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AGED GARLIC EXTRACT AT THE ENDOCRINE–VASCULAR INTERFACE

Can S-allyl-L-cysteine reposition a cardiovascular botanical as a dual-domain ingredient for men's healthy ageing?

A critical appraisal of emerging testosterone data, established vascular evidence, category benchmarks and the evidence still required before "superiority" claims are scientifically defensible.

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ABSTRACT

Aged garlic extract (AGE) has a substantially more mature human evidence base in cardiovascular physiology than in endocrine health. Randomized trials have examined blood pressure, central haemodynamics, endothelial function, arterial stiffness and coronary calcification, while the steroidogenic hypothesis rests principally on S-allyl-L-cysteine (SAC) mechanistic work and a newly disclosed, manufacturer-sponsored 60-day randomized trial of a proprietary 1% SAC-standardized AGE. The latter reportedly increased total and free testosterone and improved Aging Males' Symptoms (AMS), International Index of Erectile Function (IIEF), fatigue and quality-of-life scores relative to placebo. However, the full clinical dataset has not yet been located in a peer-reviewed publication, and key information required to judge clinical relevance—baseline hormone concentrations, repeated morning sampling, assay methodology, prespecified endpoints, dispersion, multiplicity control, analysis population and trial registration—is not publicly available. The most defensible scientific interpretation is therefore not that AGE has already outperformed established category ingredients, but that it may represent an unusually coherent endocrine–vascular research platform. This review critically compares that emerging signal with 2026 meta-analytic evidence for ashwagandha and fenugreek, earlier evidence for Eurycoma longifolia, and the larger AGE vascular literature. It also identifies the next experiments required to establish whether the apparent dual-domain profile is real, reproducible and clinically meaningful.

Keywords: *aged garlic extract; S-allyl-L-cysteine; testosterone; Leydig cell; PKA; CYP11A1; endothelial function; nitric oxide; arterial stiffness; erectile physiology; men's health; healthy ageing.*

EDITORIAL NOTE

AGE is already credible as a vascular-support research platform; it is only emerging as a testosterone-support platform. The dual-domain hypothesis is scientifically attractive, but "rivals" or "surpasses" category mainstays is premature without peer-reviewed replication and direct comparator trials.

1. Why this question matters now

The testosterone-support category has matured into a crowded market dominated by a small set of recognizable botanicals. Ashwagandha, fenugreek and Eurycoma longifolia (tongkat ali) are repeatedly positioned around testosterone, vitality, sexual health, exercise performance or "male optimisation". Yet the clinical evidence behind those narratives is uneven, extract-specific and population-dependent. Against that background, a standardized aged garlic extract is scientifically interesting precisely because its legacy is not endocrine marketing: AGE has been studied primarily in vascular and cardiometabolic contexts.

The emerging proposition is therefore different from the usual "new testosterone booster" story. SAC, a characteristic water-soluble organosulfur compound enriched during garlic ageing, has a plausible direct steroidogenic signal in Leydig-cell models. At the same time, AGE has human randomized evidence involving blood pressure, endothelial responsiveness, arterial stiffness and central haemodynamics. If both domains can be demonstrated in the same individuals with the same chemically characterized extract, the value proposition becomes a mechanistic convergence model rather than a single-biomarker claim.

That hypothesis is compelling. It is not yet proven. The distinction is central to a publication that aims to be scientifically durable rather than merely timely.

"The important question is not whether AGE is a stronger testosterone booster. It is whether one standardized organosulfur platform can reproducibly influence both steroidogenesis and vascular physiology."

2. AGE is not simply "garlic in a capsule"

The term garlic is chemically imprecise. Fresh garlic, dehydrated garlic powders, garlic oils, aged garlic extract and aged black garlic differ materially in processing, sulfur chemistry, marker compounds and pharmacological profile. This matters because biological findings cannot be freely transferred from one preparation to another.

Fresh garlic contains alliin, which can be converted by alliinase to the reactive thiosulfinate allicin after tissue disruption. Allicin is unstable and undergoes further transformation. During controlled ageing, the chemical profile shifts toward more stable water-soluble organosulfur compounds, of which S-allyl-L-cysteine (SAC) is among the best characterized and is commonly used as a standardization marker. This relative chemical stability is one reason SAC-standardized AGE has been attractive for clinical research.

A critical formulation principle follows: an extract standardized to SAC should not be treated as interchangeable with another AGE merely because the label says "aged garlic", and neither should aged black garlic automatically be equated with classical AGE. Marker concentration, process, co-constituents and exposure all matter.

The dose-standardization issue

The proprietary AGE currently promoted for men's health is stated by its manufacturer to provide 200 mg/day at 1% SAC, corresponding nominally to approximately 2 mg SAC/day. Several cardiovascular AGE trials have used total extract doses around 960–1,200 mg/day with SAC intakes in the approximate 1.2–2.4 mg/day range. The superficial similarity in SAC exposure is mechanistically provocative, but it does not establish bioequivalence: the surrounding phytochemical matrix and manufacturing process remain relevant.

3. The steroidogenic hypothesis: what SAC actually does in preclinical models

The strongest mechanistic evidence comes from the 2021 study by Rana and colleagues. SAC was administered as a single intraperitoneal dose of 50 mg/kg to male BALB/c mice, and parallel experiments were performed in mouse testis-derived I-10 cells. SAC increased testosterone in plasma and testicular tissue without increasing circulating luteinizing hormone (LH). In I-10 cells, SAC increased testosterone secretion, enhanced phosphorylated protein kinase A (p-PKA), and increased Cyp11a1 mRNA, while StAR mRNA was not significantly changed under the reported conditions.

This pattern is biologically coherent with a direct testicular effect. Leydig-cell steroidogenesis is classically driven through LH receptor signalling, cAMP and PKA activation, with subsequent regulation of cholesterol transport and mitochondrial conversion of cholesterol to pregnenolone by CYP11A1. An intervention that increases PKA activation and Cyp11a1 expression can therefore be situated within a plausible steroidogenic pathway.

However, three limitations are non-negotiable. First, the animal exposure used an intraperitoneal route rather than oral supplementation. Second, the dose cannot be linearly translated to a 200 mg/day human botanical formulation. Third, a tumour-derived mouse Leydig-cell line is a mechanistic model, not a human endocrine system. The work justifies a hypothesis; it does not validate a human dose or clinical claim.

Figure 1. Working endocrine–vascular convergence model

INPUT	MOLECULAR NODE	SIGNALLING	PHYSIOLOGY	POTENTIAL OUTCOME
SAC-standardized AGE	S-allyl-L-cysteine	↑ p-PKA ↑ Cyp11a1	Leydig-cell steroidogenesis	**Testosterone signal (emerging human evidence)**
AGE organosulfur matrix	SAC + related sulfur chemistry	NO / H_2S biology ACE-related pathways*	Vascular tone endothelial function arterial stiffness	**Blood-pressure and vascular endpoints (human RCT evidence)**

**Mechanisms proposed across garlic/AGE literature; strength of evidence varies by preparation and experimental system. Arrows denote a working model, not a demonstrated single causal chain in humans.*

4. The new human testosterone signal: promising, but not yet mature evidence

The central new data come from a manufacturer-sponsored randomized, double-blind, placebo-controlled study described in a 2026 partner feature and on the ingredient manufacturer's website. Fifty-two men aged 25–60 years with symptoms identified using the AMS scale and suboptimal erectile-function scores by IIEF reportedly received either 200 mg/day of a proprietary 1% SAC-standardized AGE or maltodextrin placebo for 60 days.

According to the publicly displayed manufacturer chart, percentage changes from baseline at day 60 were reported as approximately +23.5% for total testosterone and +27.0% for free testosterone in the AGE arm, versus -5.8% and -3.9%, respectively, in placebo. The same chart reports favourable changes in AMS, IIEF, fatigue and SF-36 quality-of-life measures. The promotional report states that between-group differences were statistically significant ($p<0.05$), that DHEA did not differ significantly, and that no adverse events or notable clinical chemistry changes were observed.

CRITICAL EVIDENCE LIMITATION

These percentages are manufacturer-reported promotional data, not effect estimates extracted from a peer-reviewed paper. Until the full trial report is available, they should not be compared numerically with meta-analytic effects for other botanicals.

What is still missing from the public record?

- Baseline total testosterone, free testosterone and SHBG values by randomized group, together with dispersion and clinically interpretable absolute changes.
- Confirmation that testosterone was measured in standardized morning fasting samples and, ideally, repeated on separate mornings to reduce biological and analytical variability.
- Assay methodology, including whether total testosterone was measured by LC–MS/MS or immunoassay and how free testosterone was measured or calculated.
- Prospective trial registration, protocol-defined primary and secondary endpoints, sample-size calculation and multiplicity strategy.
- Full CONSORT flow, attrition, adherence, missing-data handling and the exact analysis population.
- Independent replication, longer duration, and evidence in men with biochemically confirmed low testosterone rather than symptom-selected participants alone.

These omissions do not invalidate the trial. They determine the level of confidence that can currently be assigned to it.

Why AMS and IIEF are useful but insufficient

AMS and IIEF can capture clinically relevant symptom domains, but neither instrument establishes hypogonadism. Symptoms such as fatigue, low libido, reduced well-being and erectile difficulties are nonspecific and can reflect sleep disorders, depression, obesity, vascular disease, medications, relationship factors, diabetes or other endocrine conditions. Contemporary Endocrine Society guidance continues to define male hypogonadism by the combination of compatible symptoms/signs and unequivocally and consistently low testosterone concentrations, with confirmation using repeated morning measurements. In July 2026 the Society reiterated that symptoms alone are not diagnostic.

5. The vascular evidence: AGE enters the discussion from a much stronger position

The vascular literature is where AGE has genuine depth. It is not free of limitations—sample sizes are often modest, preparations vary, several studies come from overlapping research groups, and surrogate endpoints predominate—but multiple randomized human trials exist.

Endothelial function

Williams et al. studied 15 men with angiographically documented coronary artery disease in a randomized placebo-controlled crossover design. Short-term AGE supplementation increased brachial artery flow-mediated dilation (FMD) by 44% relative to baseline, with end-of-treatment FMD significantly higher than after placebo after adjustment. The result is small-study evidence, but it is directly relevant to endothelial responsiveness.

Blood pressure and central haemodynamics

In a 2010 double-blind RCT involving 50 patients with treated but uncontrolled hypertension, 960 mg/day AGE containing 2.4 mg SAC lowered systolic blood pressure by an average 10.2 ± 4.3 mmHg versus control among participants with baseline systolic blood pressure ≥140 mmHg. A subsequent 12-week AGE at Heart trial in 88 participants used 1.2 g/day AGE containing 1.2 mg SAC and found an average placebo-adjusted systolic reduction of 5.0 ± 2.1 mmHg across the cohort, with larger reductions in responders.

The GarGIC trial, also using 1.2 g/day AGE containing 1.2 mg SAC for 12 weeks, reported placebo-adjusted changes of approximately -10.0 mmHg systolic and -5.4 mmHg diastolic in 49 completers, together with improvements in central systolic pressure, pulse pressure and age/sex-adjusted pulse-wave velocity. These studies support a vascular signal, although they should not be interpreted as evidence that AGE substitutes for antihypertensive therapy.

Arterial elasticity

A separate randomized double-blind placebo-controlled study of 57 participants with slightly elevated blood pressure reported a 21.6% improvement in EndoPAT-derived augmentation index adjusted to 75 beats/minute (AI75) in the AGE group over 12 weeks. This again points toward vascular compliance rather than simply a clinic blood-pressure effect.

Coronary calcification and atherosclerotic progression

Several small randomized studies have examined coronary artery calcification (CAC), sometimes using AGE alone and sometimes within combination formulations. A 2020 one-year double-blind trial randomized 104 participants at elevated Framingham risk to 2,400 mg/day AGE or placebo and reported findings favouring AGE for CAC progression alongside selected metabolic/inflammatory endpoints. A 2023 systematic review of interventions for cardiovascular calcification identified six AGE trials and described a consistent attenuation signal, while still emphasizing that the studies were small and generally short. The appropriate conclusion is therefore “promising surrogate vascular evidence”, not prevention of cardiovascular events.

A 2026 evidence-integrity update that should not be ignored

An AGE-specific blood-pressure systematic review and meta-analysis published in late 2024 was formally retracted in July 2026 after the journal reported substantial plagiarism from an unpublished manuscript and loss of confidence in the originality and provenance of the work. It should therefore be excluded from current evidence synthesis. This is not a peripheral editorial detail: it materially affects any 2026 publication that claims to be up to date. The vascular case for AGE should rest on the primary RCTs and non-retracted syntheses, not on the withdrawn paper.

6. Why vascular function matters in a men’s-health article

Male sexual physiology cannot be reduced to testosterone. Penile erection is fundamentally a neurovascular event dependent on intact endothelial signalling, nitric-oxide bioavailability, smooth-muscle relaxation, arterial inflow and veno-occlusive function. Testosterone can modulate aspects of sexual desire and may interact with vascular biology, but erectile function is not a simple proxy for serum androgen concentration.

That distinction makes AGE unusually interesting. A formulation that modestly influences androgen biology while also improving objective vascular physiology could plausibly affect male well-being through more than one pathway. Conversely, an improvement in IIEF cannot be assumed to prove testosterone mediation. The endocrine and vascular pathways should be measured simultaneously before causality is assigned.

Nitric oxide, hydrogen sulfide and ACE-related signalling

Garlic-derived sulfur chemistry has been linked experimentally to nitric oxide (NO), hydrogen sulfide (H_2S), redox signalling and angiotensin-converting-enzyme-related pathways. These mechanisms provide plausible routes to vasodilation and improved vascular tone. A 2023 randomized triple-blind trial of an optimized aged garlic preparation in grade I hypertension reported blood-pressure effects together with increased circulating NO and reduced ACE activity. However, “aged garlic” preparations are not chemically identical, and mechanistic findings must be mapped to the exact extract studied.

7. Does AGE rival the category mainstays? A benchmarked answer

The fairest comparison is not marketing visibility but evidence maturity. On testosterone specifically, AGE currently has less published human evidence than several established botanicals. On vascular physiology, the reverse may be true: AGE brings a broader cardiovascular dossier than many testosterone-positioned ingredients. That asymmetry is precisely why the product concept is interesting.

Ingredient	Human testosterone evidence	Representative 2026/2022 synthesis	Free-T signal	Vascular dossier	Current interpretation
SAC-standardized AGE	One newly disclosed 60-day RCT (n=52), not yet located as a full peer-reviewed paper	Manufacturer-reported human trial + 2021 SAC mechanistic study	Reported positive; public raw/absolute data unavailable	Multiple RCTs on BP, FMD, stiffness/central haemodynamics; CAC literature	Highly interesting dual-domain candidate; testosterone evidence remains preliminary
Ashwagandha	Multiple RCTs across male populations	2026 meta-analysis: 23 hormone RCTs, 1,706 participants; male testosterone MD +57.43 ng/dL	Variable by trial; not the sole basis of the meta-analysis	Not a defining category strength	More mature endocrine evidence than AGE, but extract/population heterogeneity remains
Fenugreek	Multiple RCTs, several proprietary extracts	2026 meta-analysis: TT SMD 0.25 (95% CI 0.02–0.48), 6 studies/385 participants	No stable pooled benefit; SMD 0.08, CI crosses null	Not a defining category strength	Small favourable TT signal; GRADE certainty very low
Eurycoma longifolia	Several trials in healthy and low-T men	2022 meta-analysis: 5 RCTs; TT SMD 1.352 (95% CI 0.565–2.138)	Less consistently characterized	Not a defining category strength	Promising, but small evidence base and publication-bias concerns

Ashwagandha: the current benchmark is stronger than many formulations imply

A 2026 systematic review and meta-analysis in Planta Medica included 23 randomized placebo-controlled trials encompassing 1,706 participants across hormonal endpoints. In men, ashwagandha increased testosterone by a pooled mean difference of 57.43 ng/dL. The authors nevertheless emphasized heterogeneity and limited data for some outcomes. This constitutes a materially more mature published endocrine evidence base than AGE currently possesses.

Fenugreek: a useful cautionary example

The 2026 Frontiers in Nutrition meta-analysis is particularly instructive because it applied RoB 2 and GRADE. Thirteen studies were included; six trials (385 participants) contributed to total testosterone and produced a small favourable SMD of 0.25 (95% CI 0.02–0.48). Five studies contributed to free testosterone, where the pooled estimate crossed the null (SMD 0.08; 95% CI -0.48 to 0.63) with substantial heterogeneity. Both testosterone outcomes were graded very-low certainty. Thus, longevity in the marketplace does not guarantee high-certainty endocrine evidence.

Eurycoma longifolia: positive pooled signal, limited corpus

A 2022 systematic review and meta-analysis identified nine studies and included five RCTs in the pooled analysis. Total testosterone favoured Eurycoma longifolia (SMD 1.352; 95% CI 0.565–2.138), including a hypogonadal subgroup, but the authors called for more research and reported evidence of publication bias by Begg's test. This is a meaningful signal, but not a definitive benchmark of superiority.

Why "surpass" is currently the wrong scientific verb

No direct randomized trial has compared a SAC-standardized AGE with ashwagandha, fenugreek or Eurycoma longifolia using harmonized testosterone assays and the same population. Cross-trial comparisons are invalidated by different baseline testosterone values, age, health status, extract chemistry, duration, assay methods, endpoints and statistical models. Even apparently impressive percentage changes can be misleading when the baseline denominator is low or when regression to the mean is present.

The defendable formulation is therefore: AGE may be differentiated by its dual-domain endocrine–vascular rationale. Superiority remains an empirical question.

8. An evidence hierarchy for AGE in men's health

Claim domain	Evidence type	Representative evidence	Confidence now	What would upgrade confidence
Direct steroidogenesis	Cell + mouse	SAC → p-PKA, Cyp11a1, testosterone; LH unchanged	Moderate mechanistic plausibility	Human mechanistic biomarkers; dose-response; oral PK/PD linkage
Human testosterone	Small RCT disclosed by sponsor	200 mg/day, 60 days, n=52; TT/FT reported higher vs placebo	Low-to-moderate, pending full publication	Peer-reviewed full report; replication; longer study; confirmed low-T cohort
Symptoms / sexual function	Same sponsor RCT	AMS, IIEF, FSS, SF-36 reportedly improved	Low-to-moderate	Prespecified endpoints; MCID analysis; independent replication
Blood pressure	Multiple RCTs	960–1,200 mg/day AGE; uncontrolled hypertension populations	Moderate	Larger multicentre studies; standardized extract; ambulatory BP
Endothelial / stiffness	Small RCTs	FMD; central haemodynamics; PWV; EndoPAT AI75	Moderate but surrogate-based	Larger studies with reproducible vascular phenotyping
Cardiovascular events	None adequate	Surrogate endpoints only	Insufficient	Long-duration outcomes trial—not currently realistic for nutraceutical development

9. The endocrine measurements that future AGE trials must get right

Testosterone trials are technically easy to overinterpret because the biomarker itself is variable. A robust next-generation AGE study should follow endocrine best practice rather than supplement-industry convention.

Sampling and assay

- Total testosterone should be measured on at least two separate mornings at baseline, preferably fasting and at a consistent clock time.
- LC–MS/MS is preferred where feasible for total testosterone; the assay and calibration system should be fully disclosed.
- SHBG and albumin should be measured, with free testosterone assessed by equilibrium dialysis or calculated using a validated equation when appropriate.
- LH and FSH are needed to distinguish testicular from hypothalamic–pituitary effects; estradiol, prolactin and DHEA-S provide additional interpretive context.

Population selection

A symptom-enriched population is commercially relevant but biologically noisy. A decisive trial should either recruit men with confirmed low or low-normal testosterone using repeated measurements, or prospectively stratify participants by baseline androgen status. Obesity, sleep apnoea, diabetes, alcohol intake, medications, resistance training, caloric restriction and recent illness should be controlled or prespecified because each can materially affect testosterone.

Clinical relevance

The key endpoint is not simply $p<0.05$. Absolute hormone change, confidence intervals, proportion of participants crossing clinically relevant thresholds, within-person variability, and concordance between biochemical and symptomatic outcomes matter. If AMS or IIEF is used, minimal clinically important differences and multiplicity control should be prespecified.

10. The decisive experiment: an integrated endocrine–vascular trial

The study that could genuinely reposition AGE is not another small testosterone trial. It is a multidomain randomized trial designed to test whether endocrine and vascular changes co-occur in the same participants.

Design element	Recommended specification
Population	Men approximately 35–65 years with symptoms plus reproducibly low-normal or mildly reduced morning testosterone; exclude untreated major endocrine disease.
Design	Multicentre, randomized, double-blind, placebo-controlled; 16–24 weeks; prespecified stratification by baseline testosterone and cardiometabolic risk.
Intervention	Chemically characterized SAC-standardized AGE; include full fingerprint and batch release data; consider one dose matching the commercial 200 mg/day formulation and one mechanistically informative higher exposure if justified.
Primary endocrine endpoint	Change in repeated-morning total testosterone with SHBG/free-T analysis.
Primary vascular endpoint	Brachial FMD or validated PWV/central haemodynamic measure, ideally with ambulatory blood pressure.
Secondary endpoints	LH, FSH, estradiol, DHEA-S, NO-related biomarkers, AMS, IIEF, fatigue, SF-36, body composition, grip strength, sleep and activity.
Safety	CBC, liver/renal chemistry, blood pressure, bleeding events, medication interactions, PSA where age/clinical context makes this appropriate.
Statistics	Prospective registration; hierarchical or multiplicity-controlled endpoint plan; responder analysis; mediation/exploratory correlation between hormone and vascular changes.
Comparator phase	After placebo-confirmatory replication, consider head-to-head comparison against one standardized category benchmark—not three heterogeneous botanicals at once.

11. Safety and translational boundaries

Garlic supplements are generally well tolerated, but “natural” should not be equated with pharmacologically inert. Gastrointestinal symptoms and odour are common concerns for some preparations. NCCIH notes a potential increase in bleeding risk with garlic supplements, particularly in people taking anticoagulants or aspirin or around surgery. Although individual AGE trials have not consistently shown clinically important bleeding problems, medication context remains relevant.

The second boundary is clinical substitution. A supplement trial in men with nonspecific symptoms is not evidence for treating pathological hypogonadism. Current endocrine guidance requires symptoms/signs plus consistently low testosterone, confirmation with repeated morning testing, and investigation of cause. Men with true hypogonadism, pituitary disease, infertility concerns or significant sexual dysfunction require clinical assessment rather than a supplement-only pathway.

12. Regulatory translation: science, substantiation and claims are different questions

A scientific paper may discuss biological effects that cannot be used verbatim in commercial communication. In the European Union, a positive clinical trial does not automatically create an authorized health claim for a botanical ingredient. Formulators must distinguish scientific hypothesis, evidence substantiation, food-supplement positioning and legally permitted consumer-facing wording. Where a finished product contains nutrients with authorized claims—zinc, for example, has an authorized EU claim concerning maintenance of normal testosterone levels in the blood—those claims apply only when their specific conditions of use are met and cannot be used to imply that AGE itself has an authorized testosterone claim.

This separation is especially important if AGE is positioned as a branded men's-health ingredient. The scientifically strongest narrative at present is "emerging research at the endocrine–vascular interface", not "clinically proven to outperform established testosterone ingredients".

13. Strategic implications for product development

From an R&D perspective, AGE has three attractive features. First, SAC provides a measurable standardization anchor. Second, the active dose proposed for the proprietary men's-health extract is formulation-friendly. Third, the vascular literature offers a differentiated scientific platform that is not shared by many conventional testosterone-positioned botanicals.

The opportunity is therefore to design a male healthy-ageing system around vascular integrity, endocrine resilience and functional outcomes rather than to compete in the narrow "boost testosterone" vocabulary. In scientific terms, that is a better hypothesis; in product-development terms, it also creates more space for rational combinations with nutrients or bioactives chosen for complementary rather than redundant mechanisms.

However, formulation strategy should not outrun evidence. Until the proprietary testosterone trial is fully published and independently replicated, the ingredient should be treated internally as an emerging endocrine technology with a stronger established vascular heritage—not as a proven category leader.

14. Conclusion

Aged garlic extract has earned scientific attention in men's health for a reason that is more substantive than novelty. SAC has a plausible direct steroidogenic mechanism, and a new randomized human trial reportedly shows increases in total and free testosterone together with improvements in symptom, sexual-function, fatigue and quality-of-life measures. Independently, AGE has a multi-study vascular dossier involving blood pressure, endothelial function, arterial elasticity, central haemodynamics and coronary-calcification surrogates.

But the two evidence streams are not equally mature. The vascular evidence is established enough to justify continued translational work. The testosterone evidence remains early because the pivotal proprietary trial is not yet available as a full peer-reviewed publication and has not been independently replicated. By contrast, ashwagandha already has a 2026 meta-analytic endocrine dataset; fenugreek has a new GRADE-assessed meta-analysis showing only a small and very-low-certainty total-testosterone signal; and Eurycoma longifolia has a positive but limited meta-analytic literature.

The most defensible scientific conclusion is therefore precise: AGE does not yet have evidence to claim superiority over established testosterone botanicals. It may, however, have the potential to become more differentiated than a conventional testosterone botanical if future trials demonstrate that standardized AGE can simultaneously improve androgen biology and objective vascular physiology in the same men. That is the experiment worth doing—and the story worth publishing.

KEY TAKEAWAYS

- SAC has a plausible direct steroidogenic mechanism via PKA/CYP11A1 signalling in preclinical models.
- A 60-day, n=52 human AGE trial reports favourable testosterone and men's-health outcomes, but the complete peer-reviewed dataset is still needed.
- AGE's vascular evidence is substantially more mature than its endocrine evidence.
- 2026 data strengthen the comparator context: ashwagandha has a positive meta-analytic male testosterone effect; fenugreek's pooled signal is small and very-low certainty.
- No evidence currently supports a scientific claim that AGE "surpasses" established botanicals.
- The highest-value next study measures testosterone and vascular function together in a registered, multicentre RCT.

15. References

Endocrine Society. (2018). Testosterone therapy in men with hypogonadism: An Endocrine Society clinical practice guideline. Journal of Clinical Endocrinology & Metabolism, 103(5), 1715–1744. https://doi.org/10.1210/jc.2018-00229

Endocrine Society. (2026, July 16). Statement on testosterone replacement therapy. Endocrine Society.

Fornalik, M., Malkiewicz, A., Adamczak, D., Nawrocki, F., Zielinska, A., & Mondal, S. A. (2026). Hormonal modulation with Withania somnifera: Systematic review and meta-analysis of randomized-controlled trials. Planta Medica, 92(8), 790–805. https://doi.org/10.1055/a-2802-8363

Gruenwald, J., Bongartz, U., Bothe, G., & Uebelhack, R. (2020). Effects of aged garlic extract on arterial elasticity in a placebo-controlled clinical trial using EndoPAT technology. Experimental and Therapeutic Medicine, 19(2), 1490–1499. https://doi.org/10.3892/etm.2019.8378

Leisegang, K., Finelli, R., Sikka, S. C., & Panner Selvam, M. K. (2022). Eurycoma longifolia (Jack) improves serum total testosterone in men: A systematic review and meta-analysis of clinical trials. Medicina, 58(8), 1047. https://doi.org/10.3390/medicina58081047

National Center for Complementary and Integrative Health. (2026). Garlic: Usefulness and safety. U.S. National Institutes of Health.

Rana, M. M., Shiozawa, K., Mukai, K., Takayanagi, K., Eguchi, K., Sultana, H., Ohsaki, Y., Komai, M., & Shirakawa, H. (2021). S-allyl cysteine enhances testosterone production in mice and mouse testis-derived I-10 cells. Molecules, 26(6), 1697. https://doi.org/10.3390/molecules26061697

Ried, K. (2020). Garlic lowers blood pressure in hypertensive subjects, improves arterial stiffness and gut microbiota: A review and meta-analysis. Experimental and Therapeutic Medicine, 19(2), 1472–1478. https://doi.org/10.3892/etm.2019.8374

Ried, K., Frank, O. R., & Stocks, N. P. (2010). Aged garlic extract lowers blood pressure in patients with treated but uncontrolled hypertension: A randomised controlled trial. Maturitas, 67(2), 144–150. https://doi.org/10.1016/j.maturitas.2010.06.001

Ried, K., Travica, N., & Sali, A. (2018). The effect of Kyolic aged garlic extract on gut microbiota, inflammation, and cardiovascular markers in hypertensives: The GarGIC trial. Frontiers in Nutrition, 5, 122. https://doi.org/10.3389/fnut.2018.00122

Ried, K., Travica, N., & Sali, A. (2016). The effect of aged garlic extract on blood pressure and other cardiovascular risk factors in uncontrolled hypertensives: The AGE at Heart trial. Integrated Blood Pressure Control, 9, 9–21. https://doi.org/10.2147/IBPC.S93335

Serrano, J. C. E., Castro-Boqué, E., García-Carrasco, A., Morán-Valero, M. I., González-Hedström, D., Bermúdez-López, M., Valdivielso, J. M., Espinel, A. E., & Portero-Otín, M. (2023). Antihypertensive effects of an optimized aged garlic extract in subjects with grade I hypertension and antihypertensive drug therapy: A randomized, triple-blind controlled trial. Nutrients, 15(17), 3691. https://doi.org/10.3390/nu15173691

Vidya. (2026). Alliàge™ aged garlic extract: Product and clinical-research information. Manufacturer website. Accessed September 16, 2026.

Vidya / NutraIngredients Partner Feature. (2026). Aged garlic extract shows promise for men's testosterone support in new findings. Sponsored content; 60-day randomized placebo-controlled study summary. Accessed September 16, 2026.

Williams, M. J. A., Sutherland, W. H. F., McCormick, M. P., Yeoman, D. J., & de Jong, S. A. (2005). Aged garlic extract improves endothelial function in men with coronary artery disease. Phytotherapy Research, 19(4), 314–319. https://doi.org/10.1002/ptr.1663

Yang, P., Niu, Z., Guo, J., Huang, T., Chang, D., & Huang, X. (2026). Effects of oral fenugreek-derived preparations on testosterone-related outcomes in adult men: A systematic review and meta-analysis of randomized placebo-controlled trials. Frontiers in Nutrition, 13, 1903508. https://doi.org/10.3389/fnut.2026.1903508

Murali, S., Smith, E. R., Tiong, M. K., Tan, S.-J., & Toussaint, N. D. (2023). Interventions to attenuate cardiovascular calcification progression: A systematic review of randomized clinical trials. Journal of the American Heart Association, 12(23), e031676. https://doi.org/10.1161/JAHA.123.031676

Saadh, M. J., et al. (2024). Effects of aged garlic extract on blood pressure in hypertensive patients: A systematic review and meta-analysis of randomized controlled trials. Prostaglandins & Other Lipid Mediators, 175, 106914. RETRACTED 2026. Retraction DOI: 10.1016/j.prostaglandins.2026.107100.

Evidence notes. *This review distinguishes peer-reviewed evidence from manufacturer-reported unpublished/promotional clinical data. It intentionally excludes the retracted 2024 AGE blood-pressure meta-analysis from evidentiary support. Scientific discussion does not constitute an authorized health claim or medical advice.*

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