THE STUB CLUB PROTOCOL

TIER 1

THE BIOLOGICAL KILL-SWITCH

The Hardware Reboot

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Introduction: You Cannot Build on a Swamp

You are reading Tier 1 of the Stub Club Protocol. This is where every protocol in this series begins — not because it is the most exciting tier, not because it produces the most dramatic results in the shortest time, but because without it, nothing else works.

You can't build a skyscraper on a swamp. If your Homocysteine is sitting at 12 and your Prolactin is at 15, your body is biochemically operating like a 70-year-old man with a chronic illness — regardless of your age, your training, your diet, or your intentions. You are working against yourself at the cellular level.

Tier 1 is the Hardware Reboot. It is the work of stopping the internal damage before attempting to build anything on top of it. It is five compounds, a 90-day commitment, and a clear-eyed understanding of what each one is actually doing and why.

> *"If you don't wake up with a response in 30 days, your gut is likely too inflamed to absorb these. Fix the gut or you're throwing money down the drain."*

The NYC Reality Check above is not a disclaimer. It is a diagnostic. If Tier 1 is not producing results within 30 days, the gut is the next variable — and this book covers that in detail in the later chapters.

Read this book completely before you start. Understanding the mechanism behind each compound is what separates a man who follows a protocol from a man who owns it. And when something is not working, the man who owns it knows where to look. The man who is just following instructions does not.

Chapter 1: The Internal Rust — What You Are Actually Fighting

Before we discuss what Tier 1 does, you need to understand what Tier 1 is fighting. The enemy has two primary faces: Homocysteine and Prolactin. One is rusting your pipes from the inside. The other is biochemically switching off your engine.

Homocysteine: The Slow Poison

Homocysteine is an amino acid produced as a byproduct of methionine metabolism. In small quantities, it is normal. In quantity — and "quantity" begins much lower than your doctor has told you — it is a systematic destroyer of the vascular machinery that makes male sexual function possible.

Your doctor will tell you that a Homocysteine level up to 15 µmol/L is "normal." This is technically accurate in the sense that it describes what is common in the population being measured. It is not accurate in any sense that matters for performance, longevity, or sexual health. Above 8 µmol/L, Homocysteine is actively injuring your endothelial lining — the thin layer of cells coating the inside of every blood vessel in your body — every hour of every day.

The optimal range is 5-8 µmol/L. Anything above 8 is a problem. At 12, you are in the danger zone. At 15+, you are in a vascular crisis that your doctor considers unremarkable.

Four Ways Homocysteine Destroys You

- Direct endothelial injury: Homocysteine is a thiol compound that oxidises to form hydrogen peroxide and other reactive oxygen species directly at the endothelial membrane. It physically injures the cells lining your blood vessels — including the penile arteries, which are among the smallest in the body and therefore the first to show functional impairment.
- The ADMA trap: Elevated Homocysteine increases levels of ADMA (Asymmetric Dimethylarginine) — the body's own built-in inhibitor of nitric oxide synthase. ADMA competes with arginine at the eNOS enzyme and shuts down nitric oxide production at the source. This is the mechanism by which high Homocysteine directly suppresses the biochemical signal required for erection. And it is why taking more arginine when Homocysteine is elevated does not work — ADMA wins the competition every time.
- Methylation failure: Elevated Homocysteine is the primary clinical marker of impaired methylation. When the methylation cycle is failing — when SAM:SAH ratio is compromised — your body cannot repair DNA efficiently, cannot produce neurotransmitters at the rate it needs, cannot detoxify estrogens and xenoestrogens, and cannot synthesise the phosphatidylcholine required to maintain cell membrane integrity throughout the vascular system. Every system in the body that depends on methylation degrades.

- BH4 destruction: High Homocysteine generates peroxynitrite — a particularly destructive reactive nitrogen species — which destroys BH4 (tetrahydrobiopterin), the essential cofactor that keeps eNOS functioning correctly. Without BH4, eNOS "uncouples." An uncoupled eNOS enzyme stops producing nitric oxide and starts producing superoxide — a free radical that causes further endothelial damage and inflammation. This creates a self-reinforcing cycle: high Homocysteine destroys BH4, BH4 depletion uncouples eNOS, uncoupled eNOS produces superoxide, superoxide generates peroxynitrite, peroxynitrite destroys more BH4.

> *"You are not dealing with a simple "deficiency." You are dealing with a cycle of biochemical damage. The only way to stop the cycle is to address the upstream cause — the methylation failure that allows Homocysteine to accumulate in the first place."*

The Downstream Cascade

Elevated Homocysteine does not only damage erections. It damages everything. The downstream consequences documented in the research literature include:

- Estrogen dominance — impaired methylation means impaired estradiol clearance through the liver's Phase 2 detoxification pathways, particularly glucuronidation and sulfation. Estrogen that should be packaged and excreted is instead reabsorbed.
- Cognitive decline — Homocysteine is one of the most robust independent predictors of dementia and cognitive decline in the longitudinal research. The mechanism is the same: vascular damage and methylation failure in the brain.
- Depression and mood disruption — methylation failure impairs the production of serotonin, dopamine, and norepinephrine, all of which require SAMe as a methyl donor in their synthesis pathways.
- Bone loss — elevated Homocysteine interferes with collagen cross-linking and bone matrix formation.
- Elevated clotting risk — Homocysteine promotes a prothrombotic state by increasing platelet aggregation and reducing the activity of natural anticoagulants.
- Rosacea and skin changes — described in clinical coaching as a late-stage visible marker of chronic methylation impairment and oxidative vascular stress.

The single intervention that addresses all of these simultaneously is fixing the methylation cycle. That is what the B-Complex in Tier 1 does. Everything else in the stack supports the environment in which that repair becomes possible.

The Folate Trap — Why Standard Blood Tests Miss It

Here is the trap that catches most men who try to address their Homocysteine with standard folate supplementation: your standard blood test may show normal serum

folate and normal serum B12, and yet your methylation cycle is functionally impaired and your Homocysteine is elevated.

This happens when the folate cycle is blocked at the MTHFR (methylenetetrahydrofolate reductase) step. MTHFR variants — particularly the C677T polymorphism, present in roughly 10-15% of the population in homozygous form and 40%+ in heterozygous form — impair the conversion of dietary folate to its biologically active form, 5-MTHF. The serum folate test measures total folate, including the inactive unconverted forms. The patient appears folate-replete on paper while being functionally folate-deficient at the cellular level.

Supplementing with standard folic acid in this situation makes things measurably worse. Folic acid is the synthetic, inactive, unmetabolised form. It competes with 5-MTHF at folate receptors. You flood the system with a molecule your body cannot use, that blocks the receptors for the molecule your body needs.

This is why the Active B-Complex in Tier 1 must contain 5-MTHF (methylfolate), not folic acid. This is not a minor distinction. It is the difference between the supplement working and the supplement actively preventing recovery.

> **What to look for on the label:** 5-MTHF or methylfolate (not folic acid), methylcobalamin or adenosylcobalamin (not cyanocobalamin), P5P (not pyridoxine HCl), riboflavin-5-phosphate (not riboflavin)

Chapter 2: The Prolactin Brake — Your Biochemical Off Switch

While Homocysteine is destroying the hardware, Prolactin is applying the brakes. Elevated Prolactin is the single most underdiagnosed hormonal cause of male sexual dysfunction, libido suppression, and dopaminergic blunting. And the standard "normal" range for Prolactin is, like the standard range for Homocysteine, dramatically too permissive.

What Prolactin Does in Men

Prolactin is produced by the anterior pituitary gland. In women, it drives milk production postpartum. In men, it is the hormone released after orgasm to signal the end of the sexual response — it is the body's biological "off switch."

When Prolactin is chronically elevated in men, the off switch is permanently stuck in the "on" position. The consequences are comprehensive:

- Direct D2 receptor antagonism: Prolactin is a dopamine antagonist. It directly blocks D2 receptors — the primary receptor through which dopamine drives motivation, desire, and the initiation of sexual response. A man with chronically elevated Prolactin is biochemically in a state analogous to someone taking an antidopaminergic drug. The system that starts the entire sexual response cascade is pharmacologically suppressed.
- 5-alpha-reductase blockade: Prolactin directly inhibits 5-alpha-reductase activity — the enzyme that converts testosterone into DHT. DHT is the androgen that matters for erectile quality, penile tissue health, sexual sensitivity, and the maintenance of penile smooth muscle integrity. A man with elevated Prolactin who is trying to build DHT is pushing against a brake that is permanently applied.
- LH suppression: Prolactin suppresses the pulsatile release of luteinising hormone (LH) from the pituitary, which is the primary stimulus for testosterone production in the Leydig cells. Elevated Prolactin directly reduces testicular testosterone output at the source.
- Gynecomastia and water retention: Prolactin promotes the growth of estrogen-sensitive breast tissue and causes water retention via its action on renal tubular sodium reabsorption. Both are direct consequences of chronically elevated levels that many men attribute to other causes.
- Chronic serotonin elevation: Prolactin and serotonin are mutual amplifiers. Elevated serotonin drives Prolactin production; elevated Prolactin drives serotonin synthesis. The two hormones exist in a feedback loop that — once established — tends to self-perpetuate without intervention.

The Target Range — What Normal Actually Means

Standard laboratory reference ranges list male Prolactin as normal up to 15-18 ng/mL. From the perspective of optimised male hormonal function, this range is useless. It tells you what is common. It does not tell you what is functional.

The clinical data is clear on this:

- Above 10 ng/mL: measurable D2 receptor suppression is present, detectable 5-alpha-reductase inhibition begins, libido reduction is typical.
- Above 15 ng/mL: significant sexual dysfunction in most men. At this level, the Prolactin-LH suppression is severe enough to produce secondary hypogonadism-like symptoms even in men with structurally normal testes and pituitaries.
- 4-7 ng/mL: the functional target. This is where D2 receptors are free to respond to dopamine normally, where 5-alpha-reductase is operating without inhibition, and where the dopamine-testosterone-DHT cascade can run without a biochemical hand on the brake.

If your Prolactin is consistently above 20 ng/mL on multiple tests, rule out a prolactinoma (a benign prolactin-secreting pituitary microadenoma) before pursuing natural intervention. This is a straightforward, treatable condition that is frequently missed. An MRI of the pituitary is the diagnostic test.

The Root Cause: LPS, Gut, and the Prolactin-Inflammation Link

In men without a pituitary tumour or prolactin-elevating medication, the most common root cause of chronically elevated Prolactin is gut dysbiosis and LPS (lipopolysaccharide) endotoxaemia.

LPS is a component of the outer membrane of gram-negative bacteria in the gut. When the gut barrier is compromised — from poor diet, alcohol, chronic stress, or microbial imbalance — LPS translocates from the gut lumen into systemic circulation. Systemic LPS produces a chronic low-grade inflammatory state that directly stimulates prolactin secretion from the anterior pituitary via its action on lactotroph cells and via IL-6 and TNF-alpha signalling.

This creates the root-cause chain that Tier 1 is designed to interrupt: poor diet and gut dysbiosis → leaky gut barrier → systemic LPS endotoxaemia → chronic inflammation → elevated Prolactin → blocked DHT and suppressed dopamine → impaired sexual function.

No supplement stack will fully normalise Prolactin against a backdrop of chronic LPS endotoxaemia. Taurine, in Tier 1, is the first defensive move against BPA and microplastic-driven testicular inflammation. But if the gut is severely dysbiotic — if the "NYC Reality Check" applies to you — the gut must be addressed as a parallel protocol, not an afterthought.

> *"You can't medicate your way past a leaky gut. You can slow the damage. But until you fix the source, the LPS keeps coming, the*

> *inflammation keeps running, and the Prolactin keeps the brake applied."*

The Alcohol and Wheat Connection

Two dietary factors deserve specific mention because they directly and acutely elevate Prolactin through distinct mechanisms:

- Alcohol: metabolised primarily in the liver, alcohol directly stimulates prolactin secretion from the pituitary within hours of consumption. Even moderate intake (2-3 drinks) produces measurable Prolactin elevation that persists for several hours. Regular alcohol consumption maintains a chronically elevated Prolactin baseline. This is not a fringe claim. It is a documented, reproducible effect.
- Wheat and gluten: in men with gluten sensitivity (which does not require full coeliac disease to produce measurable systemic effects), gluten-derived peptides increase gut permeability directly and drive LPS translocation. The LPS-inflammation-Prolactin chain then follows. Many men notice significant improvements in libido, morning erections, and energy within 2-3 weeks of eliminating wheat, without making any other changes. The mechanism is Prolactin reduction via reduced LPS exposure.

Chapter 3: The Five Compounds — What Each One Is Doing

The Tier 1 stack is five compounds. Not five supplements chosen because they are popular, or because they test well in marketing copy. Five compounds chosen because each one addresses a specific, documented mechanism in the Homocysteine-Prolactin-mitochondria damage cycle.

Here is the stack, the mechanism, and the non-negotiable sourcing criteria for each.

Compound 1: Active B-Complex — 1 capsule, morning with food

This is the foundational intervention in Tier 1. Everything else in the stack depends on the methylation cycle being repaired. The B-Complex is the tool that repairs it.

The word "Active" is doing significant work in that name. A standard B-Complex from a high-street pharmacy will contain folic acid (inactive), cyanocobalamin (the cheapest and least bioavailable form of B12, which requires hepatic conversion before it becomes useful and which provides the body with a small dose of cyanide as a byproduct of that conversion), and pyridoxine hydrochloride (the standard form of B6, which can cause peripheral neuropathy at high doses and which must be converted to P5P before it is biologically active).

None of those standard forms are what your methylation cycle needs.

What you need:

- 5-MTHF (methylfolate): the active, bioavailable form of folate that bypasses the MTHFR bottleneck. This is the molecule that actually participates in the methylation cycle. Without it, Homocysteine cannot be recycled into methionine, and the cycle breaks down.
- Methylcobalamin or adenosylcobalamin (methyl-B12): the active forms. Methylcobalamin is the form that participates in the methylation cycle directly. Adenosylcobalamin is the form used in the mitochondria. Both are superior to cyanocobalamin. Look for a formula that contains one or both.
- P5P (pyridoxal-5-phosphate): the active form of B6. P5P serves two functions in this context: it is a required cofactor in the homocysteine transsulfuration pathway (converting Homocysteine to cystathionine, reducing the circulating Homocysteine load), and it is the natural antagonist to Prolactin at the pituitary. A single compound doing two jobs in this protocol. This is also why P5P is taken separately and at higher dose in the second compound in the stack.
- Riboflavin-5-phosphate (active B2): required as a cofactor for MTHFR enzyme activity itself. Without adequate B2, even taking 5-MTHF does not fully resolve the methylation bottleneck because the enzyme needs B2 to function. This is the most commonly omitted active form in B-Complex formulations.

- Thiamine (B1): as TTFD or benfotiamine for superior CNS and mitochondrial penetration. Standard thiamine HCl has poor bioavailability. B1 is an essential cofactor at the pyruvate dehydrogenase step of the Krebs cycle and at the alpha-ketoglutarate dehydrogenase step — both of which are rate-limiting for mitochondrial ATP production.

Read the label. If it says "folic acid," put it back. If it says "cyanocobalamin," put it back. The difference between an active B-Complex and a standard B-Complex is the difference between the supplement working and the supplement not working.

> **Expected effect:** Homocysteine begins falling within 4-8 weeks of consistent use. The methylation improvement also supports neurotransmitter production (serotonin, dopamine, norepinephrine all require SAMe as a methyl donor) and estrogen detoxification.

Compound 2: P5P — 50mg, morning and evening

P5P appears in the Active B-Complex, but not in sufficient quantity for its second function in this protocol: Prolactin reduction.

Pyridoxal-5-phosphate is one of the most potent natural Prolactin antagonists available without a prescription. The mechanism is direct: P5P modulates dopamine synthesis in the tuberoinfundibular dopaminergic pathway — the specific dopaminergic circuit that controls Prolactin release from the anterior pituitary. By supporting dopamine tone in this pathway, P5P reduces Prolactin secretion.

This is not a minor secondary effect. Clinical research has demonstrated that P5P supplementation at doses of 100-300mg daily produces measurable Prolactin reduction in men with hyperprolactinaemia. At the 100mg/day dose in this protocol (50mg twice daily), the effect is more modest but consistent over weeks to months of use.

The distinction between P5P and standard pyridoxine HCl is critical here. High-dose pyridoxine HCl (typically above 200mg/day for extended periods) is associated with sensory peripheral neuropathy — a form of nerve damage that can be permanent. P5P does not carry this risk at therapeutic doses because it is the end-form that the body uses directly, without the intermediate conversion steps that the pyridoxine-neuropathy mechanism depends on.

Do not substitute standard B6 for P5P in this protocol. The risk profile is different and the mechanism efficiency is different.

> **Timing note:** Split dose morning and evening because P5P has a relatively short half-life in active circulation. Twice-daily dosing maintains more consistent plasma levels than a single large morning dose.

Compound 3: Magnesium Malate — 400mg, before bed

Magnesium is the most important mineral in the Tier 1 stack, and it is the most universally deficient in the men who need this protocol most. The reasons for this

deficiency are systemic: modern agricultural soil is magnesium-depleted, alcohol depletes magnesium acutely, chronic stress depletes it chronically, and refined food contains almost none of it.

Magnesium participates as a cofactor in over 300 enzymatic reactions. The two most relevant for Tier 1:

- Mitochondrial ATP synthesis: ATP does not exist freely in the cell. It exists as Mg-ATP — a complex of ATP and magnesium. Without adequate magnesium, ATP cannot be stabilised and cannot be used efficiently by enzymes that require it. This is not a minor supporting role. It is the literal spark plug for cellular energy production. Low magnesium = low functional ATP regardless of what your mitochondria are doing with glucose and oxygen.
- Cortisol regulation and the racing brain: magnesium modulates the HPA axis, reducing the cortisol response to stress. The "racing brain" described in the Tier 1 brief — the inability to switch off, the sleep-onset difficulty, the chronic background anxiety — is, in a significant proportion of men, a magnesium deficiency symptom. The enzyme 11-beta-HSD2, which converts active cortisol to inactive cortisone, requires magnesium as a cofactor. Before-bed dosing is specifically timed to reduce cortisol during the sleep period, which is when the majority of testosterone production occurs.

The malate form is specified for a reason. Magnesium malate contains malic acid, which is a Krebs cycle intermediate. The combination of magnesium and malate provides both the mineral cofactor and a direct substrate for mitochondrial energy metabolism. Other forms — magnesium oxide (poorly absorbed, primarily a laxative), magnesium citrate (better than oxide, lower elemental magnesium percentage), magnesium threonate (CNS-penetrating but expensive and lower elemental Mg) — have different absorption profiles and different clinical applications. Malate is the choice for the mitochondrial and cortisol applications of Tier 1.

> **Side effect note:** Loose stools at high doses are a form-specific issue more common with magnesium oxide and citrate. Magnesium malate at 400mg elemental is well-tolerated for most men. If you are particularly sensitive, start at 200mg and titrate up over two weeks.

Compound 4: Purified Shilajit — 250mg, morning on empty stomach

Shilajit — also called Mumio in Eastern European research traditions — is a tar-like resinous substance found in high-altitude mountain rock formations, primarily in the Himalayas, Altai, and Caucasus ranges. It is formed over centuries by the decomposition of plant material under geologic pressure. What it contains that matters: fulvic acid, dibenzo-alpha-pyrones (DBPs), DBP-chromoproteins, and a complex of minerals in bioavailable ionic form.

Shilajit has two primary mechanisms relevant to Tier 1:

- Mitochondrial efficiency: the dibenzo-alpha-pyrones in Shilajit function as electron carriers in the mitochondrial electron transport chain, specifically supporting NADH:CoQ10 electron transfer at Complex I. They enhance the efficiency with which CoQ10 shuttles electrons through the inner mitochondrial membrane. Research has shown up to 29% improvement in CoQ10 activity when combined with Shilajit. This translates to more ATP produced per unit of substrate — a direct mitochondrial efficiency gain.
- Testosterone and minerals: Shilajit delivers minerals — including zinc, magnesium, iron, and copper — in fulvic acid-chelated ionic form that may have superior cellular uptake compared to inorganic mineral supplements. Research in men with below-average testosterone has shown approximately 20% increases in total testosterone with standardised Shilajit supplementation over 90 days. The mechanism is via the StAR protein pathway — Shilajit supports the mitochondrial transport of cholesterol that is the rate-limiting step in testosterone synthesis.

The sourcing warning cannot be overstated. Shilajit is one of the most adulterated supplements on the market. Cheap products — and most products sold online are cheap products dressed in premium packaging — contain heavy metals (lead, arsenic, cadmium) at levels that cause more damage than the Shilajit itself is intended to fix. You are not fixing a heavy-metal toxicity problem by adding more heavy metals.

Sourcing requirements:

- Third-party tested for heavy metals — not the manufacturer's own testing, independent laboratory certification.
- Standardised to >60% fulvic acid content. This is the active fraction. Products without standardisation are selling you unknown amounts of the active component.
- Resin form is preferred over powder. The traditional form is a resin. Powders have frequently been adulterated with fillers and sometimes with entirely different substances.

Morning on empty stomach is specified because fulvic acid absorption is reduced when competing with food-derived compounds in the digestive tract. Take it first thing, with a glass of water, before breakfast.

> **Cycling:** Shilajit must be cycled to avoid receptor habituation. 12 weeks on, 1 week off as a minimum. Extended use without breaks has been associated with reduced response — the mechanisms involved in testosterone support appear to require periodic withdrawal to maintain sensitivity.

Compound 5: Taurine — 3g, morning

Taurine is an amino sulfonic acid — technically not a classical amino acid because it lacks a carboxyl group — present in high concentrations in the brain, heart, skeletal

muscle, and retina. It is one of the most abundant amino acids in human physiology and one of the most consistently beneficial in the context of the Tier 1 goals.

Its role in Tier 1 is threefold:

- BPA and microplastic protection of the testes: this is the mechanism specified in the original Stub Club protocol and it is clinically grounded. BPA (Bisphenol A) and related endocrine-disrupting compounds are present in the blood of approximately 90% of men in Western populations. BPA is estrogenic, directly damages Leydig cell mitochondria (the cells that produce testosterone), and degrades androgen receptor expression. Taurine has demonstrated direct protective effects against BPA-induced Leydig cell apoptosis in research: it reduces BPA-driven oxidative stress in testicular tissue, preserves mitochondrial membrane potential in Leydig cells exposed to BPA, and maintains StAR protein expression (the rate-limiting step for testosterone synthesis) under BPA insult. In a world of ubiquitous plastic exposure, this protective function is not hypothetical. It is ongoing, relevant, and non-redundant with anything else in the stack.
- GABA-mimetic effect and sympathetic tone reduction: taurine activates GABA-A receptors and modulates the glycine receptor, producing an inhibitory neurotransmitter effect without the tolerance, dependence, or cognitive blunting associated with pharmaceutical GABAergic drugs. In practical terms, this means taurine reduces the sympathetic nervous system tone that is the primary neural contributor to performance anxiety and stress-driven testosterone suppression. Cortisol and testosterone compete for the same cholesterol precursor. Anything that reduces chronic cortisol load improves the environment for testosterone synthesis.
- Cardiovascular and vascular support: taurine is a natural calcium channel modulator that reduces blood pressure, improves endothelial function, and supports heart rate variability — a marker of vagal tone and parasympathetic health. By improving the vascular environment, taurine supports the nitric oxide pathway that is simultaneously being repaired by the methylation corrections of the B-Complex and P5P.

3 grams is the clinically relevant dose for these mechanisms. Standard "daily support" doses in the literature are 500mg-1g. The protective and functional effects described above are more consistently observed at 2-4g daily. 3g is the practical middle of that range.

> **Important note:** Taurine supplementation is generally well-tolerated. The main adverse effect reported is loose stools at doses above 4-5g. At 3g, gastrointestinal side effects are uncommon. Take with food if sensitivity is present.

Chapter 4: The Schedule — 90 Days, No Exceptions

The 90-day commitment is not arbitrary. It reflects the biology of what Tier 1 is repairing.

Homocysteine normalisation via methylation support typically shows measurable reduction within 4-8 weeks. Full stabilisation of the methylation cycle — including the downstream effects on neurotransmitter production, estrogen clearance, and DNA repair capacity — requires 90+ days of consistent supplementation.

Prolactin reduction via P5P and lifestyle modification is slower. Meaningful reduction from natural interventions typically requires 8-12 weeks. The LPS endotoxin load that is driving the prolactin elevation will not resolve until the gut environment begins to improve, which itself takes weeks to months.

Shilajit's testosterone effects, in the research demonstrating the ~20% increase, were observed at 90 days. Not 30. Not 60. The mineral delivery and mitochondrial efficiency effects accumulate over time.

Taurine's protective effects are ongoing and accumulative — the compound is being depleted by BPA exposure continuously, and daily supplementation maintains the protective tissue concentrations required for its effects.

The Daily Protocol

Active B-Complex — 1 capsule | Morning with food | *Repair the methylation cycle. Kill Homocysteine at the source.*

P5P — 50mg x 2 | Morning and evening | *Lower Prolactin. Unlock D2 receptors. Support methylation cofactor needs.*

Magnesium Malate — 400mg | Before bed | *Spark plug for ATP synthesis. Lower cortisol. Stop the racing brain.*

Purified Shilajit — 250mg | Morning, empty stomach | *Drive mitochondrial efficiency. Support testosterone via StAR pathway.*

Taurine — 3g | Morning with or without food | *Protect testes from BPA. Lower sympathetic tone. Support vascular function.*

Take the B-Complex and the first P5P dose together in the morning with food. Take Shilajit separately, before food, with water. Take the second P5P dose in the evening with dinner. Take Magnesium Malate 30-60 minutes before your intended sleep time.

The 12-Week Cycle and Break

After every 12 weeks of continuous use, take a one-week break from the full stack. During the break week, you may optionally continue Magnesium Malate (as a non-hormonal compound it does not require cycling) and taurine (same reasoning), but step off the B-Complex, P5P, and Shilajit for 7 days.

The cycling serves two purposes:

- Receptor sensitivity maintenance: compounds that interact with hormonal pathways — P5P at the pituitary, Shilajit via the StAR protein and mitochondrial efficiency — can produce receptor down-regulation or reduced responsiveness with uninterrupted long-term use. A break period resets sensitivity.
- Biological assessment: the break week gives you an opportunity to assess your baseline without the stack. If the improvements you experienced during the 12-week cycle are retained during the break, the underlying biology has shifted. If they deteriorate significantly, the stack is still compensating for an unresolved underlying cause — most likely gut health.

The 30-Day Check: The NYC Reality Check

At 30 days, you are looking for two specific signals that the hardware is responding:

- Morning erections — specifically, the return or improvement of nocturnal penile tumescence (NPT), which is the parasympathetically-driven erection that occurs during REM sleep. Morning erections are the most reliable low-tech indicator that the vascular and hormonal systems are improving. Their presence indicates that the NO pathway is functioning, Prolactin is declining toward the functional range, and REM quality is adequate for the testosterone-release pulse that peaks during early morning hours.
- Sleep quality improvement — specifically, deeper sleep, reduced cortisol-driven waking in the early morning hours (typically 3-4am), and improved morning energy. Magnesium Malate and taurine together produce measurable improvements in sleep architecture within 2-4 weeks in men who are deficient, which is most men taking this protocol.

If neither of these signals is present at 30 days, the gut is the most likely explanation. LPS endotoxin load prevents the absorption and utilisation of fat-soluble nutrients, impairs the B-vitamin dependent enzymes in the methylation cycle by generating competing inflammatory signals, and maintains the Prolactin-elevating inflammatory state regardless of what supplements are being taken.

A simple gut-status test: do you have regular bowel movements (1-2 daily), minimal bloating, and do you tolerate a variety of foods without significant digestive discomfort? If the answer is no to any of these, the gut is the bottleneck.

The gut intervention is covered in Chapter 6. Do not skip directly to Tier 2 if the 30-day check is negative. Fix the gut. Then Tier 1 will work. Then Tier 2 will build on a foundation that actually holds.

Chapter 5: What You Are Fighting From the Outside

Tier 1 is fixing the internal damage. But if you are continuously re-importing the external sources of that damage during the 90 days, you are filling the bathtub with the drain open. This chapter covers the primary external inputs that must be reduced or eliminated for Tier 1 to produce its full effect.

Alcohol

Alcohol is the single most counterproductive substance during a Tier 1 protocol. It does damage on every front simultaneously:

- Directly depletes B-vitamins — B1, B2, B6, B12, and folate are all depleted by alcohol metabolism through the liver. You are supplementing active B-vitamins in the morning and the alcohol is degrading them by the evening.
- Elevates Prolactin acutely and chronically — two drinks produce measurable Prolactin elevation within hours. Regular drinking maintains a chronically elevated Prolactin baseline.
- Disrupts REM sleep — alcohol suppresses REM sleep, which is where the majority of daily testosterone production occurs. Even moderate intake (2-3 drinks) measurably reduces REM fraction for the first 4-6 hours of sleep.
- Increases gut permeability — alcohol is directly toxic to intestinal tight junction proteins, increasing LPS translocation and driving the endotoxaemia-inflammation-Prolactin chain.

This is not a lifetime alcohol prohibition. It is a 90-day protocol. The minimum requirement is significantly reducing intake. The optimal approach is complete abstinence during the protocol period.

Plastics and Endocrine Disruptors — Practical Reduction

BPA is present in approximately 90% of Western men at detectable blood levels. Complete elimination is not realistic. Meaningful reduction is.

- Do not heat food in plastic containers. Heating dramatically increases BPA and phthalate leaching into food. Use glass or stainless steel for reheating.
- Do not drink from plastic bottles that have been in a warm environment (a car, direct sunlight, a gym bag). The leaching rate increases exponentially with temperature.
- Reduce tinned food consumption. Most tin cans are lined with BPA-containing epoxy resin. BPA-free cans exist but may substitute other problematic compounds.
- Use a water filter. Municipal water supplies in most cities contain measurable levels of endocrine disruptors including phthalates, atrazine, and pharmaceutical estrogens. A basic activated carbon filter reduces but does not eliminate these.

- Choose glass, stainless steel, or ceramic for food storage and cooking. The capital cost is modest; the ongoing benefit is continuous.

Taurine is providing internal defence against BPA-driven testicular damage. External reduction of BPA exposure works in parallel with that internal defence. Both matter.

The 5-AR Blockers — The Supplements That Are Working Against You

Several compounds commonly taken by men for "health," hair, or prostate support are powerful inhibitors of 5-alpha-reductase — the enzyme that converts testosterone into DHT. Taking any of these during a Tier 1 protocol is not a neutral act. It is an active counterforce to the hormonal restoration you are attempting.

- Saw palmetto: one of the most widely sold supplements for "prostate health." Its mechanism is direct 5-AR inhibition, comparable in potency to pharmaceutical finasteride in some research. Avoid entirely.
- Finasteride and dutasteride: pharmaceutical 5-AR inhibitors. If you are taking these, Tier 1 will not fully restore DHT because the primary conversion enzyme is blocked. Discuss cessation with your physician before beginning this protocol if you are on these medications.
- Pygeum: included in many prostate blends alongside saw palmetto. Strong 5-AR inhibitory activity. Avoid.
- High-dose curcumin: anti-inflammatory and useful for some applications, but at supplement doses (500mg+) it demonstrates moderate 5-AR inhibitory and androgen receptor antagonist activity. Dietary turmeric is fine. Supplement-dose curcumin capsules are not during this protocol.
- DIM (Diindolylmethane): widely marketed as an "estrogen blocker." At doses above 100mg it is not primarily an estrogen blocker — it is an mTOR inhibitor and androgen receptor antagonist. It blocks testosterone at the receptor level. Avoid entirely.
- Ashwagandha: serotonergic (MAO-inhibiting, tryptophan-elevating) and has demonstrated 5-AR inhibitory activity in some research. The serotonergic effect directly worsens the 5-HT2C overactivation that suppresses dopamine. Remove from your stack for the 90-day protocol period.
- Black pepper in large quantities: contains piperine, which at high concentrations demonstrates 5-AR inhibitory activity. Normal dietary use is fine. Mega-dosed "black pepper extract" in supplement form is not.

Ibuprofen and Paracetamol — The Silent Androgen Receptor Suppressors

Both ibuprofen (NSAID) and paracetamol/acetaminophen (analgesic) have been documented in clinical research to reduce androgen receptor expression with regular use. This is not a contraindication to occasional use. If you have a headache or an

acute injury, using one of these medications is a reasonable choice. The issue is chronic daily use during a protocol that is attempting to restore androgen receptor sensitivity.

If you are using ibuprofen or paracetamol daily — for chronic pain management, for example — discuss alternatives with your physician before and during the Tier 1 protocol.

Chapter 6: The Gut — Why It Can Make or Break Tier 1

The 30-day check in Chapter 4 will tell you if your gut is the bottleneck. This chapter tells you what to do about it.

The Root-Cause Chain

The gut-hormone connection is not indirect. It is a direct, documented causal chain:

Mitochondrial dysfunction from chronic inflammation → reduced ATP production in intestinal epithelial cells → reduced vagal tone (the vagus nerve requires ATP to function as a gut-motility driver) → slowed intestinal motility → bacterial overgrowth (SIBO) → increased LPS load → leaky gut → systemic LPS endotoxaemia → chronic inflammation → suppressed mitochondrial steroidogenesis → elevated Prolactin → blocked DHT → suppressed dopamine.

Every step in that chain is documented. This is why the gut is the foundational prerequisite and why fixing it is not optional if Tier 1 is not producing results.

The Basic Gut Protocol

This is not a full gut protocol — that is a separate subject. This is the minimum viable intervention to create the gut environment in which Tier 1 can function:

- Eliminate or significantly reduce: alcohol, refined sugar, seed oils (canola, soybean, sunflower, corn), wheat and gluten (especially if symptomatic), and ultra-processed food generally. These are the primary drivers of gut barrier disruption and LPS-producing dysbiosis.
- Add dietary fibre: the gut bacteria that produce butyrate (the short-chain fatty acid that maintains gut barrier integrity, reduces inflammation, and directly supports DHT production via its effects on testosterone metabolism in the gut) are fed by fermentable fibre. Target 25-35g of fibre daily from diverse plant sources. If you currently eat minimal fibre, introduce it gradually to avoid bloating.
- Butyrate support: either via dietary fibre as above, or via sodium butyrate supplementation (300-600mg daily). Butyrate serves as the primary fuel for colonocytes (gut lining cells), maintains tight junction integrity, and has demonstrated HDAC inhibitory activity — which is relevant for androgen receptor gene expression. It also directly stimulates SIRT3 in the gut wall, reducing NF-kB inflammatory signalling.
- Raw carrot salad: a simple and underrated gut intervention. Raw carrot contains specific fibres that bind and help clear excess estrogens and endotoxins in the gut. 1-2 raw carrots daily, grated or whole, with a small amount of vinegar and coconut oil (which has antimicrobial properties against

LPS-producing bacteria). This was a specific recommendation from the clinical coaching source data — simple, cheap, and reproducible.

- Bile flow support: poor bile flow is a major cause of fat malabsorption (which means poor absorption of fat-soluble vitamins including D, K2, and the fat-soluble components of your B-Complex), estrogen recirculation (estrogens are excreted in bile and reabsorbed when bile flow is inadequate), and SIBO (bile is naturally antimicrobial against the bacteria that overgrow in SIBO). Support bile flow with: bile salts (ox bile) taken with high-fat meals if gallbladder function is suspected to be poor, bitter foods (dandelion greens, arugula, radicchio), and adequate dietary fat (do not eat a very low-fat diet; fat stimulates bile release).
- Consider GI-MAP testing if gut symptoms are significant: a stool test that identifies specific pathogens, dysbiotic organisms, markers of gut permeability (zonulin), and inflammatory markers. This is the functional medicine standard for understanding what is actually happening in the gut rather than guessing. Available through private labs in most markets.

Timeline: Basic dietary reform produces measurable changes in gut microbiome composition within 2-4 weeks. Barrier integrity restoration takes 8-12 weeks of consistent dietary adherence and targeted supplementation. The LPS endotoxin load begins falling within weeks of dietary reform — which is why the 30-day check is the right timeframe to assess gut impact on Tier 1 efficacy.

The Lab Work That Matters

If you invest in one set of lab tests before and after Tier 1, this is what to test:

- Homocysteine (fasting): your primary baseline marker. Target < 8 µmol/L. Test before starting Tier 1 and at 12 weeks.
- Prolactin: your secondary baseline marker. Target 4-7 ng/mL. Test before and at 12 weeks.
- Active B12 (holotranscobalamin / holo-TC): not standard B12. Standard B12 measures total circulating B12 including inactive forms. Holo-TC measures only the biologically available fraction. Target > 400 pmol/L.
- hs-CRP: high-sensitivity C-reactive protein. Marker of systemic inflammation. Above 1 mg/L indicates active BH4 depletion and ADMA production — the vascular environment that makes Homocysteine damage worst.
- Fasting insulin: target < 10 µIU/mL. Insulin resistance raises SHBG, impairs eNOS, and worsens the LPS-driven inflammatory state.
- Free T3 (thyroid): Free T3 is the active thyroid hormone and the #1 driver of 5-alpha-reductase activity. Low Free T3 — even with normal TSH — dramatically reduces DHT conversion. TSH alone is an inadequate thyroid assessment. Test Free T3, Free T4, and Reverse T3.

- DHT: the downstream marker that tells you whether the testosterone-to-DHT conversion is working. If Homocysteine is falling and Prolactin is falling but DHT is not rising, look at Free T3 and 5-AR blocker exposure.

These tests are available through your GP or through private/direct-access labs in most markets. The investment is modest relative to the information it provides.

Chapter 7: The Complete Tier 1 Reference

A single-page reference for the complete Tier 1 protocol.

The Stack

Active B-Complex — 1 cap | Morning with food | *Repair methylation. Kill Homocysteine. Must contain 5-MTHF, methylcobalamin, P5P, and active B2.*

P5P (active B6) — 50mg x 2 | Morning + evening | *Lower Prolactin. Unlock dopamine. Support homocysteine transsulfuration.*

Magnesium Malate — 400mg | Before bed | *ATP spark plug. Cortisol reduction. Fix the racing brain and improve REM.*

Purified Shilajit — 250mg | Morning, empty stomach | *Mitochondrial efficiency. Testosterone via StAR pathway. Heavy-metal tested only.*

Taurine — 3g | Morning | *BPA/microplastic testicular protection. GABA support. Vascular function.*

The Schedule

- ☐ Daily. No exceptions. 90 days minimum.
- ☐ Cycle: 12 weeks on, 1 week off. Repeat.
- ☐ 30-day check: morning erections present? Sleep improved? If no to both — address the gut before continuing.

Remove During the 90 Days

- ☐ Alcohol (or significantly reduce)
- ☐ Saw palmetto, pygeum, and any other "prostate support" supplement
- ☐ Ashwagandha
- ☐ High-dose curcumin supplements
- ☐ DIM at doses above 100mg
- ☐ Plastic food containers and bottles for hot food/drink
- ☐ Refined sugar and seed oils where possible

Lab Targets

Homocysteine: < 8 µmol/L (ideally 5-8). Test at baseline and week 12.

Prolactin: 4-7 ng/mL. Test at baseline and week 12.

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

hs-CRP: < 1.0 mg/L.

Free T3: Upper third of reference range.

DHT: ≥ 10% of total testosterone.

Fasting Insulin: < 10 µIU/mL.

The 30-Day Signals

- Morning erections: the return or improvement of NPT is the primary clinical indicator that the NO pathway and hormonal environment are improving.
- Sleep quality: deeper sleep, reduced early-morning waking, improved morning energy within 2-4 weeks if Magnesium Malate and Taurine are addressing deficiency.
- If neither signal is present at 30 days: the gut is the bottleneck. See Chapter 6. Do not skip to Tier 2.

Appendix: Supplements to Avoid During Tier 1

These compounds are directly counterproductive to the Tier 1 goals. Remove them for the 90-day protocol period.

Saw palmetto

5-AR inhibitor comparable in potency to finasteride. Directly blocks DHT production. Found in most "prostate support" blends. Avoid entirely.

Pygeum

Strong 5-AR inhibitory activity. Almost always combined with saw palmetto. Avoid.

Finasteride / dutasteride

Pharmaceutical 5-AR inhibitors. Tier 1 cannot restore DHT while these are active. Discuss cessation with your physician.

Ashwagandha

Serotonergic (MAO-inhibiting), possible 5-AR inhibitory activity. Works against dopamine tone and DHT simultaneously.

High-dose curcumin (>500mg supplement dose)

Moderate 5-AR inhibitory and androgen receptor antagonist activity at supplement doses. Dietary turmeric is fine.

DIM above 100mg

Testosterone blocker and mTOR inhibitor, not an estrogen blocker, at doses above 100mg. Avoid entirely.

High-dose quercetin (supplement dose)

5-AR inhibitory effects at supplement doses. Dietary sources are fine.

Rosemary oil (topical or high-dose oral)

Demonstrated 5-AR inhibitory activity. Avoid topical scalp application during protocol.

Ibuprofen / paracetamol (chronic daily use)

Reduces androgen receptor expression with chronic use. Occasional use is acceptable.

IV NAD+ drips

Heavy methyl-donor drain, raises Homocysteine, temporary plasma spike with minimal cellular benefit. Directly counterproductive to the methylation repair goals of Tier 1.

Standard folic acid (not 5-MTHF)

The inactive synthetic form competes with 5-MTHF at folate receptors. If your B-Complex contains folic acid, replace it.

Cyanocobalamin

The least bioavailable B12 form. Replace with methylcobalamin or adenosylcobalamin.

What Comes Next

Tier 1 is the foundation. It is not the ceiling.

Once the methylation cycle is running, Homocysteine is below 8, Prolactin is heading toward the 4-7 ng/mL range, mitochondrial ATP production is restored, and the gut is not actively undermining the whole system — then you are ready to build.

Tier 2 of the Stub Club Protocol addresses the next layer: the endothelial rebuild. The glycocalyx repair. The eNOS recoupling. The nitric oxide restoration that Tier 1 has been clearing the path for. Tier 2 introduces the compounds that directly rebuild the vascular hardware. But they only work because Tier 1 has stopped the internal rust first.

Tier 3 addresses the hormonal amplification layer — DHT support, AR sensitivity, dopaminergic herbs cycled correctly — with the full understanding that signal amplifiers only work when the receiver is functional.

Do Tier 1 properly. Do it completely. Give it 90 days. Get the lab work. Check the signals.

Then build.