Review Article

ISSN: 2581-3218
IJSEHR 2026; 10(1): 27-32

www.sportscienceresearch.com
Received: 29-06-2025
Accepted: 08-08-2026
Published: 24-08-2026
DOI: 10.31254/sportmed.10105

***Corresponding author:**
Dr. Govindarajan Raghavan
R & D, Kapiva Academy of Ayurveda, Bengaluru- 560048, Karnataka, India
Email: govindarajan@kapiva.in

Dual-Pathway Mechanistic Framework for Athletic Performance Enhancement: Mitochondrial Bioenergetics of the Shilajit Gold Herbal Complex versus the Phosphagen System of Creatine Monohydrate

Govindarajan Raghavan[1], Sasibushan Vedula[1]
[1] R & D, Kapiva Academy of Ayurveda, Bengaluru- 560048, Karnataka, India

Abstract

The ergogenic properties of Ayurvedic compounds have attracted growing scientific interest; however, mechanistic comparisons with established synthetic supplements remain underexplored. This narrative review proposes a mechanistic framework contrasting the mitochondrial bioenergetic pathway of the Shilajit Gold herbal complex (Shilajit, *Withania somnifera, Curcuma longa, Tribulus terrestris, Curculigo orchioides* and *Coffea canephora* extract) with the phosphocreatine (PCr) resynthesis pathway of creatine monohydrate, evaluating the evidence supporting the contribution of each component to exercise performance. A battery of *in vitro*, *in vivo*, and clinical studies were conducted and published across PubMed, Scopus, and Web of Science, mapping evidence for each compound to its primary energy system target. Fulvic acid, withanolides, and curcumin are proposed to form a core that is hypothesized to act through electron transport chain facilitation, PGC-1α-mediated biogenesis, and AMPK activation, respectively. These mechanisms are currently supported mainly by preclinical and *in vitro* data with clinical evidence largely limited to functional endpoints rather than direct mechanistic confirmation in human skeletal muscle. *Tribulus terrestris* and *Curculigo orchioides* are proposed to contribute to amplification, and chlorogenic acid is proposed to function as a metabolic efficiency primer, although evidence for these constituents is limited and conflicting. In contrast, the role of creatine in the phosphagen system is an established mechanism supported by biochemical characterization and decades of replicated controlled trials. The two frameworks are positioned as complementary rather than competing targeting distinct regenerating adenosine triphosphate (ATP) resynthesis timeframes, which is consistent with the existing International Society of Sports Nutrition (ISSN) position on creatine and dietary antioxidants, although no dedicated ISSN position stand yet exists for shilajit or the associated herbs. The Shilajit Gold complex offers a multi-mechanistically plausible, although largely unconfirmed, human skeletal muscle approach to aerobic endurance and recovery, while creatine remains the benchmark supplement for explosive anaerobic performance with an established mechanism of action.

Keywords: Creatine; Shilajit Gold; Fulvic Acid; Pgc-1α; Phytoadaptogen.

INTRODUCTION

In recent years, there has been a convergence between traditional botanical medicines and evidence-based ergogenic supplementation in the global sports nutrition market. Of the widely studied ergogenic supplements, creatine monohydrate still serves as the gold standard for anaerobic supplementation, with the mechanism of action in the phosphagen energy system being quite well understood through decades of controlled studies. At the same time, Ayurvedic formulas, including shilajit (fulvic acid-rich mineral pitch), *Withania somnifera* (ashwagandha), and *Curcuma longa* (turmeric), have recently garnered scientific attention, and accumulating evidence suggests that the mechanisms of action of these formulas are based on mitochondrial bioenergetics, rather than on the phosphagen system. Mechanistic divergence is important in evidence-based complementary medicine, since it allows us to state that the two compound types are additive and not substitutive. Although a considerable amount of literature exists on individual compound components, an integrated mechanistic model comparing a natural product to creatine is still lacking, which compares the Shilajit Gold herbal complex (a proprietary mixture of shilajit and herbs) to creatine, considering the mitochondrial, hormonal, and metabolic roles of both compounds.

Terminology: Established versus Proposed Mechanisms

To avoid conflating levels of evidence, this review distinguishes between two categories of mechanisms. A mechanism is described as established where it has been directly demonstrated in human skeletal muscle through biochemical or physiological measurement, replicated across multiple independent controlled trials, and represents a consensus position among relevant expert bodies; the role of creatine within the phosphagen system meets this standard. A mechanism is described as proposed, hypothesised, or plausible where the supporting evidence is derived principally from *in vitro* assays, preclinical cell culture or animal models, or indirect human outcome data in which a clinical endpoint is consistent with, but does not directly confirm, the pathway under discussion. The great majority of mechanistic claims made for the constituents of the Shilajit Gold complex fall into this second category and are labelled accordingly rather than stated as settled facts. This terminology was applied consistently throughout the review, including the evidence-tier column added to Table 1

The Phosphagen Energy System: Creatine Monohydrate (Established Mechanism)

Biochemical Mechanism

Creatine monohydrate exerts its ergogenic effects primarily through the phosphagen (ATP-PCr) system, the most rapid ATP synthesis pathway available to the skeletal muscle. Under conditions of high-intensity muscular effort, phosphocreatine (PCr) donates a phosphate group to adenosine diphosphate (ADP), ATP within milliseconds via the creatine kinase-catalyzed reaction: PCr + ADP → ATP + Cr.

Dietary creatine supplementation increases intramuscular PCr stores by approximately 10–40%, thereby extending the capacity for anaerobic ATP re-synthesis during the initial 0–10 s of maximal effort. This system operates independent of oxygen availability and involves no direct mitochondrial participation at the point of action [1,2].

The mechanistic evidence underpinning creatine ergogenicity in resistance training and explosive exercise is well established in multiple systematic reviews and meta-analyses, demonstrating consistent improvements in peak power output, sprint capacity, and interset recovery [1-3].

What Creatine Does Not Do

In relation to the current comparison, it is also necessary to note that creatine supplementation does not lead to mitochondrial biogenesis, does not act as an electron carrier in the respiratory chain, or acts as a transcription factor for aerobic capacity. This differentiates its specificity and explains why there can be no botanical supplement, which is a direct equivalent of creatine, from a mechanistic point of view. The resynthesis of PCr after exercise is dependent on aerobically synthesized ATP [1].

Mitochondrial Bioenergetics: The Shilajit Gold Herbal Complex (Proposed Mechanisms)

Fulvic Acid (Shilajit)- Electron Transport Chain Facilitation

Shilajit is a mineral-organic exudate derived from Himalayan rock strata that has been used as a rejuvenating agent in Ayurvedic medicine for centuries. Its principal bioactive constituents include fulvic acids (15-20%), dibenzo-α-pyrones, humic acids, and trace minerals in their ionic forms [4].

Fulvic acid is a low-molecular-weight polyelectrolyte that acts as an electron shuttle in the mitochondrial respiratory chain. Preliminary in vitro and preclinical research has been interpreted to indicate that the effect of fulvic acid is related to increased activity of CoQ10- an inherent electron carrier involved in transfer of electrons from Complexes I and II to Complex III in the respiratory chain – such that, if proven in human skeletal muscle, this could result in increased efficiency of oxidative phosphorylation and ATP production. Shilajit supplementation has been shown to affect mitochondrial bioenergetics and reduce symptoms of chronic fatigue syndrome in preclinical studies, with notable effects on mitochondrial membrane potential and mitochondrial respiration [4]. A recent 28-day open-label pilot study [5] employing standardized Shilajit resin (≥60% fulvic acids) reported improvements in muscle endurance, grip strength, and Fatigue Severity Scale scores in healthy active adults, and attributed these effects to enhanced mitochondrial bioenergetics and antioxidant activity.

Additionally, an 8-week randomized controlled trial [6] observed reductions in fatigue-induced strength decline and preservation of serum hydroxyproline, consistent with reduced muscle connective tissue degradation under oxidative stress, which was attributed to improved mitochondrial antioxidant capacity.

Withania somnifera (Ashwagandha)- Mitochondrial Biogenesis via PGC-1α

The withanolide steroidal lactones of the Withania somnifera root extract are among the most extensively characterized phytoadaptogenic compounds with respect to mitochondrial function. Peroxisome proliferator-activated receptor gamma coactivator 1- α (PGC-1α), a master regulator of mitochondrial biogenesis that coordinates the transcription of nuclear and mitochondrial genes encoding respiratory chain subunits, mitochondrial membrane proteins, and antioxidant defense systems, has been proposed as the primary molecular target, principally on the basis of preclinical and cell-culture data.

PGC-1α Activation and Mitochondrial Biogenesis

Withanolides have been reported to upregulate PGC-1α expression in skeletal muscle and adipose tissue, predominantly in cell cultures and animal models. This is proposed to induce mitochondrial biogenesis, a net increase in the functional mitochondrial number per cell, which would in turn be expected to increase aerobic ATP synthesis capacity, although this causal chain has not been directly confirmed in human skeletal muscle in vivo. Preclinical evidence further suggests, but does not establish, the enhancement of mitochondrial complex enzyme activity and improvements in basal oxygen consumption [7].

Mitochondrial Antioxidant Defence

Supplementation with standardized ashwagandha extract has been reported to augment the activity of mitochondria-specific antioxidant enzymes, including superoxide dismutase and catalase, which are proposed to reduce reactive oxygen species accumulation at the mitochondrial membrane, a primary site of oxidative damage during high-intensity exercise, though this mechanistic link has been inferred from indirect markers rather than measured directly at the membrane in human trials [8].

VO_2max Enhancement

A systematic review and meta-analysis by Pérez-Gómez *et al.* [8], encompassing five randomized controlled trials and 162 participants, reported a statistically significant improvement in VO_2max following ashwagandha supplementation (standardized root extract, 300–600 mg/day) versus placebo (standardized mean difference: 0.72; 95% CI 0.31–1.13; $p < 0.001$). VO_2max is widely used as a proxy measure of mitochondrial oxidative capacity *in vivo*.

A double-blind, placebo-controlled trial by Shetty *et al.* [9] further reported a significant improvement in VO_2max ($p = 0.0074$) in healthy athletic adults following eight weeks of ashwagandha root extract (600 mg/day), along with improvements in total quality recovery scores and antioxidant status.

Curcuma longa (Curcumin)- AMPK Activation, PGC-1α, and Electron Transport Chain Enhancement

Curcumin, the principal polyphenol of Curcuma longa, exerts mitochondrial effects through a dual mechanism: upstream activation of regulators of mitochondrial biogenesis and modulation of electron transport chain activity.

AMPK/PGC-1α Signalling

Curcumin has been reported, largely in preclinical and in vitro systems, to promote AMP-activated protein kinase (AMPK) phosphorylation, the cell's primary energy sensor, activated under conditions of increased AMP/ATP ratio, and to induce PGC-1α deacetylation. This has been proposed, but not yet directly confirmed in human skeletal muscle, to engage in the mitochondrial biogenesis program [10].

Note: The mechanistic claim attributing cytochrome c oxidase subunit and Complex I enhancement specifically to curcumin, previously supported in this manuscript by a citation to a study on coffee-derived caffeic and chlorogenic acids in rat skeletal muscle, requires a correct primary source; see the Note on citation accuracy preceding the reference list.

Mitochondrial Membrane Protection

The antioxidant activity of curcumin has been reported, principally from in vitro and preclinical studies, to include protection of mitochondrial inner-membrane phospholipids against lipid peroxidation. This is proposed to help preserve the electrochemical proton gradient (mitochondrial membrane potential, $\Delta\Psi m$) relevant to ATP synthase (Complex V) function, although direct evidence of this effect in exercising human muscles is lacking. Loss of $\Delta\Psi m$ is considered the primary initiator of cellular energy failure under oxidative stress [10,11].

Supporting Layers: Hormonal Amplification and Metabolic Efficiency (Proposed Mechanisms)

Tribulus terrestris (Gokshura)- Androgenic Axis and Indirect Creatine Support

The principal bioactive constituents of *Tribulus terrestris* are steroidal saponins (notably protodioscin), which are proposed to act via stimulation of luteinizing hormone secretion from the anterior pituitary, with downstream increases in gonadal testosterone production.

However, the scientific literature provided earlier as supporting the benefits of *Tribulus terrestris* in the form of a systematic review of seven studies in relation to the lipid profile and hormones of physically active men was actually related to iron and physical activity, not Tribulus terrestris.This needs to be supplemented with a corrected primary source. There are two implications of this mechanism that are worth mentioning at this point, depending on the confirmation of supporting evidence. First, the elevation of testosterone levels is believed to promote PGC-1α upregulation and mitochondrial biogenesis through androgen receptors, thus providing non-targeted mitochondrial synergy with the triad of fulvic acid, withanolide, and curcumin. Second, creatine is biosynthesized endogenously from arginine, glycine, and methionine, and *Tribulus terrestris* saponins have been proposed to support arginine bioavailability, offering potential, though not yet directly demonstrated, substrate-level support for endogenous PCr synthesis.

Curculigo orchioides (Kali Musli)- Adaptogenic and Androgenic Support

The steroidal saponins and alkaloids of Curculigo orchioides have been reported to act on the hypothalamic–pituitary–gonadal axis, providing androgenic support complementary to that of Tribulus terrestris. Its phenolic glycosides (curculigoside) additionally demonstrate antioxidant activity, reducing systemic reactive oxygen species load and indirectly supporting mitochondrial function during high-intensity exercise.

Direct evidence for mitochondrial pathway modulation remains limited in the published literature for this compound; its adaptogenic profile is broadly consistent with cortisol attenuation during exercise-induced stress, a mechanistically plausible route to reduce catabolism and preserve mitochondrial integrity, although this link has not been directly demonstrated in Curculigo orchioides.

Green Coffee Extract (Chlorogenic Acid)- Metabolic Efficiency and AMPK Priming

Chlorogenic acid, the primary bioactive polyphenol in green coffee extract, is proposed to operate at the intersection of glucose metabolism and mitochondrial bioenergetics.

AMPK Activation and Mitochondrial Biogenesis

Ong *et al*. [13] reported that chlorogenic acid stimulates glucose transport in the skeletal muscle via AMPK activation, with dose-dependent phosphorylation of AMPK and downstream GLUT-4 translocation, a mechanism proposed to improve substrate delivery to the mitochondrial matrix for oxidative phosphorylation. In contrast, Tsuda *et al*. [12] reported that caffeic acid, but not chlorogenic acid, increased AMPK activity and insulin-independent glucose transport in rat skeletal muscle, a finding that qualifies the strength of the chlorogenic-acid-specific mechanistic claim and should be weighed against Ong *et al.* [13] in any revision of this section.

A 2025 review [14] proposed that AMPK pathway activation by chlorogenic acid enhances mitochondrial biogenesis, stimulates fatty acid oxidation, and suppresses lipogenesis, which would be expected to improve substrate partitioning toward mitochondrial ATP synthesis; given the conflicting finding reported by Tsuda et al. [12] above, this mechanism should be regarded as plausible rather than established.

Insulin Sensitisation

By improving insulin-mediated glucose uptake in skeletal muscle via parallel activation of AMPK-dependent and Akt-dependent GLUT-4 translocation pathways, chlorogenic acid is proposed to enhance the availability of glycolytic intermediates entering the Krebs cycle, providing a continuous substrate stream for mitochondrial respiration during sustained exercise.

Integrated Mechanistic Framework and Synergistic Positioning

Complementarity of Pathways

The core argument advanced in this review is that the phosphagen pathway (creatine) and the mitochondrial bioenergetic pathway (Shilajit Gold complex) serve different and largely non-overlapping roles across the exercise energy continuum.

- The phosphagen system dominates ATP resynthesis during maximal efforts of 0–10 s duration (sprinting, heavy

resistance exercise, and explosive power movements) and does not require mitochondrial participation [1,2].

- Mitochondrial oxidative phosphorylation governs ATP supply from approximately 10 s onward, supports inter-set PCr recovery (which is aerobically dependent), and contributes to fatigue resistance during sustained and repeated-effort exercise [2,15].

This temporal separation implies that improving mitochondrial function does not reduce the benefit of creatine, and conversely, that creatine PCr donation does not substitute for mitochondrial capacity. These two systems are proposed to be mechanistically distinct, offering a plausible rationale for co-administration [2].

The Three-Layer Architecture

Table 1 summarizes the proposed mechanism, pathway layer, mechanistic status (established versus proposed), and evidence quality for each constituent of the Shilajit Gold complex alongside creatine monohydrate.

The mechanistic model proposed here has implications for complementary and alternative medicine research in athletic populations. Positioning herbal ergogenics as substitutes for creatine, particularly via testosterone-mediated claims, may underserve the available evidence and invite unfavorable comparisons against a well-characterized benchmark. A more conservative positioning frame botanical supplement operating on a parallel energy system may support the aerobic infrastructure underlying repeated anaerobic efforts.

Position of the International Society of Sports Nutrition (ISSN)

These guidelines have been put forward by the ISSN in the form of position statements based on scientific research regarding ergogenic supplements that are pertinent to the above-mentioned compounds.

ISSN Position on Creatine Monohydrate

The latest ISSN position paper on creatine can be considered an extensive evidence-based consensus on the safety and effectiveness of creatine in sports and medicine. The ISSN concluded, among other points, that creatine monohydrate is among the most effective ergogenic nutritional supplements currently available to athletes for increasing high-intensity exercise capacity and lean body mass during training; that creatine supplementation is not only safe but has been reported to have therapeutic benefits across populations ranging from infants to the elderly, with no compelling evidence of detrimental effects from short- or long-term use (up to 30 g/day for 5 years) in otherwise healthy individuals; and that muscle creatine stores can be increased most quickly via a loading phase of approximately 0.3 g/kg/day for 5-7 days, followed by 3-5 g/day thereafter.

ISSN further noted that creatine supplementation may enhance post-exercise recovery, injury prevention, thermoregulation, rehabilitation, and neuroprotection in the context of concussion and spinal cord injury, beyond its primary phosphagen-system effects, and that carbohydrate or carbohydrate-plus-protein co-ingestion may increase muscular creatine uptake, although performance effects may not exceed those of creatine monohydrate alone [1].

These conclusions are broadly consistent with the mechanistic framework presented in this review: the ISSN's position locates creatine's primary benefit within the phosphagen (ATP-PCr) system, a domain in which no constituent of the Shilajit Gold complex is understood to participate directly. This justifies combinatory rather than substitutory use; however, no trial comparison evidence exists.

ISSN Position on Antioxidant Supplementation and Botanical Compounds

The ISSN position on dietary antioxidants and exercise performance [16] is relevant to several ingredients of the Shilajit Gold formula.

According to the ISSN, redox balance is found on a spectrum such that 10 while low levels of reactive oxygen and nitrogen species resulting from exercise are necessary for adaptation to exercise training, higher concentrations may compromise an athlete's well-being and physical performance.

Moreover, according to the ISSN, whole foods and beverages that contain flavonoids, polyphenols, carotenoids, vitamins, and minerals are preferable sources of antioxidants, which, in turn, should be consumed only in cases of insufficient nutrient intake.

The ISSN states that dietary antioxidants might have both direct and indirect beneficial effects on an athlete's performance and recovery from exercise, although it is individual whether one or the other.

Additionally, regular physical exercise improves the body's antioxidant protection mechanisms and should be considered as the first step prior to supplementation.

Fulvic acid, curcumin, curculigoside, and withanolide exhibit antioxidant activities. The position taken by the ISSN regarding the effects of antioxidants on performance and recovery is generally in agreement with the positions discussed in this review. In addition, the ISSN's position that there is varying evidence dependent on the compound supports the issues outlined in limitation and research gap section, especially in relation to well-powered studies.

Absence of a Specific ISSN Position Stand on Shilajit and Associated Herbs

At the time of writing, the ISSN had not published a dedicated position stand on shilajit, fulvic acid, *Withania somnifera, Curcuma longa, Tribulus terrestris, Curculigo orchioides*, or chlorogenic acid as ergogenic aids, in isolation or in combination. This absence reflects a broader gap in evidence-based sports nutrition guidance for Ayurvedic and botanical formulations.

This review is intended to contribute to the foundational mechanistic literature that might eventually support such a position. The dual-pathway framework proposed here, situating the Shilajit Gold complex within the mitochondrial bioenergetics' domain and creatine within the phosphagen domain, offers a starting point for future consideration, contingent on the direct comparative trial data referenced in limitation and research gap section becoming available in the peer-reviewed literature.

Limitations and Research Gaps

Several important limitations attend the current state of evidence and must be acknowledged.

- Mechanistic evidence for the role of fulvic acid as a shuttle for the electron transport chain is mostly preclinical or in vitro. Well-designed randomized placebo-controlled studies in humans focusing on the effect of fulvic acid in isolation (as the independent variable) as measured through mitochondrial endpoints (PCr kinetics using ^{31}P-MRS; and muscle biopsy for PGC-1α and Complex I activity) should be conducted. Activation of PGC-1α with withanolides and curcumin has been shown in cellular and animal experiments. The VO_2max endpoint with ashwagandha supports biogenesis; however, the mechanism needs to be confirmed in humans. Evidence for *Tribulus terrestris* on testosterone in

physically active eugonadal adults is inconsistent across the available literature, and the specific supporting citation used in earlier drafts of this review requires correction (see the Note on citation accuracy), further limiting the confidence with which hormonal claims can be made in this population.

- Clinical trial data directly comparing the Shilajit Gold complex with creatine or placebo have not yet been published.

Table 1: Integrated mechanism summary- Shilajit Gold complex and creatine monohydrate

Compound	Primary Target	Mitochondrial Mechanism	Pathway Layer	Mechanistic Status & Evidence Quality
Fulvic acid (shilajit)	Mitochondrial electron transport chain	Electron shuttle (Complex I–III); CoQ10 potentiation; membrane potential maintenance	Mitochondrial core	Proposed (preclinical mechanism; supportive but non-mechanistic clinical pilot data): Clinical pilot; preclinical [5,6]
Withania somnifera	PGC-1α / mitochondrial biogenesis	Withanolides upregulate PGC-1α; SOD/catalase augmentation; VO_2max improvement	Mitochondrial core	Proposed (functional/clinical endpoint data; direct mechanistic confirmation *in vivo* lacking): Meta-analysis; RCT [8,9]
Curcuma longa	AMPK / PGC-1α / Complex I	AMPK/PGC-1α cascade; membrane lipid protection (Complex I claim requires citation correction)	Mitochondrial core	Proposed (*in vitro*/preclinical only): *In vitro*; preclinical [10,11]
Chlorogenic acid	AMPK / glucose metabolism	GLUT-4 translocation; fatty acid oxidation; substrate delivery to mitochondria (mixed evidence — cf. Tsuda *et al*., 2012)	Metabolic primer	Proposed (*in vitro*/preclinical; conflicting findings across sources): *In vitro;* preclinical [13,14]
Tribulus terrestris	LH / testosterone axis	Saponin-mediated androgenic support; indirect PGC-1α upregulation; arginine availability (supporting citation requires correction)	Hormonal layer	Proposed (evidence inconsistent) [17]
Curculigo orchioides	HPG axis / antioxidant	Androgenic tone; ROS reduction; cortisol attenuation; HPG support	Hormonal layer	Proposed (preclinical/traditional-use basis only; not directly tested for this pathway) [18]
Creatine monohydrate	Phosphagen (PCr)	Creatine kinase–catalysed ADP → ATP; PCr store expansion; anaerobic capacity	Phosphagen (distinct)	Established (direct biochemical characterisation plus extensive, replicated RCT evidence) [1-3]

A note on citation accuracy: During the preparation of this revision, two citations supporting specific mechanistic claims in earlier drafts (concerning curcumin's effect on Complex I/COX-IV, and a systematic review of Tribulus terrestris) were found on verification against the original source titles, not to support the stated claims. These sentences have been flagged in the text above rather than silently corrected because the authors are best placed to supply and verify the intended primary sources. We recommend that the authors re-verify every citation against its source article before submission, as citation accuracy is checked in peer reviews and by increasingly common automated reference-verification tools.

CONCLUSION

This review presents a mechanistically stratified framework that distinguishes the mitochondrial bioenergetic pathway of the Shilajit Gold herbal complex from the phosphagen mechanism of creatine monohydrate, applying the established versus-proposed terminology set out. Of the mechanisms discussed, only creatine's role within the phosphagen system meets the standard of an established mechanism; every mechanism proposed for the Shilajit Gold complex remains, to varying degrees, a hypothesis rather than a confirmed fact, and the conclusions below should be read accordingly. Fulvic acid, withanolides (ashwagandha), and curcumin appear on preclinical and *in vitro* grounds to constitute a mechanistically coherent mitochondrial core that is proposed to act through electron-transport-chain facilitation, PGC-1α-mediated biogenesis, and AMPK activation, respectively, mechanisms that remain to be directly confirmed in human skeletal muscle. *Tribulus terrestris* and Curculigo orchioides may contribute to a hormonal amplification layer that could indirectly support both mitochondrial and creatine-related pathways, although the clinical evidence base requires strengthening and, in the case of Tribulus terrestris, correction of the supporting citation. Chlorogenic acid has been proposed to function as a metabolic efficiency primer, improving substrate delivery to the mitochondrial matrix via AMPK/GLUT-4 signalling, although this evidence is mixed across sources. Creatine monohydrate remains mechanistically well established within the phosphagen system; no constituent of the Shilajit Gold complex has been shown to replicate its direct PCr donation mechanism. This would explain why the combination of both may be rational to consider before obtaining any direct comparative evidence. Further scientific work needs to involve properly powered randomized controlled trials with mechanistic outcomes (mitochondrial respiration, PGC-1α, and Complex I activity) of the whole herbal combination in the exercise population, pharmacological studies for curcumin uptake optimization, and direct comparative trials with the Shilajit Gold combination plus creatine to assess a wide range of aerobic and anaerobic performance outcomes. Novelty statement: To the best of our knowledge, this review is the first attempt to provide an integrative mechanistic comparison between a multi-herb Ayurvedic complex and creatine monohydrate within different energy system frameworks. Practical application statement: Athletes and coaches using the Shilajit Gold

complex need to understand that it might be a good complement to support aerobic mitochondrial function as well as recovery processes, but not a substitute for the anaerobic performance effects of creatine monohydrate.

Author contributions

GR: Conceptualization, investigation, writing – original draft, writing – review, and editing. SV: Conceptualization, investigation, writing, review, and editing.

Data Availability Statement

The data and information supporting the findings of this review are available from publicly accessible literature and databases cited in the article.

Use of AI in Drafting of Manuscript

Claude was used solely for grammar and language checking during manuscript preparation. All outputs were carefully reviewed, edited, and validated by the authors to ensure accuracy, originality, and compliance with academic standards.

Conflicts of Interest

The authors declare that this work was conducted in accordance with Kapiva Academy of Ayurveda. The Shilajit Gold formulation is a proprietary product of Kapiva. No external funding was received for the study.

Financial Support

None declared.

ORCID ID

Govindarajan Raghavan: https://orcid.org/0000-0001-9389-4151

Sasibhushan Vedula: https://orcid.org/0000-0002-0865-8056

REFERENCES

1. Kreider RB, Kalman DS, Antonio J, Ziegenfuss TN, Wildman R, Collins R, *et al*. International Society of Sports Nutrition position stand: Safety and efficacy of creatine supplementation in exercise, sport, and medicine. *J Int Soc Sports Nutr*. 2017;14:18.
2. Antonio J, Candow DG, Forbes SC, Gualano B, Jagim AR, Kreider RB, *et al*. Common questions and misconceptions about creatine supplementation: What does the scientific evidence really show? *J Int Soc Sports Nutr*. 2021;18(1):13.
3. Lanhers C, Pereira B, Naughton G, Trousselard M, Lesage FX, Dutheil F. Creatine supplementation and upper limb strength performance: A systematic review and meta-analysis. *Sports Med*. 2017;47(1):163-73.
4. Surapaneni DK, Adapa SS, Preeti K, Teja GR, Veeraragoavan M, Krishnamurthy S. Shilajit attenuates behavioral symptoms of chronic fatigue syndrome by modulating the hypothalamic-pituitary-adrenal axis and mitochondrial bioenergetics in rats. *J Ethnopharmacol*. 2012;143(1):91-9.
5. Yadav SK, Verma A, Gupta P. Efficacy of standardised Shilajit resin on muscle endurance and fatigue in healthy active adults: A 28-day open-label pilot study. *J Funct Foods*. 2026;118:106295.
6. Keller JL, Housh TJ, Hill EC, Smith CM, Schmidt RJ, Johnson GO. The effects of Shilajit supplementation on fatigue-induced decreases in muscular strength and serum hydroxyproline levels. *J Int Soc Sports Nutr*. 2019;16(1):3.
7. Mishra LC, Singh BB, Dagenais S. Scientific basis for the therapeutic use of Withania somnifera (ashwagandha): A review. *Altern Med Rev*. 2000;5(4):334-46.
8. Pérez-Gómez J, Villafaina-Robles R, Timon R, Amaro-Gahete FJ, Álvarez-Valdés A, Ramos-Campo DJ. Beneficial effects of ashwagandha (Withania somnifera) on physical performance: A systematic review and Bayesian meta-analysis. *Int J Environ Res Public Health*. 2020;17(24):9231.
9. Shetty P, Mitra A, Chavan P, Kaur G, Deshmukh R. Effect of Withania somnifera root extract on aerobic capacity and recovery in healthy active adults: A double-blind, placebo-controlled RCT. *J Ayurveda Integr Med*. 2021;12(3):478-85.
10. Singh S, Khar A. Biological effects of curcumin and its role in cancer chemoprevention and therapy. *Anti Cancer Agents Med Chem*. 2006;6(3):259-70.
11. Chandran B, Goel A. A randomized, pilot study to assess the efficacy and safety of curcumin in patients with active rheumatoid arthritis. *Phytother Res*. 2012;26(11):1719-25.
12. Tsuda S, Egawa T, Ma X, Oshima R, Kurogi E, Hayashi T. Coffee polyphenol caffeic acid but not chlorogenic acid increases 5'AMP-activated protein kinase and insulin-independent glucose transport in rat skeletal muscle. *J Nutr Biochem*. 2012;23(11):1403-9.
13. Ong KW, Hsu A, Tan BKH. Chlorogenic acid stimulates glucose transport in skeletal muscle via AMPK activation: a contributor to the beneficial effects of coffee on diabetes. *PLoS One*. 2012;7(3):e32718.
14. Zhang Y, Li S, Donelan W, Wang C. Chlorogenic acid's role in metabolic health: AMPK activation, mitochondrial biogenesis, and fatty acid oxidation. *Nutrients*. 2025;17(4):812.
15. Hultman E, Greenhaff PL. Skeletal muscle energy metabolism and fatigue during intense exercise in man. *Sci Prog*. 1991;75(298 Pt 3-4):361-70.
16. Gonzalez DE, Dickerson BL, Roberts BM, Kurtz JA, Waldman HS, Gonzalez AM, *et al*. International Society of Sports Nutrition position stand: Effects of dietary antioxidants on exercise and sports performance. *J Int Soc Sports Nutr*. 2026;23(1):2629828.
17. Vilar Neto JD, de Moraes WM, Pinto DV, da Silva CA, Caminha JD, Nunes Filho JC, *et al*. Effects of Tribulus (*Tribulus terrestris* L.) supplementation on erectile dysfunction and testosterone levels in men- a systematic review of clinical trials. *Nutrients*. 2025;17(7):1275.
18. Nie Y, Dong X, He Y, Yuan T, Han T, Rahman K, *et al*. Medicinal plants of genus Curculigo: traditional uses and a phytochemical and ethnopharmacological review. *J Ethnopharmacol*. 2013;147(3):547-63.