haem cofactor in eNOS. Low ferritin directly impairs eNOS function. High ferritin (> 200 ng/mL) indicates iron overload and contributes to oxidative stress. Men with high ferritin should consider periodic blood donation.

The Self-Monitoring Signals

Between lab tests, three self-monitored signals track Tier 2 progress:

- Morning erections: quality, rigidity, and frequency. These are the most reliable non-invasive indicator of improving penile vascular function. The return or strengthening of morning erections reflects improved NPT — which requires functional eNOS, adequate NO production, and parasympathetic nervous system health. Track weekly.
- Peripheral vascular response: in cold environments, men with significant endothelial dysfunction experience more pronounced vasoconstriction in the extremities. Improving vascular responsiveness in the fingers and toes in cold conditions is an indirect marker of improving systemic endothelial function.
- Blood pressure: as eNOS coupling improves and NO bioavailability increases, systemic blood pressure typically decreases. A significant reduction in resting blood pressure over the Tier 2 protocol period is a direct marker of improved endothelial function. Men on blood pressure medication should monitor closely and discuss with their physician.

Chapter 8: The Complete Tier 2 Reference

The Stack

Pycnogenol — 60mg x 2 (120mg total) | Morning + afternoon | *Glycocalyx repair. ADMA reduction. eNOS upregulation. Use registered Pycnogenol brand.*

Vitamin C — 1g x 2 (2g total) | Morning + evening | *BH4 protection via ascorbate-dependent regeneration. Non-negotiable for eNOS recoupling.*

Active B-Complex — 1 cap | Morning with food | *Riboflavin for enzymatic BH4 recycling. Methylation continuation. Thiamine for NADPH.*

CoQ10 Ubiquinol — 200mg | With largest meal | *Mitochondrial membrane antioxidant. Additional BH4 protection. Must take with dietary fat.*

GlyNAC — Glycine 3g + NAC 600mg | Morning (with food for NAC) | *Glutathione restoration. BH4 peroxynitrite protection. Glycocalyx substrate (glycine).*

L-Citrulline — 3g | From Week 5 onwards, morning | *Arginine substrate for recoupling eNOS. Synergistic with Pycnogenol. Not before Week 5.*

All Tier 1 compounds — As per Tier 1 | As per Tier 1 | *Continue unchanged throughout Tier 2. Tier 1 is the foundation. Remove it and Tier 2 stops working.*

The Sequence

Weeks 1-4: Steps 1-3 (Oxidative reduction, glycocalyx repair, BH4 restoration)

- ☐ Start: Pycnogenol 60mg x 2, Vitamin C 1g x 2, GlyNAC, CoQ10 Ubiquinol 200mg.
- ☐ Dietary: arugula daily, raw cacao daily, pomegranate juice 200ml daily.
- ☐ Remove: alcohol completely, seed oils, refined sugar, soy.
- ☐ Continue: all Tier 1 compounds.

Weeks 5-8: Step 4-5 (ADMA clearance + NO amplification)

- ☐ Add: L-citrulline 3g daily.
- ☐ Optional: begin low-pressure VED protocol (4-6 inches Hg, 10 min daily).
- ☐ Check 8-week signals: morning erections improving? Blood pressure trending down? Sleep quality maintained from Tier 1?

Weeks 9-24 (months 3-6): Consolidation and glycocalyx maturation

- ☐ Maintain full stack.
- ☐ Lab retest at Week 12: Homocysteine, hs-CRP, prolactin, DHT, Free T3, fasting glucose.
- ☐ If plateau at Week 12: review dietary compliance (alcohol, sugar, seed oils), gut LPS status, and Free T3. These are the three most common reasons for plateau.

- Consider acoustic wave therapy referral if structural vascular remodelling appears necessary from exam and Doppler.
- Lab retest at 6 months: full panel including ADMA if accessible.

The Daily Dietary Non-Negotiables

- Arugula 80-100g: dietary nitrate for NO bypass pathway.
- Raw cacao 20-30g or 90%+ dark chocolate: epicatechin, ADMA reduction.
- Pomegranate juice 200ml (100%, unsweetened): eNOS upregulation, ADMA reduction.
- 2-3 eggs: choline for endothelial phosphatidylcholine, sulphur for glutathione.
- Raw carrot salad with garlic: DDAH activation (allicin), gut barrier support.
- 150-250g clean carbohydrates: T3 support, DHT conversion maintenance.
- NO mouthwash: antiseptic mouthwash kills the oral bacteria required for nitrate-nitrite-NO conversion.

The Cycling Rule

Tier 2 does not cycle the same way as Tier 1. The glycocalyx repair is a structural restoration that requires uninterrupted support for 4-7 months. Do not take a protocol break during the first 6 months of Tier 2.

After 6 months of full protocol, a transition maintenance dose is appropriate:

- Pycnogenol: reduce to 60mg once daily (maintenance dose for ongoing glycocalyx protection).
- Vitamin C: reduce to 1g once daily.
- CoQ10 Ubiquinol: continue at 200mg.
- L-citrulline: continue at 3g daily or reduce to 1.5g if desired.
- GlyNAC: can reduce to 3 days per week at maintenance.
- All Tier 1 compounds: continue indefinitely as the foundational layer.

What Tier 3 Addresses

Tier 3 of the Stub Club Protocol addresses the hormonal amplification layer: DHT support, androgen receptor sensitivity, dopaminergic herb cycling, and the advanced compounds that amplify the hormonal signal through an endothelium that Tier 1 and Tier 2 have rebuilt.

Tier 3 compounds are signal amplifiers. They amplify whatever the hormonal signal is in a man whose vascular system is now capable of responding to it. Applied to the rebuilt foundation of Tiers 1 and 2, they produce their intended effects. Applied without that foundation, they have produced some of the worst outcomes documented in the men's health coaching literature — receptor downregulation,

prolactin rebounds, hormonal instability, and dependence on compounds that were intended to be cycled catalysts.

Complete Tier 2. All of it. The 4-7 months. Then build Tier 3 on top of a foundation that actually holds.

Appendix: Quick Reference — The Tier 2 Compounds

Mechanism summary and non-negotiable sourcing notes for each Tier 2 compound.

Pycnogenol (registered extract only)

Glycocalyx repair via MMP inhibition and GAG synthesis stimulation. ADMA reduction via DDAH support. eNOS upregulation via Nrf2 pathway. Requires 4-7 months. Use registered Pycnogenol brand only — generic pine bark products are not equivalent.

Vitamin C (ascorbic acid)

BH4 regeneration via non-enzymatic ascorbate-dependent reduction of BH2 to BH4. The fastest available mechanism for reversing eNOS uncoupling. Water-soluble — split dosing twice daily for maintained plasma levels.

Active B-Complex (from Tier 1)

Riboflavin as DHPR cofactor for enzymatic BH4 recycling. Continued Homocysteine reduction for ADMA suppression. Thiamine for NADPH production supporting eNOS electron transfer.

CoQ10 Ubiquinol (not Ubiquinone)

Endothelial mitochondrial membrane antioxidant. Additional BH4 protection at the mitochondrial membrane level. Required by men on statins without exception. Fat-soluble — take with dietary fat.

GlyNAC (glycine 3g + NAC 600mg)

Glutathione restoration — the endothelial cell's primary defence against peroxynitrite. Glycine as direct glycocalyx biosynthesis substrate. NAC reduces oxidised LDL and ADMA. Monitor for mood/libido effects from NAC (dopamine interaction).

L-citrulline (from Week 5 only)

Arginine substrate delivery for recoupling eNOS, bypassing first-pass hepatic arginine metabolism and avoiding viral reactivation risk. Synergistic with Pycnogenol. Do not introduce before Week 5.