Appendix A

The Author's Protocol

The Quiet Biology Protocol

Operational specification of the constraint-based maintenance framework

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What follows is a transparent account of the specific protocol the author uses to manage his own low-burden biochemical recurrence following definitive local therapy. It is not a recommendation. Every element described here was arrived at through research, clinical consultation, and individual assessment of risk and benefit. What is appropriate for one man's biology, disease state, and circumstances will not be appropriate for another's. The purpose of including this document in the clinical submission is honesty — the Quiet Biology framework has argued throughout for informed engagement with one's own biology, and it would be inconsistent to make that argument while concealing what that engagement actually looks like in practice.

The protocol operates on a single governing principle: biological containment rather than cytotoxic intervention, during a period when disease burden is low, PSA kinetics are stable, and the evidence for aggressive treatment is thin. It is designed to be reversible, time-limited, and responsive to defined biological signals. If those signals change, the protocol changes with them. The conditions under which it changes are defined in the companion document, Appendix B: Phase-Transition Criteria.

The Framework

The protocol is structured around two repeating phases. The active phase runs for eight weeks. The washout phase runs for four weeks. PSA is measured during the washout, not during the active phase, to ensure the reading reflects biology rather than pharmacological perturbation.

The active phase is not a treatment in the oncological sense. It does not aim to kill cancer cells or suppress androgen signalling. It aims to maintain a metabolic and cellular environment that is less hospitable to disease progression — to slow the evolutionary pressure that drives resistance, preserve the interpretive value of PSA as a signal, and protect the therapeutic options that will matter most if and when disease burden increases.

The washout phase is not a rest. It is a deliberate biological reset — allowing normal tissues to recover, the immune system to recalibrate, and the PSA signal to return to an unperturbed baseline before the next cycle begins.

Phase One: The Active Block (Weeks 1–8)

Rapamycin — 6mg once weekly, Sunday evening

Rapamycin is an mTORC1 inhibitor — it interrupts one of the primary signalling pathways that drives cellular growth, protein synthesis, and proliferation. At transplant or oncology doses, taken daily, it is an immunosuppressant. At 6mg once weekly, the pharmacokinetic picture is entirely different. mTORC1 inhibition is transient, normalising within days of each dose. At this dosing pattern the evidence suggests immune function is preserved and in some contexts improved, not suppressed.

The weekly dose is taken Sunday evening, which positions the peak mTORC1 inhibition on Monday — the lightest exercise day of the week. This is intentional. Rapamycin taken close to resistance training blunts the mTORC1-driven adaptation response. By Sunday evening, the heaviest training of the week — the Saturday kettlebell session — is complete. The early week Pilates and yoga sessions are low enough in intensity that the overlap with rapamycin's active window is unlikely to meaningfully compromise recovery.

The cancer biology rationale for mTORC1 inhibition at low disease burden is twofold. First, mTORC1 is frequently upregulated in prostate cancer cells and drives proliferative signalling — intermittent inhibition applies a metabolic constraint without the strong selective pressure of continuous suppression. Second, rapamycin promotes autophagy — the cellular housekeeping process by which damaged components, including dysfunctional mitochondria, are cleared. This has relevance both to cancer cell biology and to normal tissue maintenance.

Doxycycline — 50mg daily, alternate weeks only, days 3–7 of each week

Doxycycline is included here not as an antibiotic but for a set of biological effects that are mechanistically distinct from its antimicrobial properties. At 50mg — well below the 100–200mg doses used to treat infection — doxycycline inhibits mitochondrial ribosomal translation, suppresses matrix metalloproteinases involved in tissue invasion, and has anti-inflammatory and anti-stemness effects that have been characterised in cancer biology research.

The mitochondrial ribosome target is worth understanding. Mitochondria carry their own ribosomes, evolutionarily derived from bacterial ancestors. These prokaryotic ribosomes are structurally different from the cytoplasmic ribosomes that handle most cellular protein synthesis, which is why tetracycline antibiotics — evolved to disrupt bacterial protein synthesis — can affect mitochondrial function in mammalian cells without broadly disrupting cytoplasmic translation. The therapeutic interest lies in evidence that aggressive cancer cells, and cancer stem cells in particular, are disproportionately dependent on mitochondrial respiration compared to most normal differentiated cells. A constraint applied at the mitochondrial ribosome level hits harder where the metabolic dependency is greatest.

The pulsing structure — alternate weeks only, initiated 48–72 hours after the weekly rapamycin dose — is deliberate on two counts. First, it preserves the early autophagy window that rapamycin opens before mitochondrial stress is applied, separating two complementary pressures rather than running them simultaneously. Second, it limits cumulative mitochondrial exposure in normal tissues, including Schwann cells in the perineural environment, which have their own significant mitochondrial demands. A continuous low-dose antibiotic would also progressively disrupt the gut microbiome — the pulsed structure, combined with prebiotic supplementation as a constant baseline, is intended to prevent that.

PSA is not measured within fourteen days of doxycycline exposure. Doxycycline can transiently influence inflammatory signalling and PSA transcription independently of tumour burden. Measuring PSA during or immediately after a doxycycline pulse risks misreading a pharmacological effect as a biological signal.

Phase Two: The Washout (Weeks 9–12)

The four-week washout is structured rather than empty. Weeks nine and ten include Urolithin A at 500mg daily and Chinese skullcap at 500mg daily. Weeks eleven and twelve are clear of all protocol compounds.

Urolithin A — 500mg daily, weeks 9–10

Urolithin A is a mitophagy inducer — it promotes the selective clearance of damaged mitochondria through the PINK1/Parkin pathway, distinct from rapamycin's autophagy induction. After eight weeks of intermittent mitochondrial ribosome inhibition, accumulated dysfunctional mitochondria in normal tissues need clearing. Human RCT evidence supports Urolithin A's effects on mitochondrial function and muscle performance; the application to recovery from the doxycycline cycles is mechanistically coherent rather than directly proven.

It also has preclinical evidence for effects on cancer cell senescence that extend a gentler biological pressure into the washout without the systemic burden of the active compounds.

Chinese Skullcap (Scutellaria baicalensis) — 500mg daily, weeks 9–10

Chinese skullcap — baicalein and baicalin as its primary active constituents — has consistent preclinical evidence for NF-κB suppression, NLRP3 inflammasome inhibition, and macrophage repolarisation toward a resolution phenotype. It also shows mild androgen receptor pathway interference in cell-line and animal models. It provides a low-pressure continuation of the metabolic containment strategy during the first half of the washout while the body recovers from the active block. Human RCT evidence for anti-inflammatory or cancer outcomes is absent; use here is mechanistic and hypothesis-driven.

Weeks 11–12: Clear Window

The final two weeks of the washout are clear of all protocol compounds. This is when PSA is measured. The two-week clearance period is essential to signal purity — the PSA reading should reflect the biology of the disease, not the pharmacology of the protocol.

The Hormonal and Metabolic Foundation

Running continuously beneath the cycling protocol is a hormonal and metabolic foundation that does not change with the eight-week cycle structure.

Testosterone Replacement — 60mg weekly, Friday administration

Testosterone replacement maintains a stable physiological androgen environment. The short version of the reasoning: a decade of functional hypogonadism was the hormonal environment in which the cancer developed and progressed. Restoring physiological testosterone levels, with careful monitoring, was a considered decision made with oncological awareness and is not undertaken carelessly or without surveillance. Population-based analyses have not demonstrated increased prostate cancer-specific or overall mortality among men receiving TRT after definitive treatment for localised disease, though no large RCT has evaluated recurrence risk as a primary endpoint. Evidence is strongest in low- and favourable-intermediate-risk men after definitive treatment with stable PSA — which describes this setting.

Aromatase Inhibitor — Saturday and Tuesday

The Saturday dose manages the early conversion peak following the Friday injection; the Tuesday dose addresses the tail. The spacing is calibrated to avoid over-suppression of estradiol, which matters for bone health, cardiovascular function, and cognitive clarity. Estradiol is monitored at every cycle.

Pioglitazone — 7.5mg daily

Pioglitazone is a PPAR-γ agonist. At full diabetic dosing — 30–45mg — it is used for insulin resistance. At 7.5mg the primary rationale is metabolic and anti-inflammatory: PPAR-γ activation suppresses NF-κB-driven inflammatory signalling, promotes macrophage repolarisation toward a resolution phenotype, and drives cellular differentiation. These effects on the tumour microenvironment and metabolic resensitisation are the mechanistic targets. The dose is deliberately kept below the threshold associated with the bladder cancer signal seen in long-term full-dose studies, though that signal is weak and contested in the literature.

Retatrutide — 2mg weekly, Monday

Retatrutide is a triple agonist at GLP-1, GIP, and glucagon receptors. GLP-1 receptor activation enhances insulin secretion and slows gastric emptying; GIP receptor activation improves lipid and glucose metabolism; glucagon receptor activation increases energy expenditure and drives adipose reduction. Together these produce metabolic effects that exceed what dual agonists achieve. The primary effect in this context is reduction of adipose tissue as an inflammatory reservoir and secondary oestrogen source, alongside improvement of insulin sensitivity and metabolic flexibility. Retatrutide remains investigational; Phase 2 and 3 trial data demonstrate robust metabolic modulation, though no trials have evaluated it in a cancer management context. It is taken Monday, alongside rapamycin during the active block, with both compounds working in the direction of reduced anabolic signalling.

Tadalafil (Cialis) — 5mg daily

Daily low-dose tadalafil supports endothelial function and pelvic blood flow — relevant both to post-surgical recovery and to cardiovascular health in the context of the overall protocol. The daily low dose produces consistent PDE5 inhibition rather than the episodic pharmacological profile of as-needed higher dosing.

The Lifestyle Infrastructure

The pharmacological protocol does not operate in isolation. The biological effects of the compounds described above are shaped by the metabolic environment they enter, and that environment is actively maintained.

Exercise

Exercise follows a weekly progression that mirrors the pharmacokinetic arc of the rapamycin dose: Pilates on Monday and Tuesday during the period of peak mTORC1 inhibition, building through power yoga on Wednesday, a rest day Thursday, high-intensity cardio with light weights on Friday, and the heaviest session — HIIT with kettlebells — on Saturday. Sunday is rest. This structure ensures the most demanding adaptive stimulus arrives when rapamycin's acute effects have substantially cleared.

Protein intake is timed to at least three hours after exercise on most days. The mTORC1 re-activation signal that follows a protein-rich meal — driven primarily by leucine — is most useful when it arrives into a tissue environment that has completed its exercise-induced AMPK activation. Combining a protein bolus with active exercise-induced AMPK and concurrent rapamycin-mediated mTORC1 suppression would produce conflicting signals. The timing separates them.

Sauna and Cold

Sauna at therapeutic temperature for fifteen minutes followed by three minutes of cold plunge at eight to ten degrees Celsius, five times weekly. The sauna component drives heat shock protein expression, supports cardiovascular adaptation, and has evidence for growth hormone pulsatility. Large prospective cohort data from Finland associate regular sauna use with reduced cardiovascular and all-cause mortality in a dose-dependent manner; five sessions per week at this duration falls within the range associated with the strongest observed benefit. The cold plunge produces substantial norepinephrine release — 200 to 530 percent above baseline within minutes — and activates brown adipose tissue through AMPK and PGC-1α pathways, compounding the metabolic effects of the retatrutide. The combination of sauna and cold as a structured hormetic stress — applied consistently, not occasionally — contributes to the same AMPK activation and mitochondrial biogenesis that the pharmacological protocol is trying to support.

Sleep

Sleep is anchored consistently: bed between eight-thirty and nine, wake between four-thirty and five. Sleep supplementation includes magnesium glycinate 500mg, sustained-release melatonin 3mg, and glycine 3g — the glycine at a dose shown in the literature to improve sleep quality and reduce core body temperature during sleep. D3 at 5000 IU with K2 and DHA at 1000mg are taken daily as nutritional foundations rather than protocol-specific interventions.

Gut Health

Gut health is maintained through miso soup four times weekly as a consistent fermented food source, and partially hydrolysed guar gum (PHGG) as a daily prebiotic fibre. PHGG ferments slowly, producing butyrate without significant gas load, and provides the microbiome with a consistent fermentable substrate to rebuild around during and after each doxycycline cycle. The microbiome's integrity matters for immune function, inflammatory tone, and metabolic signalling — all of which the protocol is trying to support.

Monitoring

The protocol is monitored through a comprehensive biological panel assessed at the end of each washout period — during the two-week clear window before the next active block begins. The panel is not primarily a cancer monitoring tool. It is a systems biology snapshot: a full picture of whether the biological environment is moving in the intended direction and whether the protocol is imposing costs that outweigh its benefits.

Panel	Markers	Rationale
Full blood count	CBC with differential	*Immune and red cell health; bone marrow signal*
Hormonal	Total and free testosterone, SHBG, estradiol, LH, FSH, DHEA-S, cortisol, TSH	*TRT titration; adrenal function; stress response*
Metabolic	HbA1c, fasting glucose, fasting insulin, HOMA-IR	*Insulin sensitivity; metabolic environment*
Lipids	Full lipid panel, ApoA1, ApoB, Lp(a)	*Cardiovascular risk; atherogenic particle burden*
Inflammation	Sensitive CRP, IL-6 (where available)	*Inflammatory tone; tumour microenvironment signal*
Organ function	Full metabolic panel, electrolytes, kidney function, ALP, LDH	*Liver and kidney tolerance of protocol compounds; systemic aggressiveness sentinels*

Panel	Markers	Rationale
Cancer markers	PSA (sensitive assay)	*Measured during washout only; not within 14 days of doxycycline*

PSA is interpreted in the context of the full panel rather than in isolation. A stable PSA alongside worsening inflammatory markers or declining metabolic health is as relevant as a rising PSA alongside an otherwise clean panel. The goal is not to manage a number. It is to understand what the biology is doing.

Stop Criteria

The protocol is discontinued — not escalated — if any of the following criteria are met:

- PSA acceleration that persists across two consecutive washout measurements
- Worsening insulin resistance or lipid burden despite the metabolic interventions
- Persistent fatigue or immune susceptibility that does not resolve during the washout period
- Any clinical evidence suggesting disease progression that warrants imaging or oncological review

Discontinuation means returning to watchful surveillance and clinical management under the oncology team. It does not mean the framework has failed. It means the biology has moved into a different phase and the response should move with it. The full escalation criteria — including the six phase-transition signals that govern transition to conventional oncologic management — are defined in Appendix B.

The protocol is designed to be one phase of a longer strategy, not a permanent alternative to that strategy.

A Final Note on Transparency

> *What is described here is one man's protocol, arrived at through research and refined through clinical engagement, applied to a specific biology and a specific disease state. It is presented transparently because the quality of clinical dialogue depends on the clinician understanding what their patient is*

actually doing and why — not a sanitised summary of it. Another man with the same diagnosis might reasonably arrive at entirely different conclusions.

The point is not the protocol. The point is the quality of reasoning that produced it — and the willingness to remain accountable to the biology rather than to the framework.

References

Organised by compound and domain. Where a paper is relevant to multiple sections it is listed at first appearance only. Evidentiary limitations are noted throughout; mechanistic and preclinical sources are distinguished from clinical and epidemiological evidence.

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