The Testosterone Project

The Hidden Cost of Missed Testosterone Deficiency

A policy evidence brief for The Testosterone Project

July 2026

Manoj Teltumbade, PhD
CognitiveCare Inc.

Chetan Chavan, PhD
CognitiveCare Inc.

Table of Contents

Executive Summary 4

Visual Abstract 5

Why This Matters Now 6

The Case for Targeted Testing 7

What Responsible Testosterone Care Requires 8

What the Clinical Evidence Supports 9

Economic Burden and Savings Boundaries 11

Access, Persistence, and Distributional Value 12

Policy Implications 13

Conclusion 14

Bibliography 15

Disclaimer

This paper was prepared in response to a request for proposals from The Testosterone Project, a nonprofit advocacy initiative focused on education, access, and reform around testosterone and hormone health. The authors received financial support from The Testosterone Project after selection through that process. The Testosterone Project provided feedback during revision, and selected suggestions were incorporated into the final paper. The resulting document should be read as a commissioned policy evidence brief prepared through an iterative review process.

This paper is a rapid evidence synthesis and policy framing document. It is intended to support evidence-based discussion of testosterone therapy, men's health, economic burden, access, and distributional value within the scope described in the RFP.

The authors used generative AI tools to support editing and language refinement. The authors reviewed, revised, and approved the final paper before submission.

When citing, excerpting, or adapting this paper for educational, policy, payer-facing, employer-facing, or public communications, users should preserve distinctions between established evidence, reasonable inference, and areas where further research is needed.

Suggested citation:

Teltumbade M, Chavan C. *The Hidden Cost of Missed Testosterone Deficiency*. Prepared for The Testosterone Project; July 2026.

Executive Summary

A low-cost testosterone test may be the missing first step for symptomatic, at-risk men.

The individual signal is concrete. In a matched claims analysis, men with newly diagnosed primary or secondary hypogonadism incurred approximately $2,821 more in annual direct medical spending than matched controls[1], and privately insured employees with hypogonadism carry roughly $4,869 more per year in combined direct and workforce costs ($11,957 vs. $7,088).[2] Aggregated, Moskovic et al. projected that testosterone deficiency among U.S. men ages 45 to 74 could be associated with $190 billion to $525 billion in inflation-adjusted direct healthcare expenditures over 20 years, with about $8.4 billion in the first year alone.[3] Together, the national projection illustrates the scale of the burden, while patient-level studies provide measurable evidence of associated healthcare and workforce costs.

The most concrete metabolic prevention signal comes from T4DM. In T4DM, among selected high-risk men, testosterone plus lifestyle intervention reduced the two-year OGTT-defined diabetes endpoint from 21% to 12% — an absolute reduction of 9 points, or a number-needed-to-treat of about 11 to prevent one case of diabetes.[4] Against a backdrop of 115.2 million U.S. adults with prediabetes[5] and $412.9 billion in annual diabetes cost,[6] that is a notable prevention signal in this selected high-risk population. Scaled to 1 million similar high-risk men, the 9-point difference implies roughly 90,000 fewer diabetes endpoints and, using Parker et al.'s $12,022 in annual diabetes-attributable cost, about $1.1 billion in one-year direct-cost exposure delayed.[6] Anemia correction adds a second measurable pathway: testosterone corrected anemia in 41.0% of anemic hypogonadal men versus 27.5% on placebo at six months,[7] implying ~135,000 additional corrections and an illustrative $68–$270 million in downstream utilization value per 1 million men treated.

These benefits only reach the men who are found. The central failure is recognition. Low testosterone hides beneath the conditions that already dominate men's healthcare spending — type 2 diabetes, metabolic syndrome, obesity, unexplained anemia, bone-density loss, fatigue, and depressed mood — and is repeatedly written off as aging, weight, or stress. The men with the strongest case for evaluation are often the ones who never get tested. In June 2026, HHS announced that FDA was requesting class-wide revisions to the age-related limitation of use and to prostate and BPH safety language. The proposed action changes the regulatory context for treatment discussions but does not create an affirmative efficacy indication or eliminate diagnostic and monitoring requirements.[8]

The ask is disciplined case-finding, not universal treatment. The policy priority is to make targeted evaluation easy to trigger for symptomatic and clinically risk-enriched men, to require repeat morning biochemical confirmation before any diagnosis, and to reserve therapy for confirmed deficiency tied to a specific, evidence-supported goal — improved sexual function or correction of anemia, with metabolic outcomes evaluated in appropriately selected, monitored populations. Savings should be counted only when they are measured after treatment, net of product, monitoring, and adverse-event costs, and adjusted for persistence.[4,7,9] The evidence does not support testosterone as a general wellness intervention. It does support finding the men who are being missed.

Visual Abstract

Testosterone Deficiency: From Recognition to Measured Value

A disciplined pathway for targeted evaluation, confirmed diagnosis, selective treatment, and outcome measurement

$2,821/year

Adjusted 12-month cost difference

Newly diagnosed primary/secondary hypogonadism vs matched controls

2014 USD | observed association, not avoidable cost

9-point difference

NNT ≈ 11 over 2 years

T4DM: testosterone + lifestyle vs lifestyle alone

Selected high-risk men without pathological hypogonadism

$190B-$525B

20-year modeled national burden

U.S. men ages 45-74

Scale estimate | 2013 model | not measured savings

1 TARGET

Evaluate men with compatible symptoms, signs, or guideline-supported risk histories

2 CONFIRM

Repeat early-morning testosterone; assess etiology before diagnosis

3 TREAT SELECTIVELY

Tie therapy to a specific evidence-supported clinical goal

4 MEASURE

Track response, safety, persistence, utilization, and total cost

ILLUSTRATIVE OUTCOME SCENARIOS

DIABETES

90,000 fewer endpoints per 1M similar high-risk men

~$1.1B

One-year direct-cost exposure scenario

ANEMIA

135,000 additional corrections per 1M anemic hypogonadal men treated

$68M-$270M

Utilization-value sensitivity range

☑ Evidence Supports

- ☑ Targeted evaluation
- ☑ Selected clinical benefits
- ☑ Prospective outcome measurement

☒ Does Not Establish

- ☒ Universal screening or treatment
- ☒ Systemwide savings
- ☒ Risk-free therapy

Sources: Moskovic et al.; Grabner et al.; Wittert et al.; Pencina et al.

Why This Matters Now

In June 2026, HHS announced that FDA was requesting class-wide updates to testosterone replacement therapy (TRT) prescribing information. The proposed revisions would remove the limitation of use concerning age-related hypogonadism, narrow the prostate-cancer contraindication to metastatic disease, and revise the BPH warning. HHS reported that FDA considered the limitation no longer warranted after reviewing TRAVERSE and other evidence. These proposed revisions change the regulatory discussion but do not create an affirmative efficacy indication for age-related symptoms. Prostate-risk assessment, pretreatment screening, treatment monitoring, and uncertainty concerning long-term prostate outcomes and severe benign prostatic hyperplasia (BPH) remain.[8,10-12]

Low testosterone also sits beneath several domains that already drive clinical burden and health care spending: sexual dysfunction, anemia, bone-density loss, obesity, metabolic syndrome, type 2 diabetes, cardiovascular-risk markers, fatigue, depressed mood, and lower quality of life. These conditions often overlap, which can cause testosterone deficiency to be missed when symptoms are attributed only to aging, weight, diabetes, chronic illness, medication use, or depression.

The economic scale gives that clinical problem policy urgency. Moskovic et al. estimated a first-year attributable burden of about $8.4 billion and a 20-year inflation-adjusted direct health care burden of approximately $190 billion to $525 billion among U.S. men ages 45 to 74.[3] The diabetes context raises the stakes further: CDC reported 115.2 million U.S. adults with prediabetes in 2023, and diagnosed diabetes cost the United States an estimated $412.9 billion in 2022.[5,6]

The policy opportunity is to build a better pathway for men with properly diagnosed testosterone deficiency: clearer testing triggers, repeat biochemical confirmation, appropriate monitoring, affordable access when treatment is indicated, and outcome measurement that follows diabetes progression, anemia correction, utilization, adverse events, and total cost.

Exhibit 1. Policy and Evidence Timeline

2023

TRAVERSE

5,246 symptomatic men with two low testosterone results and cardiovascular disease or risk. Testosterone was noninferior to placebo for major adverse cardiovascular events in this selected, monitored population.

2025

FDA Labeling

FDA removed cardiovascular boxed-warning language after TRAVERSE, added class-wide blood-pressure warnings, and retained the age-related limitation of use.

2026

REQUESTED CLASS-WIDE TRT LABEL UPDATES

HHS announced that FDA was requesting revisions that would remove the age-related limitation of use, narrow the prostate-cancer contraindication to metastatic disease, and revise the BPH warning. The proposed revisions retain pretreatment risk assessment and monitoring.

Sources: Lincoff et al.; FDA 2025; HHS 2026; FDA 2026.

The Case for Targeted Testing

The economics of testosterone care begin before the first prescription, when a man is either evaluated or overlooked. A serum total testosterone measurement is inexpensive, low-risk, and widely available. Claims and cohort studies consistently associate hypogonadism or low testosterone with higher medical utilization, direct costs, and workforce costs.[1,2,13] These studies do not establish that testing or treatment recovers the observed differences; they identify a burden signal that warrants prospective evaluation. Targeted testing can determine whether testosterone deficiency is part of the clinical picture and whether further assessment is justified.

Evaluate the men most likely to be missed. Guideline-recognized and clinically relevant triggers include persistent symptoms together with selected risk histories such as unexplained anemia, bone-density loss, chronic opioid or corticosteroid exposure, pituitary or testicular disease, infertility evaluation, and diabetes or metabolic risk when the clinical context raises suspicion.[14-16] In these settings, symptoms can be attributed to the more familiar diagnosis and testosterone deficiency can remain unrecognized. These are reasonable settings for targeted testing, provided diagnosis still requires compatible symptoms or signs, repeat biochemical confirmation, and etiologic assessment.

Confirmation protects everyone. Making testing easier must be paired with making diagnosis harder to overstate. A single low value is not a diagnosis. The Endocrine Society and AUA both require symptoms or signs plus repeat early-morning testosterone confirmation before treatment, with free testosterone when total is borderline or SHBG may distort interpretation.[14,15] This is what separates a credible targeted-testing pathway from indiscriminate prescribing — and it is what makes the resulting cost case defensible to payers.

Equity is an evaluation problem before it is a treatment problem. The men with the strongest clinical case — high symptom burden, unexplained anemia, metabolic risk — may also face substantial barriers to repeated morning lab visits, specialist referral, prior authorization, and cost sharing. Those barriers determine who gets tested, who gets confirmed, and who is left untreated despite genuine need. A testing pathway that ignores these frictions risks missing the patients it should reach first. Building low-friction, well-documented testing into primary care — especially for comorbidity-enriched men — is the most direct way to close that gap.

If the observed annual cost associations persisted for approximately 27 years, their discounted gross value at 3% would be on the order of $50,000 to $90,000 per man.[1,2,4,7] This is an illustrative extrapolation—not an estimate of observed lifetime costs or proven treatment savings—and any realizable value would be reduced by treatment costs, monitoring, nonresponse, and discontinuation.

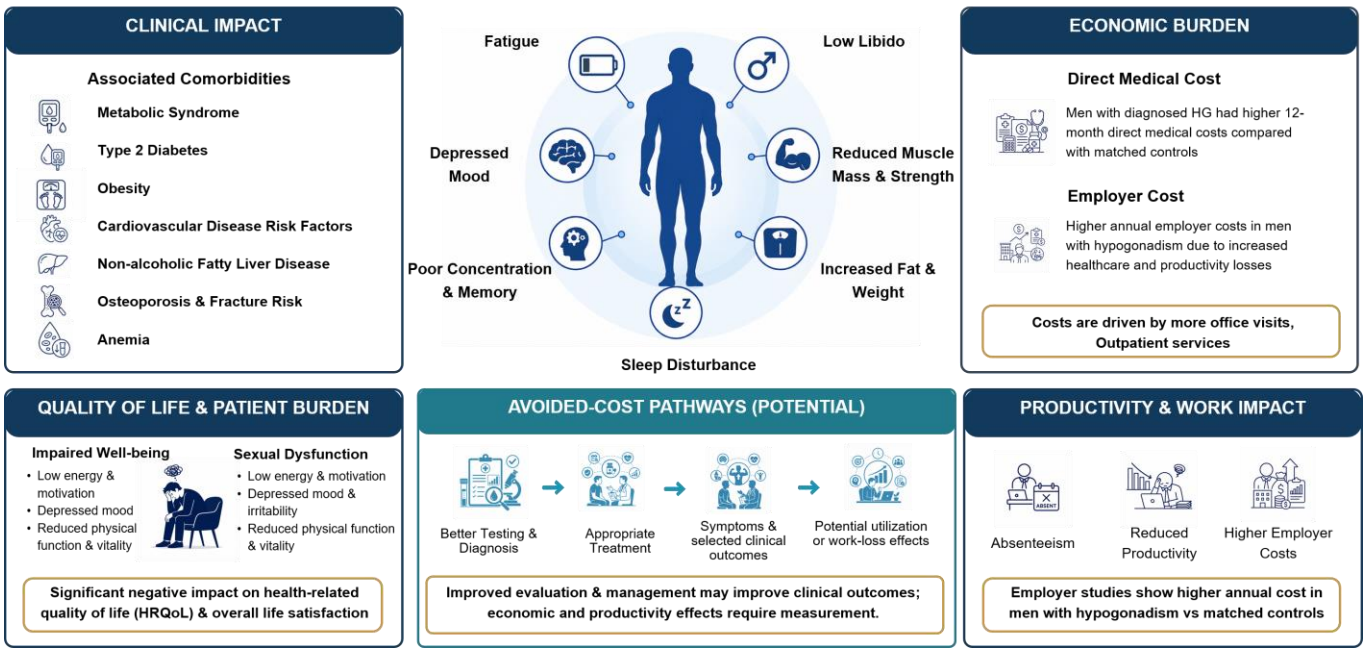
The bottom line for policy. A system that pays for conditions associated with low testosterone but does not reliably evaluate symptomatic, risk-enriched men may be absorbing burden without determining whether testosterone deficiency is part of the clinical picture. A practical, potentially high-leverage reform is to make targeted, confirmed testing routine for symptomatic and clinically risk-enriched men.

What Responsible Testosterone Care Requires

Responsible testosterone care should make testing easier to trigger and diagnosis harder to overstate. Men with symptoms or signs of testosterone deficiency deserve assessment, and so do men with comorbidities or clinical histories in which low testosterone is more likely to be present, clinically relevant, or mistaken for another problem. The Endocrine Society recommends diagnosing hypogonadism only when compatible symptoms or signs are accompanied by unequivocally and consistently low testosterone, confirmed through repeat fasting morning total testosterone testing; free testosterone is recommended when total testosterone is near the lower limit or when sex hormone-binding globulin may distort interpretation.[14] The American Urological Association uses total testosterone below 300 ng/dL as a reasonable cutoff supporting diagnosis, while requiring two early-morning total testosterone measurements on separate occasions plus symptoms or signs.[15]

The denominator changes sharply depending on whether testosterone deficiency is defined biochemically, symptomatically, or clinically. In the Boston Area Community Health (BACH) Survey, symptomatic androgen deficiency affected 5.6% of men ages 30 to 79, while approximately 24% had total testosterone below 300 ng/dL.[17] The HIM study reported 38.7% biochemical hypogonadism among men ages 45 and older in participating U.S. primary-care practices.[18] Using a deliberately restrictive definition — three sexual symptoms together with total and free testosterone thresholds — the European Male Ageing Study estimated late-onset hypogonadism at approximately 2.1% among European men ages 40 to 79.[19] This is not an estimate of all biochemical or symptomatic testosterone deficiency; it illustrates how sharply prevalence depends on the clinical case definition. Therefore, a low laboratory value, symptomatic testosterone deficiency, claims-defined hypogonadism, and treatment-eligible disease should not be collapsed into one category.

Exhibit 2. Low Testosterone: Multidimensional Impact



Sources: Moskovic et al.; Grabner et al.; Kaltenboeck et al.; Basaria; Ding et al.; Corona et al.; Kupelian et al.; Goldstein et al.

Definition choice materially changes the estimated population. The BACH investigators also projected as many as 6.5 million affected U.S. men ages 30 to 79 in 2025, based on their symptomatic-androgen-deficiency estimate.[17] That projection illustrates potential population scale but does not establish how many men are unevaluated, treatment-eligible, or likely to benefit from therapy.

Diabetes, metabolic syndrome, chronic opioid exposure, HIV/AIDS, pituitary disease, testicular disease, infertility evaluation, chronic corticosteroid exposure, unexplained anemia, bone-density loss, and other guideline-recognized risk histories should prompt targeted testosterone testing because deficiency in these settings can be missed when fatigue, sexual symptoms, mood changes, anemia, or metabolic complications are attributed only to age, weight, chronic illness, medication use, or depression.[14-16] Testing is the entry point for better classification. It allows clinicians to identify men with true hypogonadism, rule out reversible or secondary causes, and avoid leaving deficiency untreated simply because it appears alongside a more familiar diagnosis.

Men most likely to benefit are those with persistent symptoms or signs, confirmed low testosterone on repeat morning testing, and an etiologic evaluation that supports testosterone deficiency as a meaningful clinical diagnosis. For those men, therapy can be linked to specific goals such as improvement in sexual function, correction of unexplained anemia, or management of selected bone-health risks.[14-16] A responsible pathway expands assessment for men whose comorbidities make low testosterone plausible, while reserving treatment for confirmed deficiency and a clear therapeutic rationale.

What the Clinical Evidence Supports

Once the right men are identified for evaluation, the clinical evidence points most clearly to a limited set of treatment goals. The strongest randomized patient-reported signal is sexual function. In the Testosterone Trials, selected older symptomatic men with low testosterone reported improved sexual activity, sexual desire, and erectile-function scores over 12 months.[9] ACP guidance reflects that evidence base: clinicians should discuss testosterone treatment with men who have age-related low testosterone and sexual dysfunction and who want improvement in sexual function, while avoiding initiation for energy, vitality, physical function, or cognition alone.[16]

Anemia is another pathway where the evidence is clinically actionable in selected men. In the TRAVERSE anemia substudy, testosterone corrected anemia more often than placebo among anemic hypogonadal men, including 41.0% versus 27.5% at 6 months and 45.0% versus 33.9% at 12 months.[7] Among testosterone-treated anemic men, greater hemoglobin improvement was associated with better energy scores, although cognition did not improve.[7] For men with unexplained anemia and confirmed testosterone deficiency, these findings support evaluation and treatment discussion as part of a broader diagnostic workup.

Other domains require more careful interpretation. Quality of life, vitality, physical function, bone health, body composition, and metabolic outcomes are clinically relevant, especially because they often drive patient concern and healthcare use. Yet the randomized evidence does not support treating these domains as interchangeable. The Testosterone Trials did not show statistically significant benefit on the primary fatigue/vitality responder endpoint or the Physical Function Trial primary walking-distance responder endpoint, although several continuous measures of vitality, mood, depressive symptoms, and physical function modestly favored testosterone.[9] Low testosterone is also linked to bone-density loss and body-composition changes in clinical reviews and guidelines; treatment can improve selected intermediate endpoints, but those improvements should not be translated into fracture prevention, functional recovery, or avoided costs without direct evidence.[14,20]

The metabolic literature deserves attention because it helps identify men who should be tested, while also creating a risk of overinterpretation. Ding et al. reported lower total testosterone among men with type 2 diabetes and a lower prospective risk of incident diabetes in men in higher testosterone

categories.[21] Corona et al. reported lower total testosterone, free testosterone, and sex hormone-binding globulin among men with type 2 diabetes; small randomized-trial meta-analysis evidence also suggested improvements in HbA1c, fasting glycemia, triglycerides, and fat mass among hypogonadal men with type 2 diabetes.[22] Similar evidence links low testosterone and sex hormone-binding globulin with metabolic syndrome.[23,24] These findings strengthen the case for targeted testosterone assessment in risk-enriched men. They do not establish that testosterone treatment prevents diabetes complications, cardiovascular events, medical utilization, or employer costs.

Cardiovascular evidence should shape safety expectations rather than be used as a promise of cardiovascular benefit. TRAVERSE randomized 5,246 symptomatic men ages 45 to 80 with two fasting testosterone values below 300 ng/dL and preexisting or high cardiovascular risk. Primary major adverse cardiovascular events occurred in 7.0% of testosterone-treated men and 7.3% of placebo-treated men, meeting noninferiority for cardiovascular safety in that selected monitored population.[10] Pulmonary embolism, atrial fibrillation, and acute kidney injury were more frequent in the testosterone group.[10] RCT meta-analysis evidence is broadly consistent with no statistically significant short- to medium-term increase in cardiovascular events or mortality, while long-term and event-specific uncertainties remain.[25]

Monitoring is part of responsible treatment. The Endocrine Society recommends post-initiation clinical evaluation, testosterone and hematocrit measurement, and prostate-risk monitoring when appropriate, with hematocrit above 54% requiring interruption and evaluation.[14] The AUA recommends baseline hemoglobin/hematocrit, baseline PSA in men over 40 years, follow-up testosterone testing, testosterone levels every 6 to 12 months while on therapy, and discussion of cessation after 3 to 6 months when testosterone levels normalize without symptom or sign improvement.[15]

The clinical case is strongest when treatment is tied to a specific, evidence-supported goal: improved sexual function, correction of anemia in appropriately selected men, and careful management of other guideline-recognized indications. The evidence supports selected benefits in selected men. It does not support testosterone therapy as a general wellness intervention.

Exhibit 3. Clinical Evidence by Domain

Domain	Evidence-Supported Interpretation
Sexual function	Strongest randomized patient-reported signal in selected older symptomatic men.
Anemia	TRAVERSE anemia substudy showed greater anemia correction with testosterone.
Vitality & physical function	Primary responder endpoints were mixed or not statistically significant.
Metabolic endpoints	Associations and small RCT meta-analyses suggest selected intermediate changes.
Cardiovascular safety	TRAVERSE showed MACE noninferiority in a selected monitored population.
Monitoring	Guidelines require testosterone, hematocrit, and prostate-risk monitoring when appropriate.
Prostate and BPH safety	FDA's 2026 review reported that available data generally did not show increased prostate-cancer risk and did not demonstrate worsening of mild-to-moderate BPH; long-term and severe-BPH uncertainties remain.

Sources: Lincoff et al.; Bhasin et al.; Mulhall et al.; Snyder et al.; Pencina et al.; Qaseem et al.; Ding et al.; Corona et al.; Kupelian et al.; Hudson et al.; HHS 2026.

Economic Burden and Savings Boundaries

The clinical evidence defines where testosterone treatment can be justified; the economic evidence defines where burden is concentrated and how far savings claims can responsibly go. The economic case should begin with the national cost anchor. Moskovic et al. projected that testosterone deficiency among U.S. men ages 45 to 74 could be associated with $190 billion to $525 billion in inflation-adjusted direct health care expenditures over 20 years.[3] That estimate identifies a large, under-measured men's health burden. It does not establish recoverable savings from therapy.

The most concrete near-term economic pathway is diabetes-risk progression. CDC reported 115.2 million U.S. adults with prediabetes in 2023.[5] Diagnosed diabetes cost the United States an estimated $412.9 billion in 2022, including $306.6 billion in direct medical costs and $106.3 billion in indirect costs.[6] In T4DM, among selected high-risk men without pathological hypogonadism, testosterone plus lifestyle intervention reduced the two-year OGTT-defined diabetes endpoint from 21% to 12%.[4] At population scale, that 9-point absolute difference translates into roughly 90,000 fewer diabetes endpoints per 1 million similar high-risk men reached.

The diabetes scenario can be expressed in budget terms without overstating the evidence. Parker et al. estimated that approximately $12,022 in annual medical expenditures is attributable to diabetes for each person with diagnosed diabetes.[6] One year of delayed diabetes-attributable direct medical cost exposure for 90,000 cases would equal about $1.1 billion. This figure is a direct-cost exposure scenario for prospective evaluation, not a measured treatment-savings result.

Anemia correction offers a second measurable system endpoint. In TRAVERSE, testosterone corrected anemia more often than placebo among anemic hypogonadal men, including 41.0% versus 27.5% at six months.[7] The 13.5-point difference corresponds to about 135,000 additional anemia corrections per 1 million anemic hypogonadal men treated. This is crucial because unresolved anemia can drive repeat laboratory evaluation, clinical follow-up, referral, fatigue-related impairment, and downstream utilization.

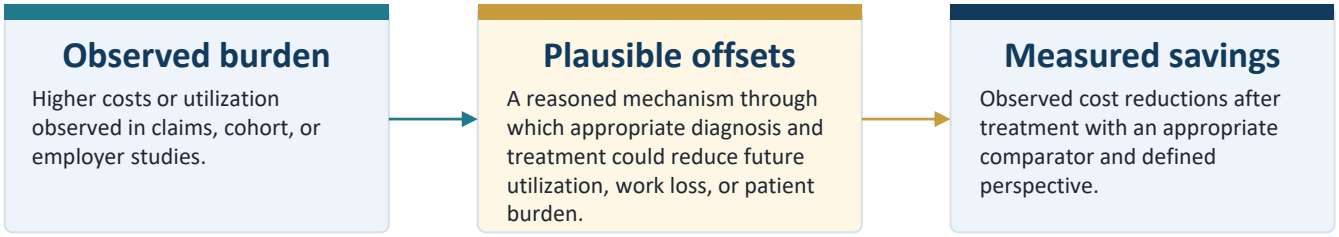
If each additional anemia correction is associated with $500 to $2,000 in lower downstream utilization, the 135,000 additional corrections implied by the TRAVERSE six-month difference would translate to approximately $68 million to $270 million per 1 million anemic hypogonadal men treated. This estimate can be tested in future studies by examining changes in follow-up visits, repeat laboratory testing, referrals, fatigue-related care, and total utilization after anemia correction.

Patient-level claims, employer analyses, and cohort data all show that hypogonadism and low testosterone are associated with higher utilization and cost. Grabner et al. compared 5,777 men with newly diagnosed primary or secondary hypogonadism with 5,777 matched controls in U.S. commercial insurance or Medicare Advantage claims. During 12-month follow-up, mean all-cause direct health care expenditures were $8,813 per patient-year in the hypogonadism cohort versus $5,992 in matched controls, an incremental difference of $2,821 in 2014 U.S. dollars.[1] Because the claims data lacked laboratory values and symptoms for most patients, this estimate is best read as an observed burden benchmark.

Employer and prospective cohort data point in the same direction. Kaltenboeck et al. found higher risk-adjusted direct and indirect employer costs among privately insured employees with claims-defined hypogonadism: $11,957 versus $7,088 per employee-year in 2009 U.S. dollars.[2] Haring et al. found that low serum testosterone was associated with higher outpatient visits and outpatient costs over follow-up, including increases of 28.6% in outpatient visits and 38.0% in costs among men with low testosterone compared with reference testosterone levels.[13] These studies locate burden across medical and workforce channels while reinforcing the need for conservative attribution.

Observed costs should be treated as the beginning of the value question, not the answer. Higher spending, utilization, symptoms, and

Exhibit 4. Value Measurement Framework



Sources: Moskovic et al.; CDC 2026; Parker et al.; Wittert et al.; Pencina et al.; Grabner et al.; Kaltenboeck et al.; Haring et al.; Sullivan et al.

work-loss pressure show where the burden is concentrated among men with low testosterone. Potential offsets become credible only when they are tied to a defined clinical mechanism, such as delayed diabetes progression, anemia correction, symptom improvement, or lower follow-up utilization in selected men. Savings should be counted only when cost reductions are observed after treatment, with an appropriate comparator, defined perspective, product cost, monitoring cost, adverse-event cost, persistence, and response all included.[29]

The next step is prospective measurement in the populations most likely to benefit. Payer and health-system pilots should track diabetes progression, hemoglobin correction, symptom response, utilization, adverse events, discontinuation for nonresponse, work impairment, and total cost over time. A policy that counts prescriptions alone will miss both value and risk.

Access, Persistence, and Distributional Value

Reform should focus on the full pathway that determines whether men receive appropriate testosterone care. Access is not simply a question of whether testosterone products are available. It depends on whether symptoms are recognized, morning testosterone testing is repeated when indicated, etiology is evaluated, treatment decisions are shared and evidence-based, payer rules are navigable, formulations are affordable, monitoring occurs, response is assessed, and therapy is continued only when benefit is present.

Historical utilization patterns show why pathway reform is needed. Layton et al. found that new testosterone testing rose substantially in U.S. claims between 2000 and 2010, but many treatment initiators lacked documented recent testing or repeat testing before initiation.[26] Baillargeon et al. found that androgen therapy use among commercially insured U.S. men ages 40 and older rose from 0.81% in 2001 to 2.91% in 2011, and later work showed testosterone use rising through 2013 before falling through 2016.[27,28] These patterns do not support a simple story of underuse or overuse. They point to inconsistent movement through the right clinical pathway: some men may receive therapy without adequate diagnostic confirmation, while others with symptoms, comorbidities, or confirmed need may never reach appropriate evaluation.

Persistence is central to value because initiation is only the first exposure point. TRAVERSE reported high assigned-product discontinuation in both groups over follow-up, and ACP cited testosterone-treatment discontinuation rates of 30% to 62%.[10,16] A reform agenda should therefore measure more than starts, approvals, or prescriptions filled. It should track whether patients complete monitoring, achieve symptom or clinical response, can afford

the selected formulation, remain on therapy when benefit is present, and discontinue when treatment is not helping. Reauthorization rules can protect patients and payers when they are tied to response and safety monitoring. Used poorly, they can also create avoidable attrition for men who are benefiting but face administrative or cost barriers.

Distributional value should be built into access policy from the start. Men with high symptom burden, unexplained anemia, bone-density concerns, or comorbidity-enriched risk profiles may have the strongest case for evaluation, yet they may face larger barriers when care requires repeated morning laboratory visits, specialist access, prior authorization, cost sharing, or formulation switching. Those barriers matter because they can determine who is tested, who is confirmed, who is treated, who is monitored, and who is left untreated despite clinical need.

A fair reform framework should make appropriate care easier to reach and inappropriate care harder to sustain. That means measuring the pathway end to end: testing, confirmation, authorization, formulation access, monitoring, persistence, discontinuation for nonresponse, and untreated confirmed need. The goal is to have a more reliable system for identifying men who should be evaluated, treating those with confirmed deficiency and evidence-supported goals, and stopping therapy when benefit or safety cannot be demonstrated.

Policy Implications

For health systems, reform begins with making targeted evaluation routine and well documented. Symptoms consistent with testosterone deficiency and selected risk histories should prompt clinical assessment, especially when men present with sexual symptoms, unexplained anemia, metabolic syndrome, prediabetes, type 2 diabetes risk, chronic opioid exposure, pituitary or testicular disease, infertility concerns, bone-density loss, fatigue, or depressed mood. Low testosterone should be confirmed with repeat morning testing, and clinicians should document etiology, contraindications, fertility goals, and the specific treatment objective before therapy begins.

Payers can strengthen appropriate care without turning access management into blunt denial. Coverage criteria should support diagnostic confirmation, appropriate indications, lower-cost formulation choices when clinically suitable, safety monitoring, reauthorization tied to response, and discontinuation when benefit is absent. Affordability and persistence should also be measured directly, because a policy that approves treatment on paper may still fail if patients cannot afford the formulation, complete monitoring, or remain on therapy long enough to benefit.

Employer data identify direct and work-loss burden in claims-defined hypogonadism, while broader productivity evidence includes studies of erectile dysfunction; monetized productivity claims therefore require disciplined methods.[2,30] Employer-facing analyses should define the worker denominator, specify whether wages or total compensation are used, state the attribution factor, account for treatment response and persistence, and avoid double counting across medical costs, disability payments, absenteeism, and productivity estimates. Diabetes progression, anemia correction, work impairment, and total cost should be measured before savings are monetized.

The research agenda should now move toward linked clinical-economic evidence. Decision-ready studies should capture repeat-confirmed diagnosis, treatment initiation, product selection, monitoring completion, treatment response, discontinuation, utilization, patient-reported outcomes, diabetes progression, anemia correction, work outcomes, adverse events, and costs over time. That evidence would allow policymakers, payers, employers, and clinicians to distinguish unmet need from inappropriate use, and observed burden from measurable value.

Conclusion

Testosterone deficiency has credible national economic significance because it clusters with metabolic risk, anemia, patient-centered symptoms, higher utilization, and employer-cost pressures. The evidence supports a serious men's health reform agenda: better targeted evaluation, clearer diagnostic standards, treatment for appropriately selected men, routine monitoring, and access policies that protect patients while reducing unnecessary attrition.

The June 2026 request may reduce one regulatory concern, but it does not eliminate clinical uncertainty or the need for risk assessment and monitoring.[8] Recognition, diagnostic follow-through, access, affordability, and persistence remain important policy barriers. The next question is whether targeted case-finding and accountable treatment improve patient outcomes and reduce net cost when measured prospectively.

Current evidence supports neither universal testosterone therapy nor definitive systemwide savings claims. It supports a more focused policy position: untreated or inadequately managed testosterone deficiency can carry substantial burden; selected men can experience meaningful clinical benefits; and economic value is most plausible when care is specific, measured, and accountable.

The strongest reform pathway confirms diagnosis, selects patients carefully, states treatment goals, tracks response and persistence, counts product and monitoring costs, measures safety, and evaluates diabetes progression, anemia correction, utilization, work impairment, and total cost over time. Observed burden should motivate action, and measured outcomes should determine the value claim.

Bibliography

1. Grabner M, Bodhani A, Khandelwal N, Palli S, Bonine N, Khera M. Clinical characteristics, health care utilization and costs among men with primary or secondary hypogonadism in a US commercially insured population. *Journal of Sexual Medicine*. 2017;14(1):88-97. https://doi.org/10.1016/j.jsxm.2016.10.012

2. Kaltenboeck A, Foster S, Ivanova J, et al. The direct and indirect costs among U.S. privately insured employees with hypogonadism. *Journal of Sexual Medicine*. 2012;9(9):2438-2447. https://doi.org/10.1111/j.1743-6109.2012.02810.x

3. Moskovic DJ, Araujo AB, Lipshultz LI, Khera M. The 20-year public health impact and direct cost of testosterone deficiency in U.S. men. *Journal of Sexual Medicine*. 2013;10(2):562-569. https://doi.org/10.1111/j.1743-6109.2012.02944.x

4. Wittert G, Bracken K, Robledo KP, et al. Testosterone treatment to prevent or revert type 2 diabetes in men enrolled in a lifestyle programme (T4DM): a randomised, double-blind, placebo-controlled, 2-year, phase 3b trial. *The Lancet Diabetes & Endocrinology*. 2021;9(1):32-45. https://doi.org/10.1016/S2213-8587(20)30367-3

5. Centers for Disease Control and Prevention. *National Diabetes Statistics Report*. Updated January 21, 2026. Accessed July 6, 2026. https://www.cdc.gov/diabetes/php/data-research/index.html

6. Parker ED, Lin J, Mahoney T, et al. Economic costs of diabetes in the U.S. in 2022. *Diabetes Care*. 2024;47(1):26-43. https://doi.org/10.2337/dci23-0085

7. Pencina KM, Travison TG, Artz AS, et al. Efficacy of testosterone replacement therapy in correcting anemia in men with hypogonadism: a randomized clinical trial. *JAMA Network Open*. 2023;6(10):e2340030. https://doi.org/10.1001/jamanetworkopen.2023.40030

8. U.S. Department of Health and Human Services. *HHS Announces Requested Updates to Testosterone Therapy Product Labels*. Press release. June 18, 2026. Accessed July 6, 2026. https://www.hhs.gov/press-room/fda-requests-updates-testosterone-therapy-labeling.html

9. Snyder PJ, Bhasin S, Cunningham GR, et al. Effects of testosterone treatment in older men. *New England Journal of Medicine*. 2016;374(7):611-624. https://doi.org/10.1056/NEJMoa1506119

10. Lincoff AM, Bhasin S, Flevaris P, et al. Cardiovascular safety of testosterone-replacement therapy. *New England Journal of Medicine*. 2023;389(2):107-117. https://doi.org/10.1056/NEJMoa2215025

11. U.S. Food and Drug Administration. *FDA Issues Class-Wide Labeling Changes for Testosterone Products*. Published February 28, 2025. Accessed July 6, 2026. https://www.fda.gov/drugs/drug-alerts-and-statements/fda-issues-class-wide-labeling-changes-testosterone-products

12. U.S. Food and Drug Administration. *Testosterone Information*. Updated June 23, 2026. Accessed July 6, 2026. https://www.fda.gov/drugs/postmarket-drug-safety-information-patients-and-providers/testosterone-information

13. Haring R, Baumeister SE, Völzke H, et al. Prospective association of low serum total testosterone levels with health care utilization and costs in a population-based cohort of men. *International Journal of Andrology*. 2010;33(6):800-809. https://doi.org/10.1111/j.1365-2605.2009.01029.x

14. Bhasin S, Brito JP, Cunningham GR, et al. Testosterone therapy in men with hypogonadism: an Endocrine Society clinical practice guideline. *Journal of Clinical Endocrinology & Metabolism*. 2018;103(5):1715-1744. https://doi.org/10.1210/jc.2018-00229

15. Mulhall JP, Trost LW, Brannigan RE, et al. Evaluation and management of testosterone deficiency: AUA guideline. *The Journal of Urology*. 2018;200(2):423-432. https://doi.org/10.1016/j.juro.2018.03.115

16. Qaseem A, Horwitch CA, Vijan S, Etxeandia-Ikobaltzeta I, Kansagara D. Testosterone treatment in adult men with age-related low testosterone: a clinical guideline from the American College of Physicians. *Annals of Internal Medicine*. 2020;172(2):126-133. https://doi.org/10.7326/M19-0882

17. Araujo AB, Esche GR, Kupelian V, et al. Prevalence of symptomatic androgen deficiency in men. *Journal of Clinical Endocrinology & Metabolism*. 2007;92(11):4241-4247. https://doi.org/10.1210/jc.2007-1245

18. Mulligan T, Frick MF, Zuraw QC, Stemhagen A, McWhirter C. Prevalence of hypogonadism in males aged at least 45 years: the HIM study. *International Journal of Clinical Practice*. 2006;60(7):762-769. https://doi.org/10.1111/j.1742-1241.2006.00992.x

19. Wu FCW, Tajar A, Beynon JM, et al. Identification of late-onset hypogonadism in middle-aged and elderly men. *New England Journal of Medicine*. 2010;363(2):123-135. https://doi.org/10.1056/NEJMoa0911101

20. Basaria S. Male hypogonadism. *The Lancet*. 2014;383(9924):1250-1263. https://doi.org/10.1016/S0140-6736(13)61126-5

21. Ding EL, Song Y, Malik VS, Liu S. Sex differences of endogenous sex hormones and risk of type 2 diabetes: a systematic review and meta-analysis. *JAMA*. 2006;295(11):1288-1299. https://doi.org/10.1001/jama.295.11.1288

22. Corona G, Monami M, Rastrelli G, et al. Type 2 diabetes mellitus and testosterone: a meta-analysis study. *International Journal of Andrology*. 2011;34(6 Pt 1):528-540. https://doi.org/10.1111/j.1365-2605.2010.01117.x

23. Corona G, Monami M, Rastrelli G, et al. Testosterone and metabolic syndrome: a meta-analysis study. *Journal of Sexual Medicine*. 2011;8(1):272-283. https://doi.org/10.1111/j.1743-6109.2010.01991.x

24. Kupelian V, Page ST, Araujo AB, Travison TG, Bremner WJ, McKinlay JB. Low sex hormone-binding globulin, total testosterone, and symptomatic androgen deficiency are associated with development of the metabolic syndrome in nonobese men. *Journal of Clinical Endocrinology & Metabolism*. 2006;91(3):843-850. https://doi.org/10.1210/jc.2005-1326

25. Hudson J, Cruickshank M, Quinton R, et al. Adverse cardiovascular events and mortality in men during testosterone treatment: an individual patient and aggregate data meta-analysis. *The Lancet Healthy Longevity*. 2022;3(6):e381-e393. https://doi.org/10.1016/S2666-7568(22)00096-4

26. Layton JB, Li D, Meier CR, et al. Testosterone lab testing and initiation in the United Kingdom and the United States, 2000 to 2011. *Journal of Clinical Endocrinology & Metabolism*. 2014;99(3):835-842. https://doi.org/10.1210/jc.2013-3570

27. Baillargeon J, Urban RJ, Ottenbacher KJ, Pierson KS, Goodwin JS. Trends in androgen prescribing in the United States, 2001 to 2011. *JAMA Internal Medicine*. 2013;173(15):1465-1466. https://doi.org/10.1001/jamainternmed.2013.6895

28. Baillargeon J, Kuo YF, Westra JR, Urban RJ, Goodwin JS. Testosterone prescribing in the United States, 2002-2016. *JAMA*. 2018;320(2):200-202. https://doi.org/10.1001/jama.2018.7999

29. Sullivan SD, Mauskopf JA, Augustovski F, et al. Budget impact analysis-principles of good practice: report of the ISPOR 2012 Budget Impact Analysis Good Practice II Task Force. *Value in Health*. 2014;17(1):5-14. https://doi.org/10.1016/j.jval.2013.08.2291

30. Goldstein I, Goren A, Li VW, Maculaitis MC, Tang WY, Hassan TA. The association of erectile dysfunction with productivity and absenteeism in eight countries globally. *International Journal of Clinical Practice*. 2019;73(11):e13384. https://doi.org/10.1111/ijcp.13384