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| **ARTICLE TITLE** | MORINGA OLEIFERA LEAF EXTRACT IN MUSCLE WASTING AND SKELETAL MUSCLE DISORDERS: MECHANISMS, EVIDENCE, AND THERAPEUTIC POTENTIAL—A NARRATIVE REVIEW |
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# MORINGA OLEIFERA LEAF EXTRACT IN MUSCLE WASTING AND SKELETAL MUSCLE DISORDERS: MECHANISMS, EVIDENCE, AND THERAPEUTIC POTENTIAL—A NARRATIVE REVIEW

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**ABSTRACT**

**Background:** Skeletal muscle wasting accompanies ageing, malnutrition, glucocorticoid exposure, chronic disease, denervation, systemic inflammation, and prolonged inactivity. Interest in accessible plant-derived nutritional interventions has therefore expanded. Moringa oleifera leaves contain polyphenols, flavonoids, glucosinolates, isothiocyanates, and nutrients that may influence oxidative metabolism, redox balance, inflammation, and protein turnover.
**Objective:** To synthesize evidence on M. oleifera leaf-derived preparations in muscle mass loss, impaired muscle function, fatigue, and skeletal muscle disorders, while defining the translational limitations of the literature.
**Methods:** PubMed/MEDLINE and Google Scholar were searched for English-language, peer-reviewed publications from January 2016 through July 22, 2026. Cell studies, animal experiments, human studies, and reviews were considered. Claims were extracted from full texts when available; studies available only as abstracts or publisher previews were interpreted strictly within those limits.
**Results:** Mechanistic studies in C2C12 cells consistently indicate modulation of Nrf2/HO-1 antioxidant signaling, glutathione homeostasis, SIRT1–PPARα-associated oxidative metabolism, and protection from hydrogen-peroxide stress. Animal studies report improved endurance or functional recovery and changes in inflammatory, fibrotic, apoptotic, mitochondrial, and protein-degradation markers; a 2026 study in mdx mice reported a partial functional and biochemical signal without uniform morphological benefit. Four small human performance studies were heterogeneous and did not evaluate patients with muscle wasting.
**Conclusions:** M. oleifera leaf-derived preparations are biologically plausible candidates for further muscle research, but current evidence is predominantly preclinical, heterogeneous, and insufficient to support treatment recommendations for sarcopenia, cachexia, or other muscle-wasting disorders. Standardized products and adequately powered clinical trials are required.

**KEYWORDS**

Moringa oleifera, Skeletal Muscle, Muscle Wasting, Oxidative Stress, Mitochondrial Biogenesis

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## 1. Introduction

Skeletal muscle is not only the principal tissue responsible for movement and force production; it is also a major site of glucose disposal, amino-acid storage, thermogenesis, and metabolic adaptation. Loss of muscle quantity or quality can arise from ageing, undernutrition, cancer, chronic organ disease, infection, endocrine disorders, glucocorticoid exposure, denervation, immobilization, and critical illness. Although the clinical labels differ, many of these conditions share an imbalance between muscle protein synthesis and degradation, impaired mitochondrial function, oxidative stress, and inflammatory signaling (Hyatt & Powers, 2021; Yin et al., 2021; Zhang et al., 2023). Sarcopenia is now conceptualized as a muscle disease in which low strength is central, with reduced muscle quantity or quality used to confirm the diagnosis and poor physical performance indicating greater severity (Cruz-Jentoft et al., 2019).

The public-health relevance of muscle dysfunction is increasing as populations age and more people live with multimorbidity. At the same time, many individuals seek plant-based supplements because such products are culturally familiar, widely marketed, and often available without prescription. Accessibility, however, should not be confused with demonstrated efficacy. Botanical products can vary by species, plant part, geography, harvest conditions, processing, extraction solvent, dose, and chemical standardization. A clinically useful review must therefore distinguish a defined leaf extract from leaf powder, whole-leaf diets, seed-derived compounds, and multi-ingredient preparations rather than treating all products labelled “moringa” as equivalent.

*Moringa oleifera* Lam. is a member of the Moringaceae family and the best-known species within the genus *Moringa*. The leaves are consumed as food and used in powders, infusions, capsules, and extracts. Contemporary reviews describe a complex phytochemical profile that includes flavonoids, phenolic acids, glucosinolates, isothiocyanates, tannins, saponins, terpenoids, and other constituents (Kou et al., 2018; Padayachee & Baijnath, 2020; Pareek et al., 2023). These constituents provide several plausible links to muscle biology: redox-sensitive transcription, mitochondrial substrate utilization, inflammatory signaling, apoptosis, fibrosis, angiogenesis, and protein turnover. Nevertheless, plausibility is not clinical proof, and nutrient-rich leaf material cannot automatically be assumed to act through the same mechanisms as a concentrated methanolic, ethanolic, or aqueous extract.

Research directly connecting *M. oleifera* to skeletal muscle has expanded since 2016. Cell experiments have evaluated redox homeostasis, myoblast proliferation, and oxidative metabolism. Rodent studies have examined forced-swimming endurance, dexamethasone-associated dysfunction, valproate-related oxidative injury, peripheral-nerve injury, ageing-like frailty, voluntary running, and dystrophic muscle in mdx mice. Four small human studies have assessed exercise or cycling performance, but their designs and findings are heterogeneous (Dong et al., 2024; Gbedinhessi et al., 2025; Ray et al., 2023; Tsuk et al., 2022). This evidence base is promising enough to justify synthesis, yet too heterogeneous to support simple claims that moringa "builds muscle" or treats muscle wasting.

The review question was: What direct evidence supports an effect of *M. oleifera* leaf-derived preparations on skeletal muscle mass, structure, metabolism, function, fatigue, or recovery, and through which mechanisms might such effects occur? Secondary questions were: How consistent are the findings across cellular, animal, and human models? Which findings concern true muscle wasting rather than short-term exercise performance? How do preparation heterogeneity and study quality affect interpretation? What evidence is required before use can be recommended in clinical nutrition or medical practice?

The objective of this narrative review was therefore to summarize current knowledge on *M. oleifera* leaf-derived preparations in muscle mass loss and skeletal muscle dysfunction; integrate mechanistic and functional findings; identify overlap with existing reviews; and define priorities for translational studies. The intended contribution is narrower than broad reviews of moringa pharmacology, musculoskeletal health, or athletic performance. It emphasizes direct skeletal-muscle outcomes, separates evidence by level, and treats clinical applicability as an unresolved question rather than a presumed benefit.

## 2. Methodology

This article was designed as a structured narrative review. The reporting approach was informed by the principles underlying the Scale for the Assessment of Narrative Review Articles (SANRA), particularly the need to state the review's importance, objectives, search approach, evidence reasoning, and relevant endpoint data (Baethge et al., 2019). It was not registered as a systematic review and no meta-analysis was attempted because the evidence comprises heterogeneous cell systems, animal models, preparations, doses, durations, and outcomes.

A literature search was conducted in PubMed/MEDLINE and Google Scholar for publications dated from January 1, 2016, through July 22, 2026. In PubMed, Medical Subject Headings (MeSH) and free-text terms were combined. The principal concept was "Moringa oleifera," combined with terms including "skeletal muscle," muscle, myoblast, myotube, muscle wasting, muscular atrophy, sarcopenia, cachexia, frailty, muscle mass, muscle damage, oxidative stress, inflammation, mitochondrial biogenesis, exercise, endurance, fatigue, motor recovery, and sciatic nerve. Additional targeted searches used pathway terms such as Nrf2, HO-1, NF-κB, SIRT1, PPAR, PGC-1α, AMPK, caspase-3, and protein degradation. Reference lists of relevant primary studies and recent reviews were also examined to identify additional eligible records.

Eligible publications were peer-reviewed and written in English. All study designs were considered if they reported outcomes directly relevant to skeletal muscle cells, muscle tissue, muscle mass or morphology, strength, endurance, fatigue, locomotor function, or recovery. The primary scope was leaf-derived material, including leaf extract, leaf powder, and leaf-based diets. Studies of a purified moringa compound from another plant part were retained only when they provided direct mechanistic evidence in skeletal muscle and were clearly labelled as indirect to the leaf-extract question. Studies focused exclusively on bone, joints, cardiac muscle, livestock production, carcass quality, or non-muscle organs were excluded from the core synthesis.

Evidence was organized by experimental level: in vitro skeletal-muscle models, in vivo animal models, and human studies. Preparation type, model, dose or exposure when reported, principal muscle outcome, and major limitations were extracted. Full-text articles were used whenever accessible. When only an abstract or

publisher preview was available, no unreported methods, numerical findings, adverse events, or mechanistic details were inferred. These records are explicitly marked in Table 1. No pooled effect estimate was calculated, and the qualitative interpretation gave greater weight to direct muscle endpoints, controlled designs, product characterization, replication across models, and human data.

The search strategy intentionally included exercise and fatigue studies because muscle endurance is a relevant functional domain and because much of the available moringa literature was generated in performance models. However, improved endurance was not treated as synonymous with prevention of atrophy. Likewise, changes in oxidative or inflammatory biomarkers were considered mechanistic support rather than proof of preserved muscle mass. Foundational papers outside the ten-year search window were used sparingly for context or safety and were not treated as newly identified efficacy evidence.

## 3. Results

### 3.1. Evidence landscape and originality of the review topic

The literature identified a small but expanding cluster of direct skeletal-muscle studies. The strongest concentration of mechanistic work comes from C2C12 mouse muscle cells, particularly studies of oxidative metabolism and redox regulation. Animal evidence is broader but fragmented across acute fatigue, pharmacological injury, ageing-like frailty, denervation, exercise, and a 2026 mdx model of Duchenne muscular dystrophy. Human evidence remains sparse and was not conducted in patients with sarcopenia, cachexia, muscular dystrophy, inflammatory myopathy, or chronic disease-related muscle loss.

The topic is not completely unreviewed. A 2025 review addressed moringa as nutritional supplementation and discussed musculoskeletal health broadly, including bone, inflammation, pain, and neuroprotective themes (Manoharan et al., 2025). A separate 2025 systematic review focused on sports performance and included athletic or ergogenic outcomes (Bahauddin et al., 2025). These publications create overlap if the present topic is framed simply as “moringa and muscles.” A defensible niche remains when the scope is restricted to skeletal muscle wasting and dysfunction, direct muscle mechanisms, evidence hierarchy, preparation-specific interpretation, and translational gaps. Accordingly, this review does not claim to be the first publication to mention moringa in musculoskeletal health; rather, it provides a focused appraisal of whether direct muscle evidence supports therapeutic development for wasting disorders.

### 3.2. Botanical material, phytochemistry, and preparation heterogeneity

The term “moringa” encompasses biologically different interventions. Whole dried leaves provide macronutrients, micronutrients, fibre, and phytochemicals. Aqueous preparations preferentially extract water-soluble compounds, whereas ethanol or methanol can yield different profiles and concentrations. Purified flavonoid concentrates and isolated isothiocyanates are still more distinct. Consequently, a dose expressed as milligrams of crude leaf powder cannot be compared directly with the same mass of an extract or purified molecule.

Leaf-focused reviews consistently identify flavonoids and phenolic acids alongside glucosinolates and their isothiocyanate derivatives (Kou et al., 2018; Pareek et al., 2023). In the direct C2C12 literature, methanolic or ethanolic leaf extracts were chemically profiled in some studies, while other animal or human experiments used commercial capsules or leaf material with limited reporting of marker compounds. Ceci et al. (2024a) compared extracts from plants originating in multiple countries but grown in a common garden. Although metabolomic profiles differed, all evaluated extracts reduced hydrogen-peroxide-associated cell death in C2C12 myotubes. This finding supports functional overlap across samples under the tested conditions, but it does not establish interchangeability of all commercial products.

A major scope issue concerns moringa isothiocyanate-1 (MIC-1, also called moringin). The direct anti-inflammatory skeletal-muscle study used MIC-1 extracted from seeds rather than a leaf extract (Sailaja et al., 2022). The study is valuable for understanding a moringa-derived isothiocyanate and NF-κB signaling, but it cannot be used as direct proof that any leaf supplement provides an equivalent exposure. The distinction is clinically important because product labels rarely specify enzymatic conversion, isothiocyanate yield, stability, or bioavailability.

### 3.3. Cellular evidence: redox regulation, oxidative metabolism, and myogenic resilience

The cellular evidence provides the clearest mechanistic chain. Duranti et al. (2021a) reported that *M. oleifera* leaf extract increased oxidative metabolism in differentiated C2C12 myotubes and was associated with SIRT1–PPARα signaling. Enzymatic findings included changes compatible with greater oxidative substrate use. The authors proposed that the extract could support mitochondrial adaptation, but the study did not directly demonstrate a clinical increase in muscle mass or human endurance. The pathway is nevertheless relevant

because SIRT1, PPAR signaling, and PGC-1α-linked networks coordinate energy sensing, lipid oxidation, and mitochondrial remodeling.

In a second C2C12 study, Duranti et al. (2021b) found dose-dependent increases in Nrf2 and heme oxygenase-1, together with improved glutathione redox homeostasis and higher activities of catalase, superoxide dismutase, glutathione peroxidase, and glutathione transferase. Nrf2 is a transcriptional regulator of antioxidant-response genes. The results indicate activation of endogenous defense systems rather than only direct radical scavenging. This distinction is mechanistically relevant, because an adaptive cellular response may persist beyond immediate chemical neutralization; however, an in vitro response cannot establish oral bioavailability or an effective human dose.

Ceci et al. (2022) tested C2C12 myotubes exposed to a high hydrogen-peroxide challenge. Twenty-four-hour pretreatment with leaf extract improved indices of redox balance and antioxidant enzyme activity and reduced lipid and protein oxidation. The protective effect depended on pretreatment in the experimental design; simultaneous exposure was not presented as an equivalent intervention. This temporal feature supports a conditioning or response-induction hypothesis rather than a simple rescue effect after established injury.

Subsequent work extended the oxidative-stress model. The multi-origin metabolomic comparison found reduced hydrogen-peroxide-associated cell death after pretreatment with each tested leaf extract (Ceci et al., 2024a). A related 2024 study in proliferating C2C12 myoblasts reported that mild oxidative stress impaired redox balance and proliferation, whereas leaf extract improved glutathione-related measures, total antioxidant capacity, viability, and proliferative recovery (Ceci et al., 2024b). Because activated satellite cells must proliferate before differentiating during muscle repair, myoblast resilience is conceptually relevant to regeneration. Nonetheless, C2C12 proliferation is a surrogate endpoint and does not demonstrate improved regeneration in an aged or diseased human muscle.

The anti-inflammatory cellular evidence comes from seed-derived MIC-1. In LPS-treated C2C12 myoblasts, MIC-1 reduced nitric oxide and transcription of TNF-α, interferon-α, IL-1β, and IL-6, and inhibited nuclear accumulation of NF-κB (Sailaja et al., 2022). Transcriptomic analyses supported broad effects on inflammatory and immune pathways. These data connect a defined moringa compound to an established inflammatory regulator in muscle cells, but the model represents acute LPS stimulation and the compound source differs from the review's primary leaf-extract focus.

Taken together, the cellular studies are directionally coherent: leaf extracts influence antioxidant defenses, glutathione homeostasis, oxidative metabolism, and survival under oxidative challenge, while a moringa-derived isothiocyanate suppresses acute inflammatory signaling. The coherence strengthens biological plausibility. The main weaknesses are reliance on one cell line, repeated contributions from a limited number of laboratories, variable extract preparation, high artificial oxidant exposure, and absence of pharmacokinetic linkage to human supplementation.

### 3.4. Animal evidence: fatigue, atrophy-related models, ageing, and functional recovery

Forced-swimming studies provide early functional evidence. Lamou et al. (2016) administered aqueous leaf extract to male rats for 28 days and observed longer swimming endurance, greater glycogen availability, and changes in blood or tissue biomarkers compatible with reduced fatigue and improved antioxidant capacity. These findings support an antifatigue effect in that model, but forced swimming is influenced by motivation, stress response, thermoregulation, and systemic metabolism as well as skeletal-muscle capacity. It is therefore not a direct model of sarcopenia.

More recent murine studies used ethanol extract or flavonoid-rich fractions. Bian et al. (2023) evaluated an ethanol leaf extract using a weight-loaded swimming model and reported antifatigue effects linked in the abstract to energy metabolism and antioxidant capacity. Bian et al. (2024) evaluated a purified flavonoid-rich leaf fraction for 14 days and reported an approximately 60% increase in exhaustive swimming time with accompanying metabolic and antioxidant changes. Because these reports were assessed at abstract or publisher-preview level in the present review, interpretation is restricted to the stated endpoints; details not visible in those sources were not reconstructed.

Barodia et al. (2022) directly examined skeletal-muscle dysfunction induced by dexamethasone and exercise in rats. The study included six groups and used oral leaf extract at 300 mg/kg in treatment arms. Functional measurements included locomotor activity, grip-related or coordination outcomes, and endurance, with biochemical and histological assessment of muscle injury. The extract, exercise, or their combination improved several functional and tissue outcomes relative to injured controls. This model is relevant to glucocorticoid-associated catabolism, but the intervention was tested over a short preclinical period and the exercise arm complicates attribution of additive or independent effects.

Ertik et al. (2023) studied a 70% ethanolic leaf extract in rats with valproate-associated oxidative muscle injury. The abstract reports evaluation of antioxidant systems and protection against oxidative damage. This provides a pharmacotoxicological muscle model rather than a chronic wasting model. Because the full article was not used for detailed extraction, claims regarding individual enzymes, histological effect size, or dose-dependent superiority are not made here.

Protein-energy malnutrition represents a more direct cause of muscle loss. Olayinka and Bewaji (2017) used a leaf-based diet in a malnutrition model and reported protection against skeletal-muscle degeneration. The intervention was food-based rather than a chemically standardized extract, which may be clinically relevant for nutritional rehabilitation but prevents separation of protein, micronutrient, and phytochemical effects. The journal and experimental reporting also warrant cautious weighting compared with later, more extensively characterized studies.

Peripheral-nerve injury produces denervation-related muscle dysfunction. Razzaq et al. (2020) mixed crude leaf powder into chow at 200 mg/kg after sciatic-nerve crush in mice. Outcomes included sciatic functional index, grip strength, muscle mass, and sensory testing. Treated animals showed improved functional recovery. The study supports a potential role in recovery after nerve insult, but it cannot determine whether the primary target was the nerve, muscle, systemic inflammation, or nutrition. Small group sizes and the crude preparation further limit translation.

Ageing-like frailty was examined by Budiningsih et al. (2025) in a D-galactose model. Mice received 100, 200, or 400 mg/kg/day of leaf extract after induction. The 400 mg/kg dose produced the most pronounced reductions in reported MDA, TGF-β, caspase-3 expression in muscle, and muscle fibrosis, together with improved endurance. The study is notable because it integrates oxidative stress, fibrosis, apoptosis, and physical function. However, the animals were young at baseline and chemically induced ageing does not reproduce the multi-system biology of natural human ageing. The paper was a short communication, and the title's reference to muscle mass should not be interpreted as proof of clinically meaningful hypertrophy without a clearly reported change in validated body-composition or imaging outcomes.

Eze et al. (2024) treated adult male mice with moringa material for five weeks and added voluntary wheel running to half of each treatment group during the final two weeks. Moringa-treated running mice covered a greater average distance. Muscle analysis showed higher expression of several metabolic, glycolytic, angiogenic, and contractile markers, including PGC-1α, PPARγ, SDHB, SUCLG1, VEGF, PGAM-2, PGK1, and MYLPF. Cross-sectional area increased and markers of protein degradation decreased. These results are among the most directly relevant to muscle phenotype, yet the material was described by mass rather than a clinically standardized extract, only male mice were studied, and an increase in voluntary running may itself drive some molecular adaptations.

Ciuccio et al. (2026) evaluated a leaf extract in male mdx mice, a model of Duchenne muscular dystrophy. Two-month-old animals received 300 mg/kg/day by oral gavage for eight weeks. The treated mdx group showed a significant improvement in the inverted-screen strength measure and lower serum AST, while creatine kinase reduction was not statistically significant. Morphological effects were not uniform: minimum Feret diameter generally did not differ significantly, and internalized nuclei increased significantly only in extensor digitorum longus. The study therefore provides a direct disease-model signal for function and tissue injury, but not consistent evidence of hypertrophy or structural normalization. Interpretation is limited by small groups, one sex, a single dose, and limited chemical characterization of the extract.

The in vivo MIC-1 experiment complements these studies mechanistically. Oral MIC-1 at 80 mg/kg for three days reduced LPS-induced inflammatory gene expression in gastrocnemius muscle and suppressed pathways associated with immune activation (Sailaja et al., 2022). This is strong evidence for acute anti-inflammatory activity in muscle tissue, but it is seed-derived, short-term, and not an atrophy or chronic-disease model.

### 3.5. Human evidence

Human evidence is limited to small studies in healthy, active, or athletic young adults and does not address muscle-wasting disease. Tsuk et al. (2022) conducted a randomized, double-blind pilot study in 16 healthy physical-education students. Participants received a commercial supplement providing 310 mg twice daily or placebo for six weeks. The study found no between-group improvement in body composition, maximal oxygen uptake, Wingate performance, resting blood pressure, or resting heart rate. The authors advised caution regarding performance claims.

Ray et al. (2023) studied 16 taekwondo athletes using a randomized pre-test/post-test comparison in which both groups completed vigorous aerobic exercise four days per week for six weeks and the experimental

group also consumed 2,000 mg/day of moringa leaf material. The report described greater gains in push-up, sit-up, and wall-sit tests and improvements in aerobic measures in the supplemented group. The exercise co-intervention, very small sample, limited reporting of allocation concealment and blinding, and absence of a moringa-only group prevent attribution of the observed changes solely to the supplement.

Dong et al. (2024) randomized 44 young men to aqueous leaf extract or placebo for 30 days. According to the abstract and publisher preview, the supplemented group showed better push-up and treadmill-to-exhaustion performance and favorable changes in selected serum energy-metabolism and oxidative-stress measures. The report provides a positive human signal, but it remains a pilot study in healthy men. Detailed claims not visible in the accessible record, including product dose, adherence, dietary control, complete adverse-event data, or subgroup findings, were not inferred.

Gbedinhessi et al. (2025) used a randomized, double-blind crossover protocol in 34 active adults who consumed a moringa leaf-powder infusion or placebo for seven days before a 20-km cycling time trial. The full report described statistically significant differences in cadence, total work, completion time, and peak heart rate, while several other physiological measures were not clearly changed. The numerical direction and interpretation of some reported time and work outcomes were not entirely intuitive, and the intervention concerned short-term endurance performance rather than muscle mass, strength loss, or disease.

The four human studies used different preparations, doses, exercise programs, durations, populations, and endpoints. Their findings therefore cannot be combined as evidence of a reproducible treatment effect. None evaluated older adults, patients with low muscle strength, objectively confirmed sarcopenia, cachexia, denervation, glucocorticoid myopathy, muscular dystrophy, or inflammatory muscle disease. No human study identified in the search used imaging-based muscle quantity, standardized sarcopenia outcomes, muscle biopsy, or long-term disability as a primary endpoint.

**Table 1.** Direct or mechanistically relevant evidence on Moringa oleifera and skeletal muscle

| Study | Model and material | Main muscle-relevant findings | Evidence access | Key limitations |
|---|---|---|---|---|
| Lamou et al., 2016 | Male rats; aqueous leaf extract; forced-swimming model | Longer endurance; glycogen and antioxidant/fatigue-marker changes | Full text | Performance model; systemic effects; no atrophy |
| Olayinka & Bewaji, 2017 | Protein-energy malnutrition model; leaf-based diet | Reported protection from skeletal-muscle degeneration | Full text | Food matrix, not standardized extract; limited reporting |
| Razzaq et al., 2020 | Mouse sciatic-nerve crush; crude leaf powder in chow, 200 mg/kg | Improved motor recovery, grip-related outcomes, and muscle-associated measures | Full text | Small groups; nerve versus muscle mechanism unresolved |
| Duranti et al., 2021a | C2C12 myotubes; leaf extract | Increased oxidative metabolism associated with SIRT1–PPARα signaling | Full text | Single cell line; no in vivo exposure link |
| Duranti et al., 2021b | C2C12 myotubes; leaf extract | Nrf2/HO-1 upregulation; improved glutathione and antioxidant enzyme activity | Full text | In vitro surrogate endpoints |
| Barodia et al., 2022 | Rats; dexamethasone/exercise injury; 300 mg/kg leaf extract | Improved functional, biochemical, and histological outcomes in several treatment arms | Full text | Short duration; multiple interventions |

| Study | Model and material | Main muscle-relevant findings | Evidence access | Key limitations |
|---|---|---|---|---|
| Ceci et al., 2022 | C2C12 myotubes; H2O2 challenge; leaf-extract pretreatment | Restored redox measures and reduced lipid/protein oxidation | Full text | Pretreatment design; artificial oxidant exposure |
| Sailaja et al., 2022 | C2C12 myoblasts and LPS-treated mice; seed-derived MIC-1 | Reduced inflammatory cytokine transcription and NF-κB nuclear translocation | Full text | Not leaf extract; acute inflammation model |
| Tsuk et al., 2022 | 16 healthy adults; 310 mg twice daily commercial supplement, 6 weeks | No improvement in body composition, VO2max, or Wingate outcomes | Full text | Very small pilot; product characterization limited |
| Ray et al., 2023 | 16 taekwondo athletes; exercise plus 2,000 mg/day leaf material, 6 weeks | Reported greater gains in muscular-endurance tests and aerobic measures | Publisher article/PDF | Exercise co-intervention; small sample; attribution uncertain |
| Ertik et al., 2023 | Rats; valproate-induced muscle oxidative injury; 70% ethanolic leaf extract | Abstract reports antioxidant protection in muscle tissue | Abstract/preview only | No detailed extraction beyond abstract |
| Bian et al., 2023 | Mice; ethanol leaf extract; weight-loaded swimming | Abstract reports antifatigue effects involving energy and antioxidant pathways | Abstract/preview only | Exercise-fatigue model; limited accessible detail |
| Bian et al., 2024 | Mice; purified leaf flavonoid concentrate, 14 days | Approximately 60% longer exhaustive swimming time and metabolic/antioxidant changes | Full publisher text | Purified fraction; no wasting model |
| Ceci et al., 2024a | C2C12 myotubes; extracts from multiple geographic origins | All tested extracts reduced H2O2-associated cell death | Full text | Common-garden and cell model; not commercial-product equivalence |
| Ceci et al., 2024b | C2C12 myoblasts; oxidative stress; leaf extract | Improved redox homeostasis, viability, and proliferative recovery | Full text | Surrogate for regeneration; no aged/diseased muscle |
| Dong et al., 2024 | 44 young men; aqueous leaf extract, 30 days | Abstract reports improved push-up and treadmill-exhaustion performance | Abstract/preview only | Pilot; healthy men; detailed product data not extracted |
| Eze et al., 2024 | Adult male mice; moringa plus/minus voluntary running, 5 weeks | Greater running distance; metabolic-marker, CSA, and protein-degradation changes | Publisher HTML/preview | Male mice; exercise confounding; product standardization |
| Budiningsih et al., 2025 | D-galactose frailty model; leaf extract 100–400 mg/kg/day | Lower oxidative, fibrotic, and apoptotic markers; improved endurance at 400 mg/kg | Full text | Chemical ageing model; short communication; no human validation |

| Study | Model and material | Main muscle-relevant findings | Evidence access | Key limitations |
|---|---|---|---|---|
| Gbedinhessi et al., 2025 | 34 active adults; randomized double-blind crossover; leaf-powder infusion, 7 days | Differences in cadence, work, time, and peak heart rate during 20-km cycling | Full text | Short-term performance; outcome direction/reporting requires caution |
| Ciuccio et al., 2026 | Male mdx mice; leaf extract 300 mg/kg/day by gavage, 8 weeks | Improved inverted-screen strength and lower AST; inconsistent morphological changes | Full text | Small groups; one sex/dose; no uniform structural benefit |

### 3.6. Mechanistic pathways emerging from the evidence

The mechanistic literature can be organized into five interacting domains. First, Nrf2–HO-1 and glutathione-related responses may increase the capacity of muscle cells to tolerate oxidative challenge. Second, SIRT1, PPARα, PPARγ, and PGC-1α-associated findings suggest effects on substrate selection, mitochondrial function, and exercise adaptation. Third, NF-κB suppression and lower inflammatory cytokine expression may reduce catabolic signaling in inflammatory states. Fourth, changes in caspase-3, TGF-β, and fibrosis markers suggest possible modulation of apoptosis and maladaptive tissue remodeling. Fifth, increased cross-sectional area and lower protein-degradation markers in mice raise the possibility of effects on protein balance, although direct measurement of synthesis and breakdown was limited.

These domains should not be regarded as independent therapeutic targets proven in humans. Redox and inflammatory pathways are context-dependent. Reactive oxygen species participate in normal exercise adaptation and myogenesis at physiological levels, while excessive antioxidant exposure can theoretically blunt adaptive signaling. Similarly, NF-κB participates in host defense as well as chronic catabolism. The desirable intervention would normalize dysregulated signaling rather than abolish it. Current moringa studies do not define the dose range at which adaptive, neutral, or counterproductive effects occur in humans.

**Table 2.** Proposed muscle-relevant mechanisms and evidential boundaries

| Mechanistic domain | Supporting observations | Potential relevance | Boundary of inference |
|---|---|---|---|
| Nrf2/HO-1 and antioxidant enzymes | Higher Nrf2, HO-1, CAT, SOD, GPx, GST; improved GSH/GSSG in C2C12 cells | Protection from excessive oxidative stress and macromolecular damage | Predominantly in vitro; oral human exposure not linked to cell concentrations |
| SIRT1–PPAR and PGC-1α networks | C2C12 oxidative-metabolism findings; higher PGC-1α/PPAR-related markers in mice | Mitochondrial remodeling, lipid oxidation, endurance adaptation | Marker changes do not prove new functional mitochondria or clinical benefit |
| NF-κB and cytokine signaling | MIC-1 reduced NF-κB nuclear translocation and TNF-α, IL-1β, IL-6 expression | Lower inflammatory catabolism in selected contexts | Seed-derived compound; acute LPS model; human chronic inflammation untested |
| Apoptosis and fibrosis | Lower caspase-3 and TGF-β/fibrosis markers in D-galactose mice | Preservation of tissue architecture in ageing-like stress | Chemical model; limited replication and no human imaging/biopsy |
| Protein turnover and fibre size | Higher CSA and lower degradation markers in supplemented mice | Possible anti-atrophic or anabolic support | Exercise confounding; synthesis rates and disease models needed |
| Energy availability and fatigue | Glycogen, lactate-related or endurance changes in swimming/running models | Delayed fatigue and improved work capacity | Endurance is not equivalent to reversal of wasting |

## 4. Discussion

### 4.1. Overall interpretation

The evidence supports biological plausibility but not clinical efficacy for muscle-wasting disorders. Across cell experiments, leaf extract repeatedly improved redox-related outcomes and influenced energy-metabolism pathways. Across animal models, supplementation frequently improved endurance or recovery and altered markers associated with oxidative injury, inflammation, fibrosis, apoptosis, mitochondrial regulation, or protein degradation. Directional consistency is therefore stronger than would be expected from a single isolated experiment. However, consistency of direction does not overcome limitations in model relevance, product heterogeneity, and absence of confirmatory clinical trials.

The distinction between performance enhancement and anti-wasting therapy is central. A healthy mouse that runs farther after supplementation has not demonstrated reversal of sarcopenia. A myotube protected from hydrogen peroxide has not demonstrated preserved strength in a patient with cancer cachexia. A reduction in cytokine expression after acute LPS exposure does not establish benefit in chronic inflammatory disease. The current literature is best interpreted as a mechanistic and preclinical platform from which disease-specific hypotheses can be developed.

Among the proposed pathways, the most reproducible direct evidence concerns redox homeostasis. Nrf2/HO-1 activation, glutathione balance, and antioxidant enzymes were assessed in multiple related C2C12 experiments. This pathway is relevant to many forms of muscle injury, but it is not unique to moringa and may represent a general cellular response to phytochemicals. The SIRT1–PPARα and PGC-1α-related findings add a metabolic dimension and may explain some endurance observations. Anti-inflammatory evidence is compelling for MIC-1 but less directly transferable to leaf products because the compound was obtained from seeds.

The apparent effect on cross-sectional area and protein-degradation markers in mice is particularly relevant to muscle wasting, but it needs independent replication. Future studies should measure fractional protein synthesis, ubiquitin–proteasome and autophagy flux, myostatin/activin signaling, anabolic resistance, satellite-cell function, and contractile force. Such measurements would determine whether moringa merely changes transcriptional markers or meaningfully shifts muscle protein balance.

### 4.2. Quality and limitations of the reviewed research

The first limitation is concentration of evidence in preclinical models. C2C12 cells are useful for controlled mechanistic experiments but do not reproduce ageing, systemic disease, endocrine regulation, innervation, immune-cell interactions, digestion, metabolism, or product bioavailability. Rodent models allow tissue-level assessment, yet most used young male animals, short interventions, and induced stressors. Sex, age, baseline nutritional status, comorbidity, and concurrent medication were rarely modeled in a way that resembles the target clinical population.

The second limitation is intervention heterogeneity. Studies used aqueous, methanolic, ethanolic, or unspecified extracts; crude leaf powder; leaf-based diets; flavonoid concentrates; commercial capsules; and seed-derived MIC-1. Reporting of botanical authentication, voucher specimens, harvest conditions, extraction yield, marker compounds, contaminants, and batch consistency varied. This prevents a meaningful dose comparison and makes replication difficult. Even when the same plant species is used, the effective exposure to glucosinolates, isothiocyanates, polyphenols, and nutrients may differ by orders of magnitude.

The third limitation concerns outcome selection. Many studies emphasized swimming time, running distance, oxidative biomarkers, or gene expression. These outcomes are useful but susceptible to systemic and behavioral influences. Direct measures of muscle force, fibre-type-specific contractility, validated imaging, lean mass, histomorphometry, and longitudinal functional disability were less common. In the human studies, performance tests differed, exercise was sometimes co-administered, and sample sizes were too small to establish efficacy or safety.

The fourth limitation is publication and laboratory bias. Positive botanical studies may be more likely to be published than neutral experiments. Several mechanistic papers came from overlapping research groups and related C2C12 protocols, so they should not be treated as fully independent replications. Conversely, the negative Tsuk pilot is important because it demonstrates that plausible preclinical mechanisms do not guarantee measurable performance benefit, while the positive studies remain too heterogeneous for replication to be assumed.

The fifth limitation arises from source access. Several relevant papers were available in full text, permitting detailed appraisal. Others were available only through abstracts or publisher previews. For those studies, this review deliberately avoided reconstructing methods or adverse events from secondary

descriptions. The resulting caution may omit potentially useful detail, but it reduces the risk of presenting inferred information as fact.

Finally, this is a narrative rather than systematic review. Although the search strategy and eligibility criteria were specified, no formal dual-reviewer screening, risk-of-bias instrument, certainty grading, or meta-analysis was performed. The review is therefore intended to map and critically interpret the evidence, not to provide a definitive quantitative effect estimate.

### 4.3. Clinical practice and Nutrition Care Process implications

No identified evidence supports prescribing *M. oleifera* leaf extract as treatment for sarcopenia, cachexia, glucocorticoid myopathy, denervation atrophy, muscular dystrophy, inflammatory myopathy, or critical-illness weakness. Established clinical management should remain directed at the underlying disease, adequate energy and protein intake, resistance exercise or rehabilitation when feasible, correction of deficiencies, and guideline-based multidisciplinary care. Moringa should not displace these interventions.

For dietitians and other clinicians, the Nutrition Care Process provides a practical framework. Assessment should document the exact product, plant part, extract ratio, dose, frequency, duration, reason for use, other supplements, medication exposure, nutritional status, muscle strength or function, and the patient's expectations. The nutrition diagnosis should address actual problems such as inadequate protein-energy intake, malnutrition, or unintended weight loss rather than attributing them to a presumed need for a botanical. Intervention should prioritize evidence-based nutrition and exercise strategies. Monitoring should use clinically meaningful outcomes—weight trajectory, dietary adequacy, grip strength, gait speed, physical performance, and validated body-composition measures—rather than nonspecific antioxidant claims.

Product safety also requires caution. Leaf foods and concentrated extracts are not equivalent exposures. Botanical supplements may contain variable active compounds or contaminants and may alter drug metabolism. A pharmacokinetic study has reported an interaction signal between leaf powder and nevirapine exposure, illustrating that "natural" does not mean pharmacologically inert (Monera-Penduka et al., 2017). Patients receiving antiretrovirals, anticoagulants, antidiabetic agents, antihypertensives, anticonvulsants, immunosuppressants, or drugs with narrow therapeutic windows should discuss supplementation with a clinician or pharmacist. Pregnancy, liver or kidney disease, and perioperative use require additional caution because direct safety data for concentrated products are limited.

In healthy adults seeking performance enhancement, the evidence remains insufficient for a firm recommendation. One study was negative and three reported selected positive performance signals, but the preparations, exercise co-interventions, durations, and endpoints differed substantially. Marketing statements should not generalize animal doses or cellular findings to humans. If a person independently chooses a product, clinicians should encourage transparent labeling, third-party quality testing, avoidance of multi-ingredient blends that obscure attribution, and discontinuation if adverse symptoms occur.

### 4.4. Priorities for future research

Future development should proceed in stages rather than moving directly from cell findings to broad consumer claims. The following priorities emerge from the reviewed evidence:

- Define the intervention. Studies should authenticate the species and plant part, report cultivation and harvest conditions, specify solvent and extraction ratio, quantify extraction yield, provide a chemical fingerprint, and identify marker compounds such as glucomoringin, moringin/isothiocyanates, quercetin derivatives, and phenolic acids. Heavy metals, pesticides, microbes, and adulterants should be assessed.
- Establish pharmacokinetics and target exposure. Oral studies should determine which parent compounds or metabolites reach plasma and muscle, their time course, and whether concentrations used in C2C12 experiments are physiologically achievable.
- Use disease-relevant models. Natural ageing, cancer cachexia, chronic kidney or heart disease, chronic glucocorticoid exposure, immobilization, denervation, and inflammatory muscle disease should be studied with both sexes and appropriate ages. Pair-fed controls are needed where appetite or body weight could confound results.
- Measure muscle directly. Experiments should include muscle weight normalized appropriately, fibre cross-sectional area, force production, fatigue resistance, protein synthesis and degradation, mitochondrial respiration, capillary density, satellite-cell activity, and histological fibrosis. Molecular markers should accompany rather than replace functional endpoints.
- Clarify interaction with exercise and nutrition. Factorial studies should compare moringa alone, resistance or endurance exercise alone, adequate protein or essential amino acids alone, and combinations.

This would determine whether the extract adds benefit or merely reproduces adaptations driven by exercise and sufficient nutrition.

- Conduct staged human trials. Initial standardized-product studies should assess tolerability, pharmacokinetics, and biomarker response. Later randomized trials should enroll older adults or patients with a clearly defined muscle-wasting phenotype and use prespecified outcomes such as grip strength, chair-rise performance, gait speed, Short Physical Performance Battery, DXA or CT/MRI muscle measures, and patient-centered function.
- Improve transparency. Protocol registration, CONSORT-compliant reporting, prespecified primary outcomes, adequate allocation concealment and blinding, complete adverse-event reporting, independent replication, and publication of neutral results are essential.

### 4.5. Suggested positioning of the research niche

A broad title such as "Moringa and musculoskeletal health" would substantially overlap with existing work. A performance-only review would also overlap with the 2025 systematic review. The stronger scholarly position is a mechanistic-translational review of leaf-derived preparations in skeletal muscle wasting and dysfunction. The unique contribution should be explicit: direct muscle evidence only; separation of leaf extract, leaf powder, and seed-derived compounds; distinction between fatigue and atrophy; transparent labeling of full-text versus abstract-only evidence; and a clinical conclusion that therapeutic efficacy remains unproven.

A suitable final title is the one used in this manuscript: "Moringa oleifera Leaf Extract in Muscle Wasting and Skeletal Muscle Disorders: Mechanisms, Evidence, and Therapeutic Potential—A Narrative Review." An even more cautious alternative for submission is "Moringa oleifera Leaf-Derived Preparations in Skeletal Muscle Wasting and Dysfunction: Mechanistic Evidence, Translational Gaps, and Therapeutic Potential—A Narrative Review." The latter better reflects that several studies used leaf powder or non-standardized material rather than a single extract.

## 5. Conclusions

*Moringa oleifera* leaf-derived preparations influence several pathways relevant to skeletal-muscle health in experimental systems. Direct cellular studies support effects on Nrf2/HO-1 signaling, glutathione and antioxidant enzymes, oxidative metabolism, and resilience to oxidative stress. Animal studies report improved endurance or functional recovery and changes in inflammatory, fibrotic, apoptotic, metabolic, and protein-degradation markers. These findings justify continued investigation, particularly in models of ageing and disease-related atrophy. They do not yet justify clinical treatment claims. Human evidence consists of four small, heterogeneous studies in young or active adults, and no trial identified in this review tested a standardized product in a population with confirmed muscle wasting. The major barriers are heterogeneous preparations, incomplete chemical standardization, uncertain bioavailability, limited safety and interaction data, and insufficient clinically meaningful outcomes. At present, moringa should be regarded as a research candidate and possible nutritional adjunct, not an established therapy for sarcopenia or skeletal-muscle disorders.

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