EVIDENCE-BASED CLINICAL PLAYBOOK · 2026 EDITION

The Testosterone Recovery Playbook

Science-backed protocol to support healthy male testosterone levels — physiology, diagnostics, nutrition, training, sleep, stress, supplements, and the 90-day implementation plan.

Scope Adult male endocrinology · lifestyle medicine · preventive care
Framework Endocrine Society clinical guidance · CDC HoST assay standardization
Evidence grading Strong · Moderate · Weak · Insufficient
Format Print-ready reference document

This document is an educational synthesis for clinicians and informed lay readers. It does not constitute individual medical advice, diagnosis, or a prescription. Testosterone deficiency is a clinical diagnosis requiring history, examination, and laboratory confirmation.

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EVIDENCE TIER DEFINITIONS

STRONG Consistent benefit across multiple human randomized controlled trials or meta-analyses, with plausible mechanism and adequate sample size.

MODERATE At least one reasonable-quality human RCT or a consistent body of smaller trials, but limited by sample size, single-centre design, or industry funding.

WEAK Small, short, uncontrolled or conflicting human studies; effect sizes that overlap with measurement noise.

INSUFFICIENT Animal or mechanistic data only, observational associations without intervention trials, or no credible human testosterone outcome data.

SECTION ONE

Executive Summary

Testosterone is not a performance accessory. It is a metabolic signal that responds — measurably and reproducibly — to how a man sleeps, eats, trains, carries fat, and manages stress. The clinical task is to identify which levers have real evidence behind them, apply them in the right order, and measure the result rather than assume it.

This playbook synthesises the human evidence on testosterone optimisation into an executable protocol. Its central conclusion is deliberately unglamorous: **the interventions with the strongest evidence are the least commercial ones.** Fat loss, sleep restoration, resistance training, alcohol reduction, and correction of genuine micronutrient deficiencies move the needle in randomized and meta-analytic data. Most of the supplement aisle does not.

What the evidence clearly supports

- **Losing excess adipose tissue.** Roughly a **10% reduction in body weight** produces a measurable improvement in total testosterone, with the largest gains in men who begin with the greatest adiposity. Bariatric surgery produces the largest pooled effect — an increase of approximately **251 ng/dL** in meta-analysis — though that figure is confounded by large-scale weight loss and rising SHBG.
- **Protecting sleep duration.** In a controlled laboratory study, **one week of 5-hour sleep restriction reduced daytime testosterone by 10–15%** in healthy young men — a larger effect than most supplements on the market can claim in the opposite direction.
- **Correcting deficiency, not supplementing abundance.** Vitamin D at **3,332 IU/day** raised testosterone in a randomized trial of deficient men; zinc repletion restored testosterone in marginally deficient men; magnesium at **450 mg/day** raised testosterone by roughly **24%** over 4 weeks in one small trial. In replete men, the same compounds generally do nothing.
- **Resistance training and higher-intensity conditioning**, particularly when they produce fat loss and improved insulin sensitivity.
- **Screening for and treating obstructive sleep apnea**, which is independently associated with low testosterone and is frequently missed in men with central adiposity.
- **Reducing alcohol intake** — a 2024 meta-analysis supports a dose-dependent inverse relationship between alcohol consumption and total testosterone.

What is oversold

- **Saw palmetto** inhibits 5-alpha-reductase but has not been shown to meaningfully raise testosterone.
- **Vitamin K2** has rat data only; there is no credible human testosterone evidence.
- **DHEA** shows a modest signal principally in women and older adults, typically above a **50 mg/day** threshold — not in young eugonadal men.
- **Fenugreek** was graded as *very low certainty* evidence in a 2026 meta-analysis in *Frontiers in Nutrition*.
- **Sauna and cold exposure** produce interesting cardiovascular and subjective effects, but the testosterone literature is essentially neutral or minimal.

10–15%
Fall in daytime testosterone after one week of 5-hour sleep in healthy young men

~251 ng/dL
Pooled testosterone increase after bariatric surgery in meta-analysis

2 tests
Morning fasting total testosterone measurements required to confirm deficiency

THE CLINICAL BOTTOM LINE

A man with symptoms and a genuinely low, twice-confirmed morning testosterone deserves a structured 90-day trial of sleep, fat loss, resistance training, alcohol reduction and deficiency correction *before* pharmacotherapy is considered — and deserves an honest conversation about what that trial can and cannot achieve. Lifestyle change typically produces modest shifts, not transformations. When it fails, the conversation moves to formal endocrinology, not to a stronger supplement.

How to use this document

Sections 2 and 3 establish the physiology and the diagnostic standard — read them before acting on anything else, because acting on an unconfirmed or poorly sampled result is the most common error in this field. Sections 4 through 11 are the intervention modules, each carrying an evidence tier. Section 12 sequences them into a 90-day protocol. Sections 13 and 14 define the boundary between self-directed optimisation and medical care, and how to measure whether any of it worked.

SECTION TWO

Understanding Testosterone: Physiology & Decline

Every intervention in this playbook works by touching one of three nodes: the hypothalamus and pituitary that issue the signal, the Leydig cells that manufacture the hormone, or the binding proteins and adipose tissue that determine how much of it is available and how much is converted away.

The hypothalamic–pituitary–gonadal (HPG) axis

Testosterone production is a three-tier endocrine cascade with negative feedback at every level.

1. **Hypothalamus.** GnRH-secreting neurons release gonadotropin-releasing hormone in pulses into the hypophyseal portal circulation. Pulse frequency and amplitude are the language of this system — continuous rather than pulsatile signalling desensitises the pituitary, which is precisely why long-acting GnRH agonists suppress testosterone rather than stimulate it.
2. **Anterior pituitary.** GnRH drives gonadotrophs to secrete **luteinising hormone (LH)** and **follicle-stimulating hormone (FSH)**.
3. **Testis.** LH binds receptors on **Leydig cells** in the interstitial compartment, stimulating cholesterol uptake and transport into the mitochondrion and driving steroidogenesis through the StAR protein, CYP11A1 (cholesterol side-chain cleavage), 3β-HSD and 17β-HSD enzymes. FSH acts on Sertoli cells to support spermatogenesis.

Feedback closes the loop. Testosterone itself, and more potently its aromatised metabolite **estradiol**, suppress hypothalamic GnRH and pituitary LH release. This matters clinically: anything that increases aromatase activity — most importantly a large adipose mass — increases estradiol, which in turn dampens the LH signal driving testicular production.

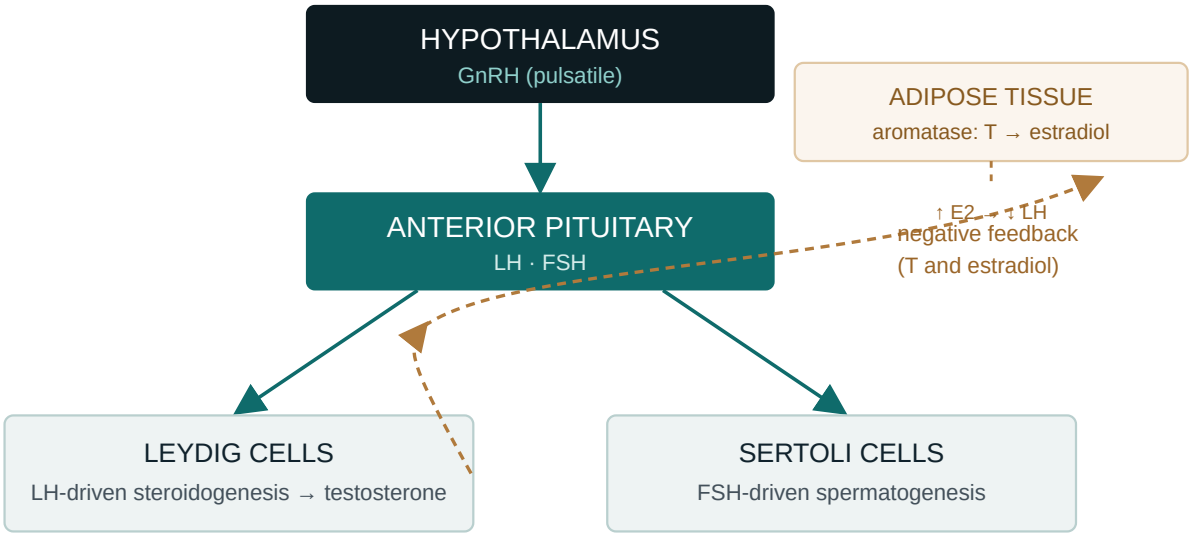


Figure 1. The HPG axis and its feedback loop. Adipose-derived aromatase is the principal reason obesity suppresses the axis: it converts testosterone to estradiol, and estradiol is a potent suppressor of GnRH and LH output.

Diurnal rhythm and why sampling time matters

Testosterone is secreted in pulses superimposed on a circadian envelope. In young, healthy, normally sleeping men, concentrations peak in the early morning — typically between roughly 06:00 and 10:00 — and decline across the day to a nadir in the evening. The amplitude of this rhythm is blunted by age, obesity, sleep deprivation, shift work, depression and chronic illness.

The clinical consequence is direct: **a testosterone drawn at 4 pm in a sleep-deprived man is not a fair measurement.** A borderline afternoon value should be repeated in the morning before any diagnostic conclusion is drawn. The Endocrine Society's guidance is built around morning, fasting sampling for exactly this reason (see Section 3).

Free, bioavailable and bound fractions

TABLE 1. FRACTIONS OF CIRCULATING TESTOSTERONE

FRACTION	APPROX. SHARE	CLINICAL SIGNIFICANCE
SHBG-bound	Majority (~60%)	Tightly bound, not available to tissue. SHBG rises with age, liver disease, thyroid excess, low insulin, and certain drugs; it falls with obesity, insulin resistance, and nephrotic-range protein loss.
Albumin-bound	Large minority (~38%)	Loosely bound; dissociates in capillaries and is considered bioavailable.
Free	Small minority (~2%)	Unbound and immediately active. Considered the most physiologically representative measure when SHBG is abnormal.

Age-related decline: real, but frequently overstated

Population studies consistently show lower average testosterone in older men, but the magnitude depends heavily on study design. Cross-sectional comparisons tend to overstate the effect because older cohorts carry a higher burden of obesity, comorbidity, medication use (opioids, glucocorticoids), sleep disturbance and alcohol exposure — each of which independently suppresses the axis. Longitudinal cohorts that follow the same men over time generally show a slower, more gradual decline than the cross-sectional picture implies.

The practical implication is important and frequently missed: **much of what looks like "age-related" decline is in fact "comorbidity-related" decline**, and is therefore at least partly modifiable. This is the therapeutic premise of the entire playbook.

Symptoms of low testosterone

No symptom is specific. Low testosterone is a syndrome diagnosed on the combination of consistent symptoms, signs on examination, and biochemical confirmation.

TABLE 2. SYMPTOM DOMAINS IN TESTOSTERONE DEFICIENCY

DOMAIN	TYPICAL FEATURES	COMMON MIMICS
Sexual	Reduced libido, weaker or less frequent erections, reduced morning/nocturnal erections, reduced ejaculate volume	Depression, relationship stress, vascular disease, SSRIs, diabetes
Physical	Fatigue, reduced muscle mass and strength despite training, increased central fat, reduced bone density, hot flushes	Sleep apnea, anaemia, hypothyroidism, deconditioning, overtraining
Psychological	Low mood, reduced motivation and drive, irritability, poor concentration, reduced self-confidence	Major depression, burnout, chronic stress, substance use

> **DIAGNOSTIC CAUTION**
>
> Fatigue plus a low-normal testosterone is not a diagnosis. Obstructive sleep apnea, depression, iron deficiency, hypothyroidism, type 2 diabetes and simply being sedentary all produce the same symptom cluster. Treating the number without investigating the cause is the most reliable way to produce a disappointed patient.

SECTION THREE

Diagnostic Standards

Testosterone is one of the most commonly mis-measured hormones in primary care. The errors are not exotic — wrong time of day, non-fasting sample, single measurement, non-standardised assay, and free testosterone estimated by an unreliable direct immunoassay. Each one is avoidable.

The 2026 Endocrine Society framework

Contemporary Endocrine Society guidance rests on a principle that has been stable across guideline iterations and was reaffirmed in the 2026 position statement on testosterone therapy: **diagnosis requires both consistent clinical findings and unequivocally low biochemical values.** Neither element alone is sufficient. The 2026 statement also emphasises assay standardisation, individualised cardiovascular and haematologic risk assessment, and explicit fertility counselling before any therapy is started.

Morning fasting testing protocol

- **Timing.** Draw between approximately 07:00 and 10:00, ideally within 2–3 hours of the patient's usual wake time.
- **Fasting.** An overnight fast avoids confounding from postprandial glucose, insulin and lipid changes, and permits simultaneous metabolic profiling.
- **Sleep and shift status.** Document recent sleep duration and any night-shift work. A man who has slept five hours for a week is temporarily hypogonadal by physiology, not by disease.
- **Avoid acute confounders.** Defer testing during acute illness, after a night of heavy drinking, during a period of extreme training load, and after recent initiation of opioids or glucocorticoids.
- **Two tests.** Abnormal values must be confirmed on a **separate morning** — not the same sample run twice.

CDC HoST-certified assays

The **CDC Hormone Standardization (HoST) Program** certifies testosterone assays against a reference measurement procedure, calibrating results across laboratories and platforms. The practical guidance is straightforward: request a **HoST-certified assay** wherever available, and prefer **liquid chromatography–tandem mass spectrometry (LC-MS/MS)** for total testosterone, particularly in the low range where direct immunoassays lose accuracy. When results are borderline, assay methodology is a legitimate reason to repeat testing at a reference laboratory rather than initiate treatment.

Free versus total testosterone

Total testosterone is the first-line measurement. Free testosterone is not a routine screen — it is a second-line test used when the total value may misrepresent the biologically available fraction.

- **Measure free testosterone** when SHBG is likely to be abnormal: obesity, type 2 diabetes, older age, chronic liver disease, hyper- or hypothyroidism, HIV, and use of glucocorticoids, anticonvulsants or opioids.
- **Prefer equilibrium dialysis** or a validated calculated free testosterone (using total T, SHBG and albumin) over direct free-testosterone immunoassays, which are notoriously unreliable at low concentrations.
- **Interpret in context.** In a man with severe insulin resistance, total testosterone may be low purely because SHBG is suppressed, while free testosterone remains adequate. Treating that number with exogenous hormone is a category error.

TABLE 3. BASELINE DIAGNOSTIC PANEL

TEST	TIMING / SPECIMEN	PURPOSE
Total testosterone (HoST/LC-MS/MS)	Fasting, 07:00–10:00, ×2 on separate days	Primary diagnostic measurement
SHBG	Same draw	Interprets total T; enables calculated free T
Free or bioavailable T	Same draw	Second-line when SHBG is abnormal
LH and FSH	Same draw	Distinguishes primary (testicular) from secondary (central) hypogonadism
Prolactin	Same draw	Excludes prolactinoma and drug-induced hyperprolactinaemia
Estradiol (sensitive assay)	Same draw	Relevant in obesity and before aromatase inhibition
HbA1c, fasting lipids, CMP	Same draw	Metabolic context; baseline before any therapy
25-hydroxyvitamin D, ferritin, TSH	Same draw	Reversible contributors to fatigue and to androgen status
Full blood count	Same draw	Baseline haematocrit before considering TRT
PSA	Per age and risk	Baseline prostate assessment where indicated

> **INTERPRETING A LOW RESULT**
>
> A single low morning value in a symptomatic man establishes a hypothesis, not a diagnosis. The sequence is: repeat on a second morning → exclude acute and reversible causes (sleep debt, alcohol, opioid or glucocorticoid exposure, uncontrolled diabetes, OSA) → if still low, characterise with LH/FSH and prolactin → then, and only then, discuss therapy. Men with low T and *elevated* LH have primary testicular failure; men with low T and low or inappropriately normal LH have secondary hypogonadism, which warrants pituitary evaluation.

SECTION FOUR

The Obesity–Testosterone Connection

The relationship between adiposity and testosterone is bidirectional and self-reinforcing. Low testosterone promotes fat accumulation; fat accumulation suppresses testosterone. Breaking that loop is the single highest-yield intervention available.

Aromatase activity in adipose tissue

Adipose tissue expresses **aromatase (CYP19A1)**, the enzyme that converts testosterone to estradiol. More fat means more aromatase capacity, more estradiol, and therefore stronger negative feedback on the hypothalamus and pituitary — which reduces LH drive to the Leydig cells and lowers testicular production. Visceral adipose tissue is particularly metabolically active in this respect, which is why central obesity correlates more strongly with low testosterone than total body weight alone.

Adipose tissue is also an endocrine organ in its own right, secreting leptin, interleukin-6 and TNF-alpha. Chronic low-grade inflammation and leptin resistance impair hypothalamic GnRH pulsatility independently of the aromatase pathway. The result is a genuine functional secondary hypogonadism that is, in principle, reversible.

Insulin resistance and SHBG

Hyperinsulinaemia suppresses hepatic SHBG production. Because roughly 60% of circulating testosterone is SHBG-bound, falling SHBG lowers *total* testosterone — while free testosterone may be relatively preserved. This produces a characteristic and frequently mishandled pattern: an obese, insulin-resistant man with a low total testosterone and a normal calculated free testosterone.

Two consequences follow. First, **measure SHBG and free testosterone before diagnosing deficiency in an obese man.** Second, treating that patient with exogenous testosterone addresses nothing — the reversible problem is insulin resistance, and the treatment is weight loss, resistance training and glycaemic improvement.

Weight loss interventions

Lifestyle-induced weight loss

Dietary and behavioural weight loss produces a consistent, dose-dependent increase in total testosterone. A widely cited benchmark is that **approximately 10% weight loss measurably improves testosterone**, with greater absolute gains in men who start with higher BMI. The effect is mediated by reduced aromatase capacity, improved insulin sensitivity and rising SHBG. It is a moderate effect — meaningful at population level and clinically relevant for men near the diagnostic threshold, but rarely sufficient to convert a frankly hypogonadal man into a robustly eugonadal one.

Bariatric surgery

Bariatric surgery produces the largest and most rapid testosterone changes of any intervention. A 2025 meta-analysis published in *Andrology* (Pozzi et al.) reported a pooled increase of approximately **251 ng/dL** following bariatric surgery. This is an impressive figure, but it must be read with its confounders intact:

- **SHBG rises** after major weight loss, inflating total testosterone independently of free testosterone.
- Follow-up in most studies is relatively short, and durability is uncertain.
- Surgical populations are highly selected and not generalisable to men with moderate overweight.
- Rapid weight loss without resistance training and adequate protein risks substantial lean mass loss, which is itself metabolically unfavourable.

GLP-1 receptor agonists

GLP-1-based pharmacotherapy produces substantial weight loss and, in observational and secondary analyses, corresponding improvements in total testosterone. The critical interpretive question — whether these agents have any *direct* gonadal or central effect beyond weight loss — has not been established. The most defensible position is that the testosterone benefit is largely weight-mediated and should be expected to track the magnitude of fat loss.

Two practical cautions apply. First, a meaningful proportion of GLP-1-induced weight loss can be lean mass; preserving it requires a protein intake at the upper end of the range in Section 5 together with structured resistance training. Second, weight should be stabilised before testosterone is re-measured, because values obtained during active rapid loss are difficult to interpret.

TABLE 4. WEIGHT-LOSS MODALITIES AND TESTOSTERONE EFFECT

INTERVENTION	TYPICAL EFFECT ON TOTAL T	EVIDENCE TIER	CAVEATS
Lifestyle weight loss (~10%)	Moderate increase	STRONG	Dose-dependent; greatest gain in the most obese; requires maintenance to persist.
Bariatric surgery	Large (~251 ng/dL pooled)	STRONG	SHBG rise inflates total T; short follow-up; selected population.
GLP-1 receptor agonists	Moderate, weight-mediated	MODERATE	Direct gonadal effect unproven; lean-mass preservation required.
Testosterone therapy in obese men	Raises T but does not treat the cause	WEAK as obesity strategy	May increase haematocrit; does not address insulin resistance.

PRACTICAL SEQUENCE

Set a first target of **5–10% body weight loss**, prioritising visceral fat and waist circumference over scale weight. Pair it with resistance training so that the loss is fat-selective. Re-measure testosterone only after weight has been stable for several weeks, and interpret the result alongside SHBG and calculated free testosterone.

SECTION FIVE

Nutrition Protocol

No food raises testosterone dramatically. But dietary pattern determines body composition, insulin sensitivity and micronutrient status — the three physiological inputs that matter most. The goal is a diet that a man will still be following in twelve months.

Mediterranean dietary pattern

The Mediterranean pattern — abundant vegetables, legumes, whole grains, nuts, olive oil as the primary fat, fish and moderate dairy, with limited red and processed meat — is the best-supported dietary framework for metabolic and androgen health. Observational cohorts consistently associate higher adherence with more favourable testosterone concentrations and metabolic profiles, and randomised trials consistently demonstrate improvements in insulin sensitivity, inflammatory markers and cardiovascular risk.

An honest caveat: **randomised trials using testosterone as the primary endpoint are scarce.** The strongest claim the evidence supports is that the Mediterranean pattern improves the metabolic substrate on which testosterone depends — adiposity, insulin resistance, inflammation — rather than raising testosterone through a direct dietary mechanism.

Macronutrient balance

TABLE 5. PRACTICAL MACRONUTRIENT TARGETS

MACRONUTRIENT	TARGET	RATIONALE AND IMPLEMENTATION
Protein	**1.2–1.6 g/kg/day**	Preserves lean mass during weight loss, supports resistance-training adaptation, and improves satiety. Distribute across meals rather than concentrating at dinner. In a 90 kg man this is roughly 110–145 g/day.
Fat	~25–35% of energy; not below ~20%	Cholesterol is the substrate for steroidogenesis, and very low-fat diets have been associated with modest reductions in total testosterone in dietary intervention studies. Emphasise olive oil, nuts, avocado, oily fish and egg yolks.
Carbohydrate	Moderate, remainder of energy	Favour fibre-rich, low-glycaemic sources. Quality matters more than quantity: the objective is glycaemic control, not carbohydrate avoidance.
Fibre	30 g+/day	Improves insulin sensitivity, feeds the microbiome, and supports satiety and lipid control.

Foods and substances to limit

- **Ultra-processed foods.** High consumption is associated with higher central adiposity, worse insulin sensitivity and lower testosterone in observational data. The mechanism is probably indirect — through weight gain and metabolic dysfunction — but the direction of association is consistent enough to justify restriction.
- **Soy in excess.** This deserves a calibrated statement. At typical dietary intakes, the evidence in men is largely reassuring, with isoflavones showing minimal effect on testosterone in most trials. The caveat applies to *very high-dose supplemental isoflavone concentrates*, not to tofu or edamame. Blanket soy avoidance is not evidence-based; megadosed isoflavone supplements are not a sensible routine.
- **Alcohol.** Covered in detail in Section 11. In brief: dose-dependent suppression of testosterone, disruption of sleep architecture, and increased aromatase activity via weight gain.
- **Sugar-sweetened beverages.** Primarily an insulin-resistance and visceral-fat issue rather than a direct androgen one.

Meal timing and fasting

Fasting is heavily marketed as a testosterone strategy. The evidence does not support that framing.

- **Intermittent fasting does not reliably raise testosterone.** Some studies report modest increases accompanying fat loss; others report acute suppression during prolonged fasting, particularly when energy availability is low.
- **Prolonged or severe restriction lowers testosterone** through the same low-energy-availability mechanism seen in endurance athletes — suppressed GnRH pulsatility is an adaptive response to an energy deficit, not a pathology to be treated with more discipline.
- **Time-restricted eating is defensible** as an adherence tool for reducing total intake, not as an androgen intervention. If it improves adherence to a calorie deficit, it helps indirectly.
- **Protein distribution matters more than meal frequency.** Three to four protein-containing meals, each with a meaningful leucine dose, is a reasonable structure.

> **THE DEFICIENCY PRINCIPLE**
>
> The micronutrients with genuine testosterone data — vitamin D, zinc and magnesium — produce their effects predominantly in men who are deficient or marginally depleted. In a replete man with an adequate diet, adding them is not a testosterone strategy. Test first; supplement second. Section 9 grades each one.

SECTION SIX

Exercise Protocol

Exercise is the most reliable non-pharmacological androgen intervention available — but not for the reason most men assume. The chronic benefit comes from body composition and insulin sensitivity, not from the transient post-set spike.

Resistance training

Resistance training is the backbone of the protocol. Systematic reviews of resistance training in older and overweight men report modest improvements in total testosterone, with the most consistent benefits in men who are untrained at baseline and who lose fat during the intervention. Strength, lean mass and functional capacity improve far more reliably than the hormone value itself — and those outcomes matter independently.

Programming principles

- **Compound movements first.** Squat, hinge, press, pull and carry patterns recruit the largest muscle mass and produce the greatest systemic training stimulus.
- **Progressive overload.** Add load, reps or sets over time. Without progression, adaptation plateaus within weeks.
- **Frequency and volume.** Three to four sessions per week, training each major muscle group roughly twice weekly, is a defensible default for a general population.
- **Intensity.** Predominantly moderate-to-heavy loads in the 6–12 repetition range, with most sets taken to 1–3 repetitions of reserve rather than absolute failure.
- **Recovery.** 48 hours before re-training the same muscle group; a deload week every 4–6 weeks.

High-intensity interval training

HIIT and sprint-interval work improve cardiorespiratory fitness, insulin sensitivity and body composition with a favourable time cost. Several small trials report improvements in total testosterone and, more consistently, in the **testosterone-to-cortisol ratio** — a marker of anabolic-to-catabolic balance that is best interpreted as a training-stress indicator rather than a clinical endpoint. A trial by Velasco-Orjuela and colleagues is representative of this literature: improvements in the hormonal ratio alongside fitness gains in a modest-sized sample.

The realistic claim is this: HIIT improves the metabolic environment in which testosterone is regulated, and it does so efficiently. It is not a direct pharmacological stimulus.

Acute versus chronic effects

This distinction is essential and routinely conflated in popular writing.

- **Acute.** A bout of heavy resistance exercise or sprint work produces a transient rise in total testosterone lasting tens of minutes. This is a normal physiological response to stress and substrate mobilisation. It does **not** predict long-term hormonal adaptation.
- **Chronic.** The sustained change in resting testosterone comes from accumulated changes in fat mass, insulin sensitivity, sleep quality and training status. These changes are real but modest, and they develop over months.

Avoiding overtraining and the endurance suppression problem

There is a well-described phenomenon — sometimes called the **exercise-hypogonadal male condition** — in which high-volume endurance training combined with inadequate energy intake suppresses the HPG axis. Low energy availability, whether from intentional restriction or from energy expenditure exceeding intake, reduces GnRH pulsatility and lowers LH and testosterone. This is an adaptive energy-conservation response, not a sign of virtue.

Warning signs include persistently low libido, loss of morning erections, unexplained performance decline, disrupted sleep, resting heart rate rising while heart rate variability falls, and recurrent minor illness. Practical safeguards:

- Match energy intake to training load; do not combine heavy training with aggressive calorie restriction for extended periods.
- Keep at least two non-negotiable recovery days per week during high-load phases.
- Track a simple readiness marker — resting heart rate, session RPE, or HRV if available — and deload when it trends unfavourably for a week or more.
- Prioritise sleep over adding training volume.

TABLE 6. EXERCISE MODALITIES AND THEIR EVIDENCE

MODALITY	PRESCRIPTION	EVIDENCE TIER	REALISTIC EFFECT
Resistance training	3–4×/week, compound lifts, progressive overload	STRONG for body composition; MODERATE for T	Modest rise in total T, largely mediated by fat loss; large gains in strength and lean mass.
HIIT / sprint intervals	2–3×/week, 4–10 × 30–60 s efforts	MODERATE	Improved T/cortisol ratio and fitness; direct T effect small.
Moderate aerobic base	150+ min/week zone 2	MODERATE (indirect)	Improves insulin sensitivity and weight maintenance; minimal direct androgen effect.
Excessive endurance volume with low energy availability	—	HARMFUL	Suppresses LH and testosterone; loss of libido and performance.

SECTION SEVEN

Sleep Optimization

If a supplement produced a 10–15% change in testosterone with a single week of use, it would be front-page news and sold out everywhere. Sleep restriction does exactly that in the opposite direction — and it is treated as a lifestyle preference rather than a medical exposure.

Duration: 7–9 hours

Adult guidance converges on **7–9 hours of nightly sleep**, with individual variation around that range. The relevant question is not merely hours in bed but sleep obtained, consolidated, and timed consistently. Men who habitually sleep under six hours, or whose sleep is fragmented by apnea or nocturia, sit in a hormonally disadvantageous state regardless of what else they do correctly.

The evidence: one week of 5-hour sleep

> **LANDMARK FINDING**
>
> In a controlled laboratory study by Leproult and Van Cauter published in *JAMA*, healthy young men restricted to **5 hours of sleep per night for one week** showed a **10–15% reduction in daytime testosterone concentrations**, accompanied by reduced vigour, increased fatigue, and measurable effects on mood and alertness. Recovery of sleep restored the hormonal profile. *Source:* [Leproult & Van Cauter, JAMA 2011] (https://pubmed.ncbi.nlm.nih.gov/?term=Leproult+Van+Cauter+sleep+restriction+testosterone).

Two features of this finding deserve emphasis. First, the magnitude is comparable to or larger than the effect sizes reported for most over-the-counter "testosterone boosters," but in the opposite direction — meaning a supplement taken on a five-hour sleep schedule is likely swimming against a larger current. Second, the effect was measurable within a single week, making sleep one of the fastest-acting levers available.

REM sleep and testosterone secretion

Testosterone is not secreted uniformly across the night. Nocturnal secretion is concentrated in sleep, with the largest and most consistent pulses occurring in association with sleep onset and the deeper, more consolidated portions of the sleep cycle. Experimental work on selective sleep-stage deprivation indicates that suppression of REM sleep blunts the normal morning rise in testosterone, independent of total sleep time.

The practical translation: **sleep continuity and architecture matter, not only duration.** Eight fragmented hours interrupted by alcohol, apnea or a partner's alarm do not deliver the same endocrine signal as seven consolidated hours. This is one of the strongest arguments for treating alcohol as a sleep disruptor even when it appears to aid sleep onset.

Obstructive sleep apnea: screen every central adiposity patient

OSA and low testosterone are tightly and bidirectionally linked. Central adiposity narrows the pharyngeal airway; fragmented sleep and repeated hypoxaemia suppress the HPG axis; low testosterone in turn promotes further visceral fat accumulation. The result is a loop that neither CPAP nor testosterone alone fully resolves.

A meta-analysis of CPAP effects on testosterone (PMC6712440) reports a **modest but real increase in total testosterone** with CPAP treatment, concentrated in men with adequate adherence and more severe baseline disease. CPAP is not a testosterone therapy — but untreated OSA is an absolute obstacle to interpreting, let alone treating, a low testosterone result.

Screening. Use a validated tool such as STOP-BANG, supplemented by the Epworth Sleepiness Scale, in any man with snoring, witnessed apnoeas, morning headache, unrefreshing sleep, nocturia, treatment-resistant hypertension, or a neck circumference and BMI that raise suspicion. Refer for polysomnography or home sleep testing when the screen is positive.

TABLE 7. SLEEP TARGETS AND INTERVENTIONS

PARAMETER	TARGET	EVIDENCE TIER	NOTES
Total sleep duration	7–9 hours	STRONG	5-hour restriction lowers daytime T by 10–15% within a week.
Consistency	Fixed wake time ±30 min	MODERATE	Stabilises circadian timing of the morning T peak.
OSA screening and treatment	Screen all at-risk men; CPAP if diagnosed	MODERATE	Modest total T increase with adherent CPAP.
Alcohol before bed	Avoid within 3 hours of sleep	STRONG	Suppresses REM and fragments the second half of the night.
Sleep hygiene (light, temperature, screens)	Dark, cool (~18–19 °C), no late bright light	WEAK for T directly	Improves sleep quality and duration, which is the actual mechanism.

SECTION EIGHT

Stress Management

The hypothalamic–pituitary–adrenal and hypothalamic–pituitary–gonadal axes share a common upstream controller. When one is driven hard, the other is suppressed — which is why chronic stress shows up in the clinic as low libido long before it shows up as a low lab value.

The cortisol–testosterone relationship

The interaction operates at several levels. Corticotropin-releasing hormone (CRH) directly suppresses GnRH release. Glucocorticoids reduce pituitary responsiveness to GnRH and inhibit testicular steroidogenesis. Chronically elevated cortisol also promotes visceral fat deposition — which, via aromatase, adds a second suppressive pathway. The relationship is best described as **inverse and physiologically coupled** rather than as a simple seesaw.

What the human evidence supports is association more than causation: chronic psychological stress, burnout and depression are consistently associated with lower testosterone. Interventional trials of stress-reduction techniques on testosterone as a primary endpoint are small, short and heterogeneous, and the effect sizes are modest. The honest summary is **moderate plausibility, weak direct trial evidence** — but with substantial independent benefits for sleep, adherence and mental health.

Mindfulness, meditation and breathing

- **Mindfulness-based programmes.** Small randomised trials report reductions in perceived stress and cortisol, with occasional modest improvements in testosterone or the testosterone-to-cortisol ratio. Sample sizes are small and effects inconsistent across studies.
- **Slow-paced breathing.** Breathing at roughly six breaths per minute increases heart rate variability and shifts autonomic balance toward parasympathetic dominance. This is well established for HRV; the downstream testosterone effect is not established.

- **Psychological treatment for depression.** Underrated in this context. Depression independently suppresses libido and testosterone, and effective treatment often improves both — sometimes more than any supplement in this document.

A pragmatic prescription: **10–20 minutes daily** of a structured practice the patient will actually sustain — mindfulness, slow breathing, a walk without a phone, or deliberate down-regulation before sleep. The specific technique matters less than the consistency and the effect on sleep quality, which is where much of the hormonal benefit is ultimately mediated.

TABLE 8. STRESS INTERVENTIONS AND EVIDENCE

INTERVENTION	DOSE	EVIDENCE TIER	EFFECT
Mindfulness / meditation	10–20 min daily	WEAK for T; MODERATE for stress	Reduced perceived stress and cortisol; small, inconsistent T effects.
Slow breathing (≈6 breaths/min)	5–10 min, 1–2×/day	WEAK	Improved HRV; T effect unproven.
Treatment of clinical depression	Per clinical guidance	MODERATE (indirect)	Improves libido and often androgen status via restored sleep and activity.
Adequate energy intake during training	Match intake to load	STRONG	Prevents low-energy-availability suppression of the HPG axis.

SECTION NINE

Supplement Protocol (Evidence-Graded)

This is where the gap between marketing and evidence is widest. A small number of compounds have genuine human randomised data — mostly in deficient populations. A larger number have intriguing pilot studies. The rest have nothing but animal models and confident packaging.

TABLE 9. EVIDENCE-GRADED SUPPLEMENT SUMMARY

AGENT	DOSE STUDIED	EVIDENCE TIER	KEY FINDING
Vitamin D	3,332 IU/day	STRONG (if deficient)	Randomised trial in deficient men showed a rise in total testosterone; benefit largely confined to the deficient.
Zinc	220 mg zinc sulfate (≈50 mg elemental)	MODERATE – STRONG in deficiency	Restored testosterone in marginally zinc-deficient men; no meaningful effect in replete men.
Magnesium	450 mg/day, 4 weeks	MODERATE	Raised testosterone by approximately 24% in one small trial; short duration, limited replication.
Ashwagandha (*Withania somnifera*)	600 mg/day, 8–12 weeks	MODERATE	14.7% increase in testosterone in one RCT; associated cortisol reductions; small samples, some industry funding.
Tongkat Ali (*Eurycoma longifolia*)	200–600 mg/day extract	MODERATE (limited)	Systematic review found significant increases in total testosterone, most consistent in men with low or hypogonadal baseline values.
Boron	6 mg/day	WEAK – MODERATE	Short-term studies show reduced SHBG and increased free testosterone; small, brief, mixed results.
Fenugreek	Variable extracts	WEAK	2026 meta-analysis in *Frontiers in Nutrition* concluded *very low certainty* of evidence.
DHEA	≥50 mg/day	WEAK (except specific groups)	Modest effects mainly in women and older adults; not supported in young eugonadal men.
Saw palmetto	320 mg/day standardised	INSUFFICIENT	Inhibits 5-alpha-reductase (DHT) but has not shown a meaningful testosterone increase.
Vitamin K2	—	INSUFFICIENT	Rat and animal data only; no credible human testosterone outcomes.
Omega-3 fatty acids	1–3 g/day EPA+DHA	INSUFFICIENT	Mixed results; cardiovascular and anti-inflammatory benefits independent of testosterone.
Iodine	—	INSUFFICIENT	Observational NHANES association only; no interventional testosterone data.

Strong evidence

Vitamin D

The strongest human data concern repletion of deficiency. A randomised controlled trial by Pilz and colleagues reported that **3,332 IU of vitamin D per day** increased total testosterone over the intervention period in men with low baseline 25-hydroxyvitamin D. Subsequent trials (including work by Lerchbaum and colleagues) broadly support a benefit concentrated in deficient men, with little or no effect in those already replete. Practical approach: measure 25-hydroxyvitamin D; if low, replete to a normal range; if normal, do not supplement *for testosterone*.

Zinc

Prasad's classic work demonstrated that **220 mg of zinc sulfate (approximately 50 mg of elemental zinc) daily** restored serum testosterone in men with mild zinc deficiency. The Santos review of zinc and testosterone reinforces the same conclusion: benefit in deficiency, no clear benefit in sufficiency. An important safety point — chronic intake above the tolerable upper limit (around 40 mg elemental zinc per day) risks **copper deficiency**, with anaemia and neuropathy. Zinc is not a supplement to take indefinitely at high dose without indication.

Magnesium

A small trial by Cinar and colleagues reported that **450 mg/day of magnesium for four weeks** raised testosterone by roughly **24%**, alongside improvements in some performance measures. Related work on zinc–magnesium combinations (Brilla and Conte) reported changes in anabolic hormone markers in resistance-trained men. The caveats are important: very small samples, short duration, and populations that were training or potentially marginally deficient. Magnesium is low-risk and reasonable to trial, particularly in men with low dietary intake, but the 24% figure should not be generalised as a reliable expectation.

Moderate or mixed evidence

Ashwagandha

Two frequently cited trials anchor this evidence. Chandrasekhar and colleagues reported a **14.7% increase in testosterone** with **600 mg/day** of a standardised root extract over 8 weeks in stressed, overweight men, alongside reductions in cortisol. Lopresti and colleagues reported improvements in DHEA-S, cortisol and perceived stress with 600 mg/day of KSM-66 over 8 weeks. Limitations: small samples, short duration, some industry involvement, and modest effect sizes. Safety notes: documented cases of **hepatotoxicity** exist, and ashwagandha can raise thyroid hormone levels — use with caution in liver disease and thyroid disorders.

Tongkat Ali (*Eurycoma longifolia*)

A systematic review (PMC9415500) found that tongkat ali supplementation was associated with significant increases in total testosterone, with the most consistent effects in men who had low or hypogonadal baseline values, and less consistent effects in eugonadal men. This is a familiar pattern across the supplement literature: the lower the baseline, the larger the apparent effect — which also means regression to the mean is a plausible partial explanation.

Boron

Naghii's short-term studies reported that **6 mg/day of boron** reduced SHBG and increased free testosterone over a one-week period, with some effects on estradiol. Examine.com's summary appropriately characterises the literature as promising but small and short. A one-week SHBG change is not the same as a durable improvement in androgen status.

Fenugreek

A 2026 meta-analysis in *Frontiers in Nutrition* graded the certainty of evidence for fenugreek and testosterone as **very low**. Some individual trials report small increases; the body of evidence is too inconsistent and too imprecise to support a recommendation.

DHEA

DHEA is a prohormone and its effects depend heavily on baseline age and sex. Meta-analytic work by Corona and colleagues, together with more recent synthesis (2025), indicates modest androgen effects concentrated in **women and older adults**, typically at doses of **50 mg/day or more**. In young eugonadal men, DHEA does not meaningfully raise testosterone and is not a rational choice. It is also a banned substance in most sporting contexts.

Weak or insufficient evidence — and what to avoid

- **Saw palmetto** (PMC7706486) inhibits 5-alpha-reductase and may modestly lower DHT, but has not been shown to raise testosterone. It is a prostate symptom remedy, not an androgen strategy.
- **Vitamin K2** has rat data (Ito 2011; MDPI 2026) suggesting effects on Leydig cell steroidogenesis. There is no human testosterone evidence. Animal findings of this kind frequently fail to translate.
- **Omega-3** has mixed and generally null testosterone data. It retains independent cardiovascular and anti-inflammatory justification, but not an androgen one.
- **Iodine** appears in observational NHANES analyses (PMC10733805) as a correlate of testosterone, almost certainly mediated by thyroid status. It is not an androgen intervention.
- **Proprietary "testosterone booster" blends** typically combine sub-therapeutic doses of several of the above with undisclosed fillers. They cannot be evaluated and should be avoided.
- **Tribulus terrestris** has repeatedly failed to raise testosterone in controlled trials in eugonadal men, despite widespread marketing.
- **SARMs and unregulated androgens** are not supplements. They suppress endogenous production, carry hepatotoxicity and cardiovascular risk, and are frequently mislabelled.

> **STACKING AND QUALITY DISCIPLINE**
>
> Introduce one agent at a time, for at least 8 weeks, so that effect and adverse effect can be attributed. Choose products with independent third-party testing (NSF Certified for Sport, USP Verified, or equivalent) and avoid proprietary blends that conceal doses. Disclose every supplement to the treating clinician — several interact with thyroid, liver and coagulation pathways, and DHEA in particular will distort any subsequent hormone panel.

SECTION TEN

Environmental Toxin Reduction

Endocrine-disrupting compounds are a legitimate concern with an honest limitation: the human interventional evidence is thin. What follows separates what is reasonably established from what is plausible but unproven — and offers only low-cost, low-risk actions.

Bisphenol A and phthalates

Phthalates are the more consistently concerning class. Several large cross-sectional analyses, including NHANES-based work, report inverse associations between urinary phthalate metabolites and total testosterone in adult men, with some of the strongest signals for DEHP metabolites. Mechanistic studies support Leydig cell toxicity and interference with steroidogenic enzymes.

Bisphenol A shows a less consistent picture. Some cohorts report inverse associations with testosterone; others find none, and study designs vary widely in exposure assessment. BPS and other "BPA-free" substitutes are not reliably safer — several show similar or unknown endocrine activity.

Reviews of endocrine disruptors and male reproduction (PMC4046332; PMC9890927) converge on a plausible causal contribution to declining sperm quality and disturbed androgen signalling, while acknowledging that human causality remains difficult to establish because exposure is ubiquitous and no unexposed comparator group exists.

Practical reduction strategies

These measures are inexpensive, have no meaningful downside, and reduce exposure measurably even where the health benefit is unproven.

- **Replace food-contact plastics** with glass or stainless steel, particularly for hot food, oily food and acidic food — heat and fat increase leaching.

- **Never microwave in plastic.** Transfer to glass or ceramic.
- **Avoid thermal receipt paper** (the shiny till receipts), which is a significant BPA/BPS source; wash hands after handling and do not use hand sanitiser before touching it, as this increases dermal absorption.
- **Choose fragrance-free personal care** where practical. "Fragrance" on a label is an umbrella term that can conceal phthalate carriers.
- **Reduce canned food consumption** where the lining is unknown, and prefer fresh or glass-jarred alternatives.
- **Wet-dust and ventilate.** Indoor dust is a major phthalate reservoir; wet mopping and HEPA vacuuming reduce it.
- **Wash hands before eating** — a simple and underrated exposure-reduction step.

> **HONEST FRAMING**
>
> No randomised trial demonstrates that reducing personal-care or packaging exposure raises testosterone. These recommendations rest on exposure-reduction plausibility and mechanistic evidence, not on demonstrated hormonal outcomes. They are worth doing because they are cheap and harmless — not because they will move a lab value.

SECTION ELEVEN

Alcohol, Caffeine & Lifestyle Factors

Three commonly asked questions — does alcohol lower testosterone, does caffeine raise it, and does sauna or cold exposure do anything? The answers are, respectively: yes, no, and essentially no.

Alcohol

A **2024 meta-analysis** supports a **dose-dependent inverse relationship between alcohol consumption and total testosterone**. Heavy chronic intake is unambiguously suppressive, acting through reduced LH pulsatility, direct testicular toxicity, hepatic dysfunction affecting SHBG and oestrogen metabolism, and increased visceral adiposity with consequent aromatase activity. Moderate intake shows a smaller but still detectable inverse association in pooled analyses.

Alcohol also damages testosterone indirectly in ways that are easy to overlook: it suppresses REM sleep, fragments the second half of the night, and adds calories that promote central adiposity. For a man with a borderline low testosterone, reducing alcohol is one of the highest-yield changes available — and it costs nothing.

Caffeine

Caffeine produces **acute, transient** effects in some studies, with small rises in testosterone reported in short-term protocols. There is no credible evidence of a chronic testosterone benefit from habitual coffee consumption. The more relevant interaction is with sleep: caffeine taken after early afternoon measurably reduces sleep duration and quality, and sleep loss lowers testosterone by 10–15% per week of restriction. The net effect of late caffeine on androgen status is therefore plausibly negative.

Sauna and cold exposure

Both are popular in the "hormone optimisation" space. The evidence does not support the claims.

- **Sauna.** Finnish studies examining repeated heat exposure report either no change or small, inconsistent increases in testosterone. Heat exposure above testicular tolerance is, if anything, adverse for spermatogenesis. Sauna is excellent for cardiovascular conditioning and relaxation; it is not a testosterone intervention.
- **Cold exposure.** Cold water immersion produces a sharp rise in noradrenaline and dopamine, which is why it feels meaningful. It has not been shown to raise chronic testosterone concentrations in controlled human data. Claims about "cold-induced androgen receptor upregulation" are extrapolations, not findings.

TABLE 10. LIFESTYLE FACTORS AND TESTOSTERONE

FACTOR	EFFECT	EVIDENCE TIER	GUIDANCE
Alcohol	Dose-dependent reduction	STRONG	Keep intake low; eliminate entirely if testosterone is borderline.
Caffeine	Acute transient rise; no chronic effect	WEAK	Not a strategy; keep intake before early afternoon to protect sleep.
Sauna	Neutral to minimal	INSUFFICIENT for T	Valuable for cardiovascular health and relaxation; no androgen claim.
Cold exposure	Minimal / unproven for T	INSUFFICIENT	Enjoyable and possibly mood-beneficial; not a hormonal lever.
Tobacco / vaping	Adverse	MODERATE	Smoke contains endocrine-disrupting compounds; cessation is indicated regardless.
Testicular heat (laptops, prolonged sitting, tight clothing)	Adverse for fertility	WEAK for T	Relevant mainly to spermatogenesis rather than serum testosterone.

SECTION TWELVE

90-Day Implementation Plan

Ninety days is the minimum realistic window in which lifestyle change can produce a measurable hormonal signal. It is also long enough to distinguish a genuine effect from the day-to-day noise of a single measurement.

Weeks 1–4: Foundation

The first month is about establishing the diagnostic baseline and removing the largest suppressive exposures. Nothing exotic happens here, and that is deliberate.

- **Testing.** Baseline panel per Table 3, with two morning fasting total testosterone measurements on separate days, plus SHBG, LH, FSH, prolactin, 25-hydroxyvitamin D, ferritin, HbA1c, lipids, full blood count and TSH.
- **Sleep.** Set a fixed wake time; target 7–9 hours; remove alcohol within three hours of bedtime; screen for OSA with STOP-BANG.

- **Nutrition.** Establish the protein target (1.2–1.6 g/kg), remove ultra-processed foods and sugar-sweetened beverages, and begin a modest calorie deficit if body weight is above target.
- **Training.** Three full-body resistance sessions per week with compound movements; daily step count of 8,000–10,000.
- **Deficiency correction.** Start vitamin D only if 25-hydroxyvitamin D is low; start zinc only if intake or status is inadequate.
- **Tracking.** Baseline waist circumference, body weight, blood pressure, libido score, sleep duration and a training log.

Weeks 5–8: Intensification

- **Training.** Move to four sessions per week or add a fourth lifting day; add two HIIT sessions of 4–10 intervals; apply progressive overload in writing, not by feel.
- **Body composition.** Target 5–10% weight loss by week 12; check waist circumference monthly rather than weighing daily.
- **Supplements.** Introduce *one* agent at a time — magnesium, then ashwagandha or tongkat ali if desired — at least four weeks apart, so effects are attributable.
- **Stress.** Establish a daily 10–20 minute practice; protect it as a scheduled appointment.
- **Sleep.** Address the sleep study result if OSA was diagnosed; initiate CPAP and troubleshoot mask adherence early, since the first two weeks determine long-term use.

Weeks 9–12: Optimisation and retesting

- **Consolidate.** No new interventions. The objective is adherence and consistency.
- **Retest** at the end of week 12 — at least 8 weeks after the last major change and after weight has been stable for several weeks — using the same assay, at the same time of morning, fasting.
- **Review.** Compare total testosterone, SHBG, calculated free testosterone, waist circumference, body weight, HbA1c and symptom scores against baseline.
- **Decide.** If testosterone has normalised and symptoms have resolved, maintain. If testosterone remains unequivocally low with persistent symptoms, proceed to Section 13.

TABLE 11. THE 90-DAY PROTOCOL AT A GLANCE

PHASE	FOCUS	KEY ACTIONS	MEASURABLE TARGET
Weeks 1–4	Foundation and diagnosis	Baseline labs ×2; sleep schedule; protein target; 3× resistance; deficiency correction	Baseline panel complete; sleep ≥7 h on 5+ nights/week
Weeks 5–8	Intensification	4× resistance + 2× HIIT; one supplement introduced; OSA treatment if indicated	Progressive load increase logged; 3–5% weight loss
Weeks 9–12	Optimisation and retest	Consolidate; retest at week 12; review and decide	5–10% weight loss; repeat morning testosterone

SETTING REALISTIC EXPECTATIONS

Across the interventions in this playbook, the typical effect on total testosterone is **modest** — meaningful for a man sitting near the diagnostic threshold, rarely enough to move a frankly hypogonadal man into the upper-normal range. The honest pitch is not "raise your testosterone dramatically." It is "remove the suppressors, correct the deficiencies, and see what your physiology does when you stop fighting it."

SECTION THIRTEEN

When to Seek Medical Intervention

Testosterone therapy is effective, legitimate, and over-prescribed in equal measure. The dividing line is a properly confirmed diagnosis, an honest risk conversation, and a clear answer to the fertility question before the first dose.

Criteria for testosterone therapy

Contemporary Endocrine Society guidance supports treatment in men with **consistent symptoms and signs** plus **unequivocally low morning fasting total testosterone confirmed on two separate occasions**, after reversible causes have been excluded. Three exclusions matter:

- **Eugonadal men** seeking body composition or athletic gains — not an indication.
- **Untreated severe OSA**, uncontrolled heart failure, elevated haematocrit, or recent myocardial infarction or stroke — address or defer first.
- **Men planning conception** — see fertility below; this is the single most important pre-treatment conversation.

The TRAVERSE trial and the modern risk picture

The TRAVERSE trial substantially updated the cardiovascular risk conversation. In men with hypogonadism and cardiovascular risk factors, testosterone gel was **non-inferior to placebo for major adverse cardiac events** — a genuinely reassuring result after years of uncertainty. However, the same trial identified **increased rates of atrial fibrillation, acute kidney injury, pulmonary embolism and fracture** in the testosterone arm.

The balanced reading: testosterone therapy is not the cardiovascular hazard it was once feared to be, but it is not benign. It requires monitoring, and it is not a cosmetic intervention.

TABLE 12. MONITORING AND RISKS OF TESTOSTERONE THERAPY

DOMAIN	SIGNAL	ACTION
Haematocrit	Erythrocytosis	Check at baseline, 3–6 months, then annually; hold or reduce dose if haematocrit rises markedly.
Cardiovascular	Atrial fibrillation, VTE signals in TRAVERSE	Assess baseline risk; counsel; investigate palpitations or dyspnoea promptly.
Renal	Acute kidney injury signal in TRAVERSE	Baseline and periodic renal function, particularly in CKD.
Bone	Fracture signal in TRAVERSE	Consider bone health, especially in older men and those on other fracture-risk medications.
Prostate	No clear cancer signal; PSA may rise modestly	Baseline PSA; monitor per age-appropriate schedule; urology referral for abnormal kinetics.
Fertility	Suppression of spermatogenesis	Discuss before initiation; consider fertility-sparing alternatives.

Fertility considerations

Exogenous testosterone suppresses LH and FSH through negative feedback. In the testis, that means reduced intratesticular testosterone — which is required at concentrations far above serum levels for spermatogenesis. The predictable consequence is **impaired sperm production, up to azoospermia**, in a substantial proportion of men. This is not a rare idiosyncratic effect; it is the mechanism of action.

Recovery after cessation is variable and can take many months, and is not guaranteed. For any man who may want children, the options are:

- **Sperm banking** before starting therapy.
- **Fertility-sparing alternatives** — hCG to stimulate Leydig cells directly, or selective oestrogen receptor modulators such as clomiphene or enclomiphene to increase endogenous LH and FSH. These are used off-label for hypogonadism and are not equivalent to testosterone for all endpoints, but they preserve spermatogenesis.
- **Aromatase inhibitors** in selected men with a high estradiol-to-testosterone ratio — off-label, and requiring specialist supervision.
- **Optimise the modifiable factors first.** Weight loss, sleep and alcohol reduction improve endogenous production without suppressing the axis.

> **THE FERTILITY CONVERSATION HAPPENS FIRST**
>
> The most common irreversible regret in this field is a man in his thirties who started testosterone for fatigue and discovered two years later that he and his partner could not conceive. Ask about fertility plans *before* writing the prescription, document the answer, and offer sperm banking where relevant.

SECTION FOURTEEN

Monitoring and Tracking

What gets measured gets managed — but only if the measurements are taken consistently enough to be comparable. A testosterone result from a different laboratory, at a different time of day, after a different night's sleep, is not a follow-up; it is a new baseline.

Biomarkers to track

TABLE 13. MONITORING PANEL

MARKER	FREQUENCY	INTERPRETATION
Total testosterone (same assay)	Baseline, 3 months, then 6–12 monthly	Always fasting, 07:00–10:00, same laboratory.
SHBG and calculated free T	With each total T	Detects SHBG-driven artefact, especially with weight change.
Haematocrit / full blood count	Baseline; 3 and 6 months on TRT; annually	Erythrocytosis is the commonest dose-limiting adverse effect of TRT.
PSA	Baseline; per age and risk	Monitor kinetics rather than absolute values alone.
HbA1c, fasting lipids	Baseline and 6–12 monthly	Tracks the metabolic axis driving the whole picture.
25-hydroxyvitamin D, ferritin	Baseline; 3 months after repletion	Confirms deficiency correction rather than assumed correction.
Waist circumference, body weight, blood pressure	Monthly	Cheap, reliable, and more predictive of metabolic trajectory than testosterone itself.

Retesting schedule

- **Lifestyle-only protocol:** retest at 12 weeks, after at least 8 weeks of stable behaviour and stable weight.
- **After starting a supplement:** allow 8–12 weeks before retesting, and change only one variable at a time.

- **After starting TRT:** testosterone and haematocrit at approximately 3 months, again at 6 months, then annually once stable.
- **After a diagnosis of OSA and CPAP initiation:** reassess symptoms at 3 months and testosterone at 6 months.

Self-tracking that adds signal

- **Symptom instruments.** A validated sexual function questionnaire (such as IIEF-5) and a structured symptom score provide far more usable information than "I feel better."
- **Sleep.** Average duration and a simple quality rating, weekly rather than nightly.
- **Training.** Load and reps logged, so that progressive overload is a fact rather than a memory.
- **Alcohol units per week** — a variable men routinely underestimate and which correlates directly with the outcome of interest.
- **Waist circumference** at the same landmark, monthly.

> **WHEN TO STOP AND SEEK REVIEW**
>
> Seek clinical review for new or worsening palpitations, unilateral leg swelling or breathlessness (possible VTE), rapid haematocrit rise, new urinary symptoms, gynaecomastia, mood deterioration, or any sustained loss of libido or erectile function despite normalised testosterone. These are not reasons to adjust a supplement dose; they are reasons to see a clinician.

SECTION FIFTEEN

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Important notice. This document synthesises published research and clinical guidance for educational purposes. It is not medical advice and does not create a clinician–patient relationship. Testosterone deficiency requires formal diagnosis; testosterone therapy requires prescription and monitoring; supplements can interact with medications and laboratory tests. Doses cited reflect the protocols used in the referenced studies and are not recommendations for individual use. Readers should consult a qualified clinician before beginning, changing or stopping any intervention described here.