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SCIENTIFIC & MARKET REVIEW | SEPTEMBER 2026

LOW TESTOSTERONE IN THE GLP-1 ERA

Reframing male hormonal health, metabolic hypogonadism, testosterone replacement therapy and the emerging nutraceutical opportunity

CORE THESIS

The strongest opportunity is not to recreate a “natural TRT”, but to build a clinically credible male metabolic-androgen health category around metabolic recovery, muscle preservation, normal hormonal physiology, sexual well-being and healthy ageing.

by

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Literature and regulatory status reviewed up to September 9, 2026

EXECUTIVE SUMMARY

The male-health opportunity has moved beyond “testosterone boosters”

Low testosterone is an important determinant of male health, but the consumer shorthand “low T” often collapses several distinct clinical situations into a single narrative. True hypogonadism requires compatible symptoms or signs together with repeatedly low testosterone concentrations; fatigue, low libido or reduced vitality alone are not diagnostic. A particularly important distinction is between organic hypogonadism and potentially reversible functional hypogonadism associated with obesity, insulin resistance, type 2 diabetes, sleep disorders, medications and chronic disease. [1,2]

The rapid adoption of GLP-1 receptor agonists and related incretin therapies changes this landscape. Weight loss and metabolic improvement can partially reverse obesity-associated suppression of the hypothalamic-pituitary-gonadal axis. Recent clinical studies and meta-analyses increasingly report improvements in total, free or bioavailable testosterone among metabolically compromised men receiving GLP-1-based therapy, although a direct gonadal effect remains unproven. [8-13]

At the same time, pharmacologically induced weight reduction can include loss of lean tissue, creating a second opportunity around muscle preservation, protein adequacy, resistance exercise and micronutrient sufficiency. [14,15] This convergence supports a more sophisticated category: male metabolic-androgen health. The commercial proposition should complement, not mimic, medically indicated TRT.

BOTTOM LINE

GLP-1 therapies do not turn dietary supplements into endocrine drugs. What they do create is a large, medically engaged population in which metabolic health, body composition, muscle, testosterone status and sexual well-being increasingly intersect.

Key numbers shaping the discussion

332 → 399

ng/dL mean total testosterone during incretin therapy

Portillo-Canales et al., 2026 [10]

67% → 86%

patients with total testosterone in the normal range

Portillo-Canales et al., 2026 [10]

~25%

of weight lost represented lean mass in SURMOUNT-1 DXA substudy

Look et al., 2025 [14]

7.0% vs 7.3%

MACE: TRT vs placebo in TRAVERSE

Lincoff et al., 2023 [4]

Conceptual framework

Figure 1. Strategic reframing of low testosterone in the GLP-1 era. HPG: hypothalamic-pituitary-gonadal.

CONTENTS

01. Low testosterone is important - but “low T” is not a diagnosis by itself

Testosterone influences sexual function, spermatogenesis, skeletal muscle, bone metabolism, erythropoiesis, adipose-tissue distribution and multiple aspects of male physiology. Clinically significant testosterone deficiency can therefore impair quality of life and contribute to reduced libido, loss of spontaneous erections, erectile dysfunction, decreased muscle mass, reduced bone mineral density, anaemia and impaired physical function.

Nevertheless, symptoms commonly attributed to low testosterone are highly nonspecific. Fatigue, reduced motivation, poor concentration, depressed mood, sleep disturbance and impaired exercise tolerance occur in many conditions unrelated to androgen deficiency. Sexual symptoms - particularly reduced libido and loss of spontaneous or morning erections - are more informative, but even these do not establish hypogonadism without biochemical confirmation.

The 2026 JAMA review of adult male hypogonadism reiterates that diagnosis requires clinical features of androgen deficiency together with consistently low early-morning testosterone concentrations, generally confirmed on at least two occasions. Obesity complicates interpretation because reduced sex hormone-binding globulin (SHBG) can lower measured total testosterone without an equivalent fall in biologically available androgen; free testosterone therefore becomes especially relevant in men with obesity, diabetes or altered SHBG. [1]

The practical implication is fundamental: “low testosterone”, “low T symptoms” and hypogonadism are not interchangeable concepts. A nutritional product intended to support normal hormonal physiology occupies a fundamentally different clinical and regulatory territory from a drug intended to treat established hypogonadism.

02. Organic versus functional hypogonadism: the distinction that changes the market

Male hypogonadism can arise from primary testicular failure or inadequate hypothalamic-pituitary stimulation. In primary hypogonadism, reduced testosterone is accompanied by elevated luteinising hormone (LH) and often follicle-stimulating hormone (FSH). Secondary hypogonadism is characterised by low testosterone with low or inappropriately normal gonadotropins.

Structural or genetic disease represents one end of the spectrum: Klinefelter syndrome, pituitary disorders, testicular damage, chemotherapy, radiotherapy and other organic causes can produce persistent androgen deficiency requiring specialist treatment.

At the other end lies functional secondary hypogonadism, increasingly associated with obesity and metabolic disease. Visceral adiposity, insulin resistance, hyperinsulinaemia, inflammatory signalling, altered leptin biology, sleep-disordered breathing and increased aromatisation of testosterone to estradiol can suppress the hypothalamic-pituitary-gonadal axis. Reduced SHBG additionally lowers total circulating testosterone. Lower testosterone may in turn favour increased fat mass and reduced muscle mass, creating a bidirectional metabolic-endocrine cycle. [2]

This mechanism challenges the simplistic commercial proposition that every man with low testosterone requires a direct testosterone-enhancing intervention. For a substantial subgroup, the metabolic environment itself is suppressing gonadal function.

03. Weight loss can restore testosterone - an observation that predates GLP-1 drugs

The relationship between weight reduction and restoration of male androgen status is not new. A 2024 meta-analysis incorporating 44 studies and 1,774 men found that weight reduction increased both total and free

testosterone. The magnitude of improvement correlated with weight loss and was particularly pronounced in individuals with greater obesity and lower baseline testosterone. [3]

This establishes an important causal framework for interpreting the newer GLP-1 literature: if obesity contributes to suppression of testosterone, a therapy producing substantial and durable weight reduction should be expected to improve androgen physiology in at least some patients.

GLP-1 therapies therefore entered a biological system in which the metabolic-androgen connection was already well established. What changed is the magnitude, scalability and pharmacological reproducibility of weight loss now achievable without bariatric surgery.

04. TRT remains an effective medical therapy - but it should not be confused with wellness optimisation

Testosterone replacement therapy is an established treatment for appropriately diagnosed androgen deficiency. In hypogonadal men it can improve sexual function, correct anaemia, increase lean body mass and bone density and improve selected quality-of-life parameters.

The safety discussion has evolved materially. The TRAVERSE trial randomised more than 5,200 men aged 45-80 years with symptoms of hypogonadism, two fasting testosterone concentrations below 300 ng/dL and established or increased cardiovascular risk. Major adverse cardiovascular events occurred in 7.0% of testosterone-treated participants and 7.3% of placebo recipients, establishing non-inferiority for the primary cardiovascular endpoint. Pulmonary embolism, atrial fibrillation and acute kidney injury nevertheless occurred somewhat more frequently in the testosterone arm and remain relevant to individual risk assessment. [4]

Following TRAVERSE, the FDA removed previous boxed-warning language suggesting increased risk of major cardiovascular outcomes from testosterone products in 2025 while adding class-wide attention to blood-pressure increases. In June 2026, US regulators also requested further label revisions, including removal of the longstanding limitation concerning age-related hypogonadism. [5,6]

TRT therefore occupies a stronger evidence-based position than many popular discussions imply, but it remains medical therapy, not a lifestyle supplement. A particularly important limitation is fertility: exogenous testosterone suppresses hypothalamic-pituitary gonadotropin signalling and can markedly reduce spermatogenesis. [7]

05. The GLP-1 revolution changes male hormonal health

The rapid expansion of semaglutide, liraglutide, tirzepatide and related incretin therapies is usually discussed in the context of obesity and diabetes. Their significance for men's hormonal health may ultimately prove broader.

There are two distinct effects. The first is biological: GLP-1-based therapies can dramatically improve adiposity, insulin resistance, inflammatory burden and other factors that suppress the male reproductive axis. The second is behavioural and commercial: they have normalised a model in which consumers simultaneously use prescription medicine, nutritional products, digital health monitoring and lifestyle interventions.

Scott Dicker, Senior Director of Market Insights at SPINS, has explicitly linked widespread GLP-1 adoption with reduced consumer resistance to combining pharmaceuticals and nutraceuticals and has identified testosterone and hormone support as an emerging category. [24]

That market observation becomes more powerful because emerging clinical research increasingly provides a biological basis for the same convergence.

06. Are GLP-1 therapies actually increasing testosterone?

Evidence accumulated rapidly during 2025-2026. A systematic review and meta-analysis of seven studies involving 680 patients found that GLP-1 receptor agonist treatment was associated with a significant increase in total testosterone together with favourable changes in free testosterone, SHBG, gonadotropins and metabolic

parameters. The authors nevertheless concluded that the existing literature could not demonstrate a direct testicular action independent of weight loss. [8]

A separate 2025 meta-analysis identified four eligible studies and found a significant improvement in bioavailable testosterone, while free testosterone and SHBG remained inconclusive. The small evidence base and heterogeneity are major limitations. [9]

A 2026 retrospective clinical study provides a useful real-world signal. Among 215 men with paired total testosterone measurements, mean total testosterone increased from approximately 332 to 399 ng/dL. In the subset with paired free testosterone measurements, free testosterone also increased, and the proportion of men with total testosterone in the normal range rose from 67% to 86%. Testosterone improvement correlated with weight loss. [10]

A broader 2026 systematic review of ten studies involving 639 men found that total testosterone generally increased in men with obesity, diabetes or functional hypogonadism; free testosterone responses were less consistent, and some studies suggested improvement in semen parameters. [11]

A 2026 meta-analysis by Corona and colleagues reported significant increases in total and calculated free testosterone and gonadotropins with GLP-1 receptor agonists, with analyses suggesting that hormonal improvement might not be explained entirely by weight loss. That raises the possibility of direct modulation of the hypothalamic-pituitary-testicular axis, but the hypothesis should not yet be treated as established clinical fact. [12]

A controlled pilot study of tirzepatide in men with obesity and metabolic hypogonadism similarly reported increases in total, free and bioavailable testosterone, higher LH and FSH, lower estradiol and improved erectile-function scores after treatment. The study was small and short, so the results remain hypothesis-generating. [13]

EVIDENCE POSITION

The most defensible conclusion is that GLP-1-based metabolic treatment may reverse or attenuate functional testosterone deficiency in some men with obesity or metabolic dysfunction. It does not establish GLP-1 receptor agonists as testosterone drugs or replacements for TRT in organic hypogonadism.

07. A paradox: GLP-1 treatment may improve testosterone while creating new nutritional vulnerabilities

The endocrine benefits of weight loss coexist with another physiological consideration: weight loss is not composed exclusively of adipose tissue.

In the SURMOUNT-1 body-composition substudy, tirzepatide produced a 21.3% reduction in body weight after 72 weeks. Fat mass decreased by 33.9%, but lean mass also decreased by 10.9%; approximately 75% of weight lost represented fat and 25% lean mass. [14]

This does not mean that GLP-1 therapies inherently cause pathological muscle wasting. Some fat-free mass loss accompanies substantial weight reduction through many interventions. Nevertheless, the issue becomes clinically important in older men, sarcopenic individuals, men with low baseline testosterone or muscle reserve, and patients whose appetite suppression leads to inadequate protein or micronutrient intake.

A 2026 narrative review examining potential combined use of TRT and GLP-1 therapies concluded that the concept is physiologically plausible but that no randomised trial currently supports routine combined therapy specifically for preservation of lean mass. [15]

This creates a legitimate nutritional opportunity: support the physiology of weight loss rather than claiming to pharmacologically replace testosterone.

08. The problem with the traditional “testosterone booster” category

The nutritional-supplement market historically approached testosterone through a simple promise: take ingredient X and testosterone will rise. The scientific literature does not support such a broad proposition.

A systematic review evaluating 52 studies involving 27 commonly marketed testosterone-enhancing ingredients concluded that most proposed testosterone boosters failed to consistently increase total testosterone. Evidence differed considerably by study population, ingredient standardisation and baseline hormonal status. [16]

Nutrients required for normal steroidogenesis may restore physiological function when deficiency exists. That is fundamentally different from increasing testosterone above normal physiological levels in a nutrient-replete individual. Botanical extracts may exert measurable endocrine effects, but findings obtained with one chemically standardised proprietary extract cannot automatically be transferred to all extracts from the same plant.

Improved libido does not necessarily indicate higher testosterone. Improved strength or body composition does not prove androgenic activity. An increase in total testosterone is not automatically equivalent to improvement in free testosterone, symptoms or clinically meaningful male-health outcomes.

CATEGORY RESET

The strategic shift is from “boosting T” as a single-biomarker promise toward a multi-domain model integrating metabolic health, androgen physiology, muscle, sleep, stress, sexual function and nutritional adequacy.

09. What does the evidence actually say about natural ingredients?

The emerging conclusion is not that natural interventions are irrelevant. It is that the category needs better biological targeting, ingredient standardisation and much more disciplined claims architecture.

Ingredient / approach	Evidence relevant to testosterone	Scientific interpretation	Evidence grade
Zinc	Deficiency is associated with lower testosterone; supplementation can restore status when zinc intake/status is inadequate. The EU authorises a claim on maintenance of normal blood testosterone. [20,22]	Strong physiological rationale / strong EU regulatory utility	**Established for normal physiology**
Ashwagandha	A 2026 meta-analysis of 23 RCTs (1,706 adults) reported a significant testosterone increase in men; heterogeneity and endpoint limitations remain important. [17]	Promising, especially where stress, vitality and male health overlap	**Moderate / emerging**
Fenugreek	Meta-analysis reports small increases in total testosterone in some populations; effects vary substantially by extract and study population. [18]	Potentially useful but extract-dependent	**Moderate / inconsistent**
Eurycoma longifolia	Meta-analysis of five RCTs reported increased total testosterone, including a signal in hypogonadal men. [19]	Promising, but the evidence base is small; standardisation and regulatory status matter	**Emerging**

Ingredient / approach	Evidence relevant to testosterone	Scientific interpretation	Evidence grade
Vitamin D	Observational associations exist, but the most recent meta-analysis of RCTs did not demonstrate a clear reproducible effect on total testosterone, SHBG or androgen bioavailability. [21]	Correct deficiency for established nutritional reasons; do not position as a reliable T booster	**Weak for T elevation**
Magnesium	Biologically important; evidence for clinically meaningful testosterone elevation in replete men is inconsistent.	Useful for nutritional sufficiency; conservative testosterone positioning	**Limited**
Boron	Frequently marketed for free testosterone or SHBG modulation, but human evidence is small and inconsistent.	Insufficient basis for a strong testosterone claim	**Limited**
Tribulus terrestris	Controlled evidence has not consistently demonstrated meaningful testosterone elevation. [16]	Poor scientific centrepiece for an evidence-led platform	**Weak**
D-aspartic acid	Findings are inconsistent and population-dependent; systematic review evidence does not support reliable elevation. [16]	Commercial familiarity exceeds evidential strength	**Weak**

Table 1. Evidence is graded qualitatively for strategic formulation purposes, not as a formal GRADE assessment.

10. Zinc deserves particular attention in Europe

From a European regulatory perspective, zinc is unusually valuable. Commission Regulation (EU) No 432/2012 authorises the health claim: “Zinc contributes to the maintenance of normal testosterone levels in the blood”, provided the applicable conditions of use are met. [22]

The wording is scientifically and commercially powerful precisely because it does not promise to raise abnormally low testosterone or treat hypogonadism. “Maintenance of normal testosterone” is a homeostatic nutritional claim. “Treats low testosterone”, “reverses hypogonadism”, “natural TRT” or equivalent disease-treatment positioning would occupy a completely different regulatory category.

Vitamin B6 also has an authorised EU claim that it contributes to the regulation of hormonal activity. This can support a hormone-oriented formulation, but it should not be converted into an unsupported testosterone-specific assertion. [22]

EU CLAIMS ARCHITECTURE

Defensible: “Zinc contributes to the maintenance of normal testosterone levels in the blood.”
High-risk / non-compliant positioning: “treats low testosterone”, “reverses hypogonadism”, “natural TRT” or equivalent medicinal claims.

11. The United States provides more flexibility - but not unlimited flexibility

US dietary supplement law permits structure/function claims where manufacturers possess adequate substantiation and the statements are truthful and non-misleading. A supplement may therefore potentially support normal hormonal function, vitality or physiological testosterone production when properly substantiated.

FDA draws a clear distinction between structure/function claims and disease claims. A dietary supplement cannot legally represent itself as diagnosing, treating, curing or preventing a disease without entering the drug regulatory framework. Claims positioning a supplement as a replacement for drug therapy can themselves create regulatory problems. [23]

Commercially seductive language such as “nature's TRT” should therefore be approached with caution. GLP-1 marketing already demonstrated how comparisons such as “nature's Ozempic” can achieve attention while creating weak pharmacological equivalence and regulatory exposure.

12. GLP-1 users may become one of the most important new male-health populations

The emerging GLP-1 male consumer is not necessarily the traditional sports-nutrition consumer. He may be middle-aged or older, have obesity, insulin resistance or type 2 diabetes, and be losing substantial weight for the first time in decades.

He may notice improved mobility and metabolic health while simultaneously worrying about muscle loss, fatigue, strength, sexual function, nutritional adequacy or changing body composition. He may already be measuring biomarkers through laboratory testing and wearable technologies and may be considerably more comfortable combining prescription pharmacotherapy with nutritional interventions than previous generations.

This is precisely the transition highlighted by SPINS: GLP-1 adoption has helped make medically supervised pharmacotherapy and nutraceutical consumption complementary rather than mutually exclusive behaviours. [24]

That consumer does not necessarily need a “testosterone booster”. He may need a male-health metabolic companion system.

13. From “testosterone booster” to “metabolic-androgen resilience”

A scientifically stronger product architecture can be built around four interacting domains: androgen nutritional sufficiency; musculoskeletal resilience; metabolic recovery; and male vitality / sexual well-being.

Androgen nutritional sufficiency means ensuring adequate zinc and other nutrients required for normal endocrine physiology rather than attempting to force testosterone above physiological levels. Musculoskeletal resilience means preserving skeletal muscle during energy restriction through adequate protein, resistance exercise and evidence-led nutritional support.

Metabolic recovery recognises that improving obesity, insulin resistance and visceral adiposity can itself alleviate functional suppression of the hypothalamic-pituitary-gonadal axis. Male vitality and sexual well-being create a role for carefully selected botanical interventions with human clinical data, provided their effects are not automatically presented as testosterone-mediated.

This reframing is commercially more sophisticated because it reflects what is actually happening physiologically. It also creates a category capable of serving GLP-1 users, men undergoing medically supervised weight management, middle-aged consumers concerned about body composition and vitality, sports-nutrition consumers moving into healthy ageing, and selected men receiving TRT who require general nutritional support.

Four-pillar product architecture

01	02	03	04
ANDROGEN NUTRITIONAL SUFFICIENCY	**MUSCULOSKELETAL RESILIENCE**	**METABOLIC RECOVERY**	**VITALITY & SEXUAL WELL-BEING**
Zinc-led support for normal endocrine physiology.	Protein adequacy, resistance exercise and lean-mass preservation.	Addressing adiposity, insulin resistance and functional HPG suppression.	Evidence-led botanical support without overclaiming testosterone effects.

14. GLP-1 therapy and TRT may eventually converge - but the evidence is not yet there

An intriguing future clinical territory is combined GLP-1-based obesity treatment and TRT in men who genuinely fulfil diagnostic criteria for hypogonadism. The rationale is straightforward: GLP-1 therapy can improve metabolic health and reduce adiposity, while TRT can restore androgen signalling where endogenous testosterone remains pathologically inadequate.

This makes combined treatment biologically plausible in selected patients. However, biological plausibility is not clinical proof. As of September 2026, there are no large randomised controlled trials demonstrating that routine addition of TRT to GLP-1 therapy improves long-term muscle, cardiovascular or functional outcomes sufficiently to justify broad combined use. [15]

Conversely, men whose low testosterone primarily reflects obesity-related functional suppression may experience substantial hormonal improvement from weight loss alone and may therefore avoid unnecessary lifelong exogenous testosterone. This reinforces the importance of phenotyping patients rather than treating a laboratory value.

15. Sexual function is not synonymous with testosterone

The commercial testosterone category frequently conflates testosterone, libido and erectile function. Physiologically, these overlap but are not equivalent. Libido has an important androgenic component. Erectile function depends heavily on vascular, neurological, psychological and metabolic health.

Several studies indicate improvements in erectile-function scores after metabolic treatment, but part of this effect could derive from weight reduction, improved endothelial function, glycaemic control, mobility and psychological well-being rather than a direct testosterone effect. [8,12,13]

The evidence is not entirely uniform. Observational data have also reported a small absolute increase in erectile-dysfunction diagnoses or PDE5-inhibitor prescriptions in selected younger men prescribed semaglutide. Such retrospective findings cannot establish causality and illustrate why simplistic claims should be avoided.

16. Fertility may become an unexpectedly important differentiation point

Exogenous testosterone suppresses LH and FSH and can substantially impair sperm production. GLP-1-mediated restoration of endogenous testosterone, by contrast, appears generally to preserve gonadotropin signalling. Some early studies report improvements in selected semen parameters among obese or hypogonadal men. [11]

A 2026 Endocrine Society report on clinical-trial evidence similarly highlighted the possibility that GLP-1 therapies may improve testosterone and sperm quality in men with obesity-related low testosterone while addressing the underlying metabolic state. The evidence is not sufficient to establish GLP-1 therapy as a fertility treatment.

This distinction may become increasingly relevant as younger men use GLP-1 therapies and as hormone optimisation becomes mainstream. It also demonstrates why TRT, endogenous testosterone support and metabolic restoration should never be treated as equivalent interventions.

17. A new definition of the commercial opportunity

The testosterone market is expanding at the intersection of healthy ageing, metabolic medicine, GLP-1 pharmacotherapy, sports nutrition, continuous biomarker monitoring, sexual health, body-composition optimisation and increasing consumer acceptance of hormone-directed medical care.

The traditional testosterone-booster industry addressed only one part of this landscape. The larger opportunity is male physiological optimisation during metabolic transition.

A consumer can enter the ecosystem through obesity treatment and subsequently become interested in muscle maintenance, protein, micronutrients, testosterone testing, libido, physical performance, sleep and healthy ageing. Another may enter through sports nutrition and later develop interest in metabolic health and hormone status. Another may begin TRT after formal diagnosis and require broader nutritional and cardiometabolic support.

These populations overlap substantially but should not be treated identically. This creates room for precision segmentation rather than a single generic men's-vitality formula.

18. Scientific and commercial priorities for next-generation formulations

The strongest innovation strategy should begin with phenotype rather than ingredient. A formula designed for men undergoing substantial GLP-1-mediated weight loss should prioritise nutritional adequacy, muscle preservation and metabolic resilience. A formulation for healthy middle-aged men concerned with normal hormonal physiology may place greater emphasis on zinc and appropriately substantiated botanicals.

A product positioned around stress-related vitality may rationally integrate adaptogenic mechanisms rather than pretending every benefit is mediated through testosterone. An active-ageing formula could integrate muscle, bone, energy metabolism and hormone-support claims within a broader framework.

Clinical development should move beyond measuring total testosterone alone. Better trials would incorporate free testosterone or SHBG where relevant, body composition, muscle strength, sexual-function instruments, fatigue, quality of life and relevant metabolic biomarkers.

The most credible next-generation products will not necessarily be those producing the largest headline testosterone increase. They will be those able to demonstrate clinically meaningful functional outcomes in a clearly defined male population.

19. The strategic lesson from GLP-1

Dicker's observation about the GLP-1 space is more profound than a prediction that testosterone supplements may become fashionable again. GLP-1 therapies have changed consumer expectations about health optimisation.

Consumers increasingly understand that obesity, blood glucose, body composition, hormones, nutrition and physical performance are interconnected. They are becoming accustomed to pharmaceutical therapy being only one component of a broader health-management system.

That creates an opening for the supplement industry, but it also raises the scientific standard. A consumer taking semaglutide or tirzepatide, undergoing regular laboratory testing and discussing testosterone with a physician is fundamentally different from a consumer buying an anonymous test booster because of a bodybuilding advertisement.

The new market is more medicalised, more biomarker-driven and increasingly evidence-demanding. Companies able to bridge clinical medicine, nutritional science, regulatory competence and consumer-friendly formulation are therefore likely to possess an advantage over brands relying on exaggerated testosterone claims.

20. Conclusion

Low testosterone is a genuine and clinically important component of men's health, but the subject has been oversimplified by consumer marketing and parts of the wellness industry. Not every man with fatigue has testosterone deficiency. Not every low testosterone laboratory result represents permanent hypogonadism. And not every man with obesity-associated low testosterone necessarily requires lifelong testosterone replacement.

Obesity and metabolic dysfunction can reversibly suppress the male reproductive axis. Weight loss can increase testosterone. The success of GLP-1 and incretin-based therapies therefore introduces a fundamentally new dimension into male hormonal health.

Evidence emerging in 2025 and 2026 increasingly indicates that GLP-1 therapy can improve total or bioavailable testosterone and potentially sexual or reproductive parameters in metabolically compromised men. Whether these effects are entirely mediated by weight loss or include direct actions on the hypothalamic-pituitary-gonadal axis remains unresolved.

TRT continues to have an important role when true hypogonadism is present, and its cardiovascular safety profile is now better characterised following TRAVERSE and subsequent FDA reassessment. Nevertheless, fertility suppression, erythrocytosis, blood-pressure effects, prostate surveillance and other clinical considerations reinforce the need for appropriate diagnosis and medical supervision.

Natural interventions occupy a different position. The evidence does not justify presenting nutritional supplements as substitutes for TRT. Most traditional testosterone boosters have limited or inconsistent clinical support. Zinc possesses a particularly strong physiological and regulatory rationale for supporting normal testosterone status; ashwagandha, fenugreek and Eurycoma longifolia provide potentially useful but less definitive evidence.

The greatest commercial opportunity generated by the GLP-1 revolution may therefore not be another generation of testosterone boosters. It may be the creation of a new category: MALE METABOLIC-ANDROGEN HEALTH - a scientifically grounded platform focused on metabolic recovery, preservation of muscle and functional capacity, adequate nutrition, normal hormonal physiology, sexual well-being and healthy ageing, capable of operating alongside GLP-1 therapies, lifestyle intervention and medically indicated TRT rather than attempting to replace them.

Do not compete with TRT by pretending to be TRT. Build the nutritional infrastructure around the metabolic, musculoskeletal and hormonal transition that GLP-1 therapies have made mainstream.

REFERENCES

Selected scientific, regulatory and market sources

1. *Anawalt BD, O'Connor KM, Grossmann M. Adult Male Hypogonadism: A Review. JAMA. 2026;335(24):2146-2159. PMID 42207626. Source*
2. *Fernandez CJ, Chacko EC, Pappachan JM. Male obesity-related secondary hypogonadism: Pathophysiology, clinical implications and management. 2019. PMID 31616498. Source*
3. *Ken-Dror G, Fluck D, Fry CH, Han TS. Meta-analysis and construction of simple-to-use nomograms for approximating testosterone levels gained from weight loss in obese men. Andrology. 2024;12:297-315. PMID 37345263. Source*
4. *Lincoff AM, Bhasin S, Flevaris P, et al. Cardiovascular Safety of Testosterone-Replacement Therapy. N Engl J Med. 2023;389:107-117. doi:10.1056/NEJMoa2215025. Source*
5. *US Food and Drug Administration. FDA issues class-wide labeling changes for testosterone products. 2025. Source*
6. *US Department of Health and Human Services / FDA. Requested updates to Testosterone Therapy Product Labels. 2026. Source*
7. *Bhasin S, Brito JP, Cunningham GR, et al. Testosterone Therapy in Men With Hypogonadism: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2018. Source*
8. *Effects of glucagon-like peptide-1 receptor agonists on testicular dysfunction: A systematic review and meta-analysis. Andrology. 2025. PMID 40105090. Source*
9. *Orra SH, Nabhan Martinez JV, Porto BC, et al. Effect of GLP-1 agonists on testosterone levels: a systematic review and meta-analysis. BMC Urol. 2025;25:311. doi:10.1186/s12894-025-02005-0. Source*
10. *Portillo-Canales S, Iqbal I, Akande R, et al. Effect of Incretin-Based Weight Loss Drugs on Testosterone Concentrations in Men. Endocr Pract. 2026;32(5):696-703. doi:10.1016/j.eprac.2026.01.003. Source*
11. *Deameh MG, Ramez M, Rowaiee R, et al. Effects of glucagon-like peptide-1 receptor agonists on male reproductive hormones, semen parameters, and metabolic outcomes: a systematic review. J Sex Med. 2026;23(2). PMID 41498523. Source*
12. *Corona G, Sparano C, Romeo M, Rastrelli G, Vignozzi L, Maggi M. Emerging Effects of GLP-1 Receptor Agonists and SGLT2 Inhibitors on Male Sexual Hormones and Behaviors: Systematic Review and Meta-Analysis. Andrology. 2026. PMID 42011503. Source*
13. *Short-term impact of tirzepatide on metabolic hypogonadism and body composition in patients with obesity: a controlled pilot study. Reprod Biol Endocrinol. 2025;23:92. PMID 40604795. Source*
14. *Look M, Dunn JP, Kushner RF, et al. Body composition changes during weight reduction with tirzepatide in the SURMOUNT-1 study of adults with obesity or overweight. Diabetes Obes Metab. 2025;27:2720-2729. PMID 39996356. Source*
15. *Canal de Velasco LM, González Flores JE, Kraus Fischer G, et al. Testosterone Replacement Therapy as a Potential Strategy to Preserve Lean Mass in Men With Persistently Low Serum Testosterone Receiving GLP-1 Receptor Agonists: A Narrative Review. Cureus. 2026;18(3):e105734. doi:10.7759/cureus.105734. Source*
16. *Morgado A, Tsampoukas G, Sokolakis I, Schoentgen N, Urkmez A, Sarikaya S. Do "testosterone boosters" really increase serum total testosterone? A systematic review. Int J Impot Res. 2024;36:348-364. doi:10.1038/s41443-023-00763-9. Source*
17. *Fornalik M, Malkiewicz A, Adamczak D, et al. Hormonal Modulation with Withania somnifera: Systematic Review and Meta-Analysis of Randomized-Controlled Trials. Planta Med. 2026;92(8):790-805. PMID 41740946. Source*
18. *Mansoori A, Hosseini S, Zilaee M, Hormoznejad R, Fathi M. Effect of fenugreek extract supplement on testosterone levels in male: A meta-analysis of clinical trials. Phytother Res. 2020;34:1550-1555. PMID 32048383. Source*
19. *Leisegang K, Finelli R, Sikka SC, Selvam MKP. Eurycoma longifolia Improves Serum Total Testosterone in Men: A Systematic Review and Meta-Analysis of Clinical Trials. Medicina. 2022;58:1047. PMID 36013514. Source*
20. *Te L, Liu J, Ma J, Wang S. Correlation between serum zinc and testosterone: A systematic review. J Trace Elem Med Biol. 2023. PMID 36577241. Source*
21. *Paez-Allendes L, Valenzuela-Fuenzalida JJ, Moya MP, et al. Vitamin D Supplementation, Total Testosterone, and Androgen Bioavailability Markers in Adult Men: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Nutrients. 2026;18(13):2090. Source*
22. *European Commission. Commission Regulation (EU) No 432/2012 establishing a list of permitted health claims made on foods. Source*
23. *US Food and Drug Administration. Structure/Function Claims. Source*
24. *NutraIngredients. SPINS: Protein still reigns, but fiber and hormone support gain momentum. 24 February 2026. Comments by Scott Dicker, Senior Director of Market Insights, SPINS. Source*
25. *Endocrine Society. Clinical trials suggest GLP-1s may improve fertility in men with obesity. ENDO 2026 press release, 13 June 2026. Source*

PUBLICATION & REGULATORY NOTE

MALE METABOLIC- ANDROGEN HEALTH

A framework for scientifically credible innovation at the intersection of GLP-1 pharmacotherapy, metabolic recovery, muscle preservation and normal male hormonal physiology.

SCIENTIFIC AND REGULATORY NOTE

This review is intended for scientific, strategic and professional discussion. It does not constitute individual medical advice, diagnosis or treatment. Ingredient positioning and claims must be assessed against the final formulation, dose, target population, jurisdiction, product category and current applicable legislation.

STATUS

Literature and regulatory status reviewed through September 9, 2026. Future revisions should reassess new randomised trials, evolving obesity pharmacotherapy, testosterone-label changes and jurisdiction-specific supplement claims guidance.

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