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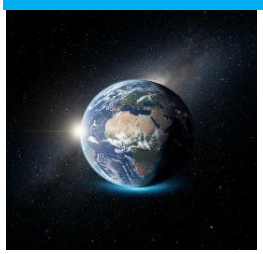

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Impact of Creatine Supplementation on Muscle and Bone Strength in Older Adults: A Narrative Review

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Abstract

Introduction and purpose: Age-related declines in skeletal muscle mass and strength, together with bone loss, reduce physical performance and increase frailty and fall risk in older adults. Creatine supplementation has been proposed as a supportive and potentially preventive strategy due to its role in cellular energy buffering and its capacity to augment exercise training

adaptations. This narrative review summarizes current evidence on the effects of creatine supplementation on muscle strength and function and on bone-related outcomes in older adults.
Review methods: A narrative review of the literature was conducted using PubMed and Google Scholar. Peer-reviewed studies published between 2000 and 2025 examining creatine supplementation and muscle- or bone-related outcomes in adults were included.
Brief description of the state of knowledge: Evidence from randomized trials and meta-analyses indicates that creatine supplementation combined with progressive resistance training increases lean mass and improves dynamic strength and functional performance in older adults more consistently than resistance training alone. Mechanistically, benefits are biologically plausible through increased intramuscular creatine/phosphocreatine availability, improved high-intensity exercise capacity, and downstream anabolic signaling that supports training responsiveness. In contrast, effects on areal bone mineral density measured by DXA are generally neutral in long-term trials and pooled analyses.
Summary (conclusions): Creatine is a well-studied supplement that, when paired with resistance training, reliably enhances lean mass and muscle strength in older adults. Current evidence does not support creatine as an effective stand-alone strategy to increase areal BMD, but potential benefits warrant further adequately powered, long-duration trials, particularly in sarcopenic and osteopenic populations.

Key words: creatine, ageing, resistance training, muscle mass, sarcopenia, dynapenia, osteoporosis

1. Introduction

Aging leads to progressive declines in skeletal muscle strength and mass as well as bone density(1). These changes contribute to reduced physical capacity(2), impaired mobility, increased functional dependence, and diminished quality of life in older adults(3).

As global populations continue to age(4), the clinical and public health impact of musculoskeletal decline is becoming increasingly significant. Rising life expectancy means that a growing proportion of older adults are exposed for longer periods to age-associated muscle weakness and bone fragility, resulting in increased risks of falls, fractures, hospitalization, and long-term care dependency(5). These trends underscore the need for effective, accessible, and evidence-based interventions that preserve musculoskeletal function and support healthy aging.

Sarcopenia is recognised as an important manifestation of age-related musculoskeletal decline(6). While no single definition exists, several international and regional expert groups have established their criteria. The European Working Group on Sarcopenia in Older People (EWGSOP/EWGSOP2) and the Foundation for the National Institutes of Health (FNIH) Sarcopenia Project have each specified diagnostic criteria used widely in Europe and globally. Despite methodological differences, both these groups describe sarcopenia through two key features: reduced muscle mass and diminished muscle strength(6,7). The related term dynapenia is often used to specifically denote the age-associated loss of muscle strength, highlighting the importance of strength decline as a core component of functional impairment(7).

Prevalence estimates highlight the scale of this challenge. Muscle weakness and mobility limitations affect a substantial proportion of older adults, increasing sharply with advancing age. Sarcopenia affects approximately 10–27% of adults(8). In parallel, age-related bone loss further contributes to morbidity and mortality in older populations. Osteoporosis and fragility fractures represent major public health concerns, particularly among postmenopausal women and older men, and are associated with substantial healthcare utilization and long-term disability(9). The close interrelationship between muscle weakness, impaired balance, and skeletal fragility reinforces the concept of a coupled muscle–bone unit, in which deterioration of one tissue amplifies vulnerability of the other.

Creatine supplementation has emerged as a promising intervention to counteract aspects of age-related musculoskeletal decline(10). Creatine is one of the most extensively studied nutritional supplements in exercise science and clinical research, largely due to its central role in cellular energy metabolism and its established ability to enhance adaptations to resistance training(11). As a key component of the phosphocreatine system, it supports rapid ATP regeneration(12), enhances training capacity, and contributes to improved muscle energetics. Importantly, intramuscular creatine stores tend to decline with age(13), suggesting that older adults may derive particular benefit from supplementation. These considerations provide a strong rationale for examining creatine's potential to support muscle and bone health in later life.

This review synthesises current evidence on the effects of creatine supplementation on muscle and bone strength in older adults, with emphasis on its clinical relevance, preventive potential, and key gaps that warrant further investigation.

2. Research materials and methods

A literature review was conducted using PubMed, Google Scholar, and ResearchGate. Search terms included: creatine, creatine monohydrate, aging, older adults, sarcopenia, muscle strength, tendon, osteopenia and bone mineral density. Articles published between 2000 and 2025 were considered. Priority was given to peer-reviewed original studies, systematic reviews, and meta-analyses written in English. Reference lists of selected papers were also screened to identify additional relevant sources. Only studies addressing the effects of creatine supplementation on muscle, tendon, or bone health in adults were included in this review.

3. Research results

Cellular and Molecular Effects of Creatine

In humans, the majority of total body creatine is stored in skeletal muscle (commonly reported as ~95%), with smaller pools distributed across tissues with high energy turnover such as the brain, heart, liver, and kidneys(12,14). Despite the predominance of storage in skeletal muscle, endogenous creatine synthesis occurs primarily in the kidney and liver, with contributions from other tissues including the pancreas(12,14). The total body creatine pool in an average adult male is commonly reported at ~120 g (free creatine plus phosphocreatine), with substantial individual variability largely driven by muscle mass(15). Creatinine and creatine phosphate are spontaneously converted to creatinine at a rate approximately 2% of total bodily creatine per day or approximately 2 g(15,16). According to Brosnan et al.(17) daily intake of creatine is about 1 g, with the remainder synthesized endogenously. The endogenous synthesis requires three different amino acids: glycine, arginine, and methionine with the synthesis of 1 g consuming a substantial amount (about 16%) of daily dietary glycine intake(17). Consistent with the absence of dietary creatine in plant-based foods, individuals consuming vegetarian or vegan diets rely almost entirely on endogenous synthesis and typically exhibit lower baseline intramuscular creatine stores, which is relevant because baseline status may modulate the magnitude of response to supplementation(15,18).

At the cellular level, creatine is transported into skeletal muscle via the high-affinity sodium- and chloride-dependent creatine transporter(19).Once inside the cell, creatine participates in a single, reversible reaction catalysed by creatine kinase (CK) isoenzymes (cytosolic and mitochondrial), which interconverts creatine and phosphocreatine and buffers ATP/ADP during fluctuating energy demand(17,19). This Cr–PCr system is commonly conceptualised as having three integrated roles: 1) a temporal buffer, 2) a spatial buffer (or “phosphocreatine shuttle”),

and 3) a metabolic regulator(19). The first aspect refers to tissues where the energy demand highly fluctuates in time, such as in the skeletal or heart muscle tissue (which contain a major share of body creatine reserves). In the muscle tissue after the ATP expenditure during contraction the ATP reserves are readily replenished and the ADP levels are kept low due to the presence of CK near the sites of ATP utilisation (myofibrils). Thus the ATP levels are kept stable in time during muscle work(20). The spatial buffering function reflects the coupling between mitochondrial ATP production and cytosolic ATP utilisation: mitochondrial CK facilitates PCr formation near mitochondria, PCr then diffuses through the fibre, and cytosolic CK regenerates ATP in proximity to ATPases (e.g., at the myofibrils)(21).

The third function of the Cr–PCr system is its role as a metabolic regulator. In this context, creatine supplementation has been linked to molecular changes consistent with enhanced anabolic capacity. A randomized placebo-controlled trial in humans showed that creatine supplementation markedly upregulated expression of mRNA and proteins involved in processes related to protein synthesis, satellite cell activity, DNA replication/repair, and cell survival; these biopsy-level changes were accompanied by increased fat-free mass and total body water in the creatine group(22). This changes cells are achieved via various mechanisms.

One proposed mechanism is creatine's osmotic effect: increased intracellular creatine increases cell water content, which can activate osmosensing pathways and shift signalling toward protein synthesis - anabolism(23,24). Importantly, creatine also appears to affect signalling independent of cell swelling, because other osmotic agents do not consistently reproduce these responses. Experimental work supports activation of pathways implicated in myogenic differentiation and hypertrophy, including p38 and Akt/PKB–p70S6K signalling(25) with other research pointing to activation of the IGF pathway potentially mediating the increase in muscle dry mass (26,27).Finally, creatine has been discussed as having anti-inflammatory and anti-catabolic effects in certain contexts(28), although other evidence suggests that these effects may be contingent on limited calorie intake(29).

Aging is associated with altered muscle energetics. Evidence from human studies suggests mild reductions in intramuscular PCr content and/or slower PCr recovery kinetics with age, with more pronounced alterations in sarcopenic muscle phenotypes(30). Importantly, creatine supplementation increases muscle total creatine and PCr availability in both young and older adults, and responses may be larger in individuals with lower baseline stores(31).

This supports the concept that age-related energetic constraints are at least partially modifiable, particularly in contexts where baseline creatine status is reduced.

The mechanistic considerations also help explain endpoint-specific effects in clinical trials related to creatine. The phosphocreatine system is most stressed during short-duration, high-intensity, repeated contractions where rapid ATP resynthesis is rate-limiting(32). This aligns with the observation that creatine's benefits are most reproducible for resistance-training outcomes that involve repeated sets with incomplete recovery, and for multi-joint lower-limb tasks, whereas "single brief maximal isometric" outcomes (e.g., a one-off handgrip squeeze) may show smaller or more variable effects because they are less constrained by repeated PCr resynthesis demands. Put differently, the PCr system's relevance increases when performance depends on repeated high-intensity efforts and recovery of PCr between efforts, which resembles typical resistance-training and functional sit-to-stand paradigms more than a single static handgrip trial(32,33).

While skeletal muscle is the dominant creatine reservoir, creatine-dependent bioenergetics may also be relevant to bone cells because bone remodeling is an energy-requiring process. Theoretical and preclinical discussions propose that improved cellular energy buffering could support osteoblast activity and alter remodeling balance, and animal models have reported improved bone mechanical properties with creatine in some settings(10,34). In addition, broader anti-catabolic/anti-inflammatory effects described for creatine could plausibly influence bone turnover indirectly via reduced inflammatory signaling(35,36).

A practical point, particularly relevant in geriatric populations, is that creatine supplementation can increase serum creatinine due to increased creatinine generation from a larger creatine pool, which can artifactually lower creatinine-based eGFR estimates without reflecting true renal injury(37). Across studies that directly assessed renal function using more robust approaches, creatine supplementation in generally healthy individuals has not been shown to cause renal damage, and meta-analytic summaries report no meaningful adverse effect on measured GFR overall(38). Clinically, this supports two implications for older adults: 1) a decline in creatinine-based eGFR after starting creatine should be interpreted cautiously, and 2) if renal safety needs confirmation, clinicians should consider confirmatory testing strategies beyond creatinine-based eGFR alone (e.g., cystatin C–based estimates or measured clearance, depending on context)(37,38).

Effects of Creatine Supplementation on Skeletal Muscle in Older Adults

A foundational meta-analysis from 2014 by Devries et al. (39) examined 357 adults aged 55 years and older and compared the effects of creatine supplementation combined with resistance training versus resistance training alone on skeletal muscle outcomes. The pooled analysis

demonstrated statistically significant increases in total body mass and fat-free mass in the creatine groups. In terms of dynamic strength, creatine supplementation in conjunction with resistance training resulted in significantly greater improvements in leg press and chest press strength compared with resistance training plus placebo. Importantly, a significant benefit was also observed for functional performance, assessed using the 30-second chair stand test, highlighting potential clinical relevance beyond isolated strength measures.

A subsequent meta-analysis published in 2017 by Chilibeck et al. (40) expanded the evidence base to 721 participants aged 57 years and older and reported largely concordant findings, confirming greater gains in lean tissue mass and muscular strength when creatine supplementation was combined with resistance training. Building on this work, the meta-analysis by Forbes et al. (41) broadened the subject with consideration on creatine dosing strategies. This analysis demonstrated that both lower (<5 g/day) and higher (≥5 g/day) daily creatine doses significantly increased lean tissue mass compared with placebo when combined with resistance training. Strength outcomes varied by protocol: upper-body strength (chest press) improved significantly in studies using a creatine loading phase followed by lower maintenance doses, whereas improvements in lower-body strength (leg press) were more consistently observed in protocols employing a loading phase followed by higher daily maintenance doses. While these findings suggest that dosing strategies may influence strength adaptations, the authors emphasized substantial heterogeneity across trials, precluding firm recommendations regarding optimal dosing for specific strength outcomes.

Evidence for creatine supplementation in the absence of structured resistance training is less consistent. Meta analysis by Candow et al (36) also looked into effects of creatine supplementation without resistance training in an aging population. The results were mixed with higher doses of creatine reducing body fatigue and low doses as well as acute bolus ingestion failing to significantly improve performance. Collectively, these findings indicate that creatine's effects on skeletal muscle are most robust and reproducible when supplementation is combined with progressive resistance exercise.

Although most meta-analyses have relied on leg press and chest press one-repetition maximum tests as proxies for muscle strength, contemporary sarcopenia definitions place greater emphasis on handgrip strength and chair rise performance. The European Working Group on Sarcopenia in Older People (EWGSOP2) highlights these measures as key predictors of adverse outcomes, disability, and reduced quality of life. (6).In this context, a recent systematic review and meta-analysis by Davies et al. published in 2024 (42) pooled randomized controlled trials assessing creatine supplementation and physical performance measured by sit-to-stand tests in older

adults and populations at risk of functional disability. Creatine supplementation was associated with a significant improvement in sit-to-stand performance compared with placebo, with a pooled standardized mean difference of 0.51 (95% CI: 0.01–1.00; p = 0.04), corresponding to a moderate effect size. Bayesian analysis further suggested a 66.7% probability that creatine improves physical function in this population, supporting earlier observations reported by Devries et al. (39).

In contrast, the impact of creatine supplementation on handgrip strength remains inconsistent. While the 2024 analysis by Davies et al. (39) reported improvements in handgrip strength as a secondary outcome in mixed cohorts that included patients with chronic disease, meta-analyses focused specifically on older adults have generally not demonstrated a statistically significant benefit. The largest and most recent strength-focused meta-analysis reported no significant improvement in handgrip strength with creatine supplementation in older adults (weighted mean difference 4.26 kg, p = 0.10) (33). These findings suggest that any effect of creatine on handgrip strength may be small, context-dependent, or limited to specific subgroups, and that lower-limb strength and functional performance measures may be more responsive endpoints in aging populations.

Taken together, the available evidence indicates that creatine supplementation, particularly when combined with resistance training, consistently enhances lean tissue mass, dynamic strength, and chair-rise performance in older adults. However, most randomized trials have enrolled generally healthy older individuals rather than cohorts with formally diagnosed sarcopenia according to EWGSOP2 criteria. As a result, while the observed improvements in lower-limb strength and functional performance are highly relevant to sarcopenia-related disability, further trials specifically targeting sarcopenic populations and employing standardized diagnostic endpoints are needed to clarify the therapeutic role of creatine in this condition.

Effects of Creatine Supplementation on Bone Health in Older Adults

Studies on animal models found varying results when it comes to the influence of creatine on bone strength. One study found that rats fed with creatine enriched diet significantly increased their lumbar bone mineral density while distant femoral density was not significantly greater comapred compared to normal diet. Despite that the load to failure for rats' femurs assessed post mortem was significalntly higher in group consuming creatine (43). Another study which assessed response to creatine supplementation specifically in a rat model of osteoporosis found it had no effect on bone mass (44). Similarly human studies trials in humans found mixed results.

In a randomised trial of 237 post-menopausal women creatine supplementation with resistance training over 2 years had no effect on BMD at the femoral neck, total hip, or lumbar spine compared to placebo with resistance training. However the creatine group preserved significantly geometric properties of the proximal femur such as sectional modulus and buckling ratio at the narrow part of the femoral neck, and cortical thickness, subperiosteal width, section modulus, and buckling ratio at the femoral shaft. Those properties were observed to deteriorate in the control group (45). The geometric properties of the femur were found to be significantly predictive of incidental hip fractures (46) with some of them like the buckling ratio being independent from bone mineral density in multivariate modeling (47). It is important to note that areal bone mineral density assessed by DXA represents only one component of bone strength and does not fully capture structural and geometric properties that contribute independently to fracture resistance (48). Another randomised trial assessed creatine's impact in the group of post menopausal women already diagnosed with osteopenia. Similarly to results from the animal model after two years there were no improvements observed in terms of bone health (49). When it comes to studies in older men a small randomised trial of 29 men of median age of 71 showed a significant increase in leg bone mineral density after 12 weeks with creatine and resistance training (50). However another small randomised trial with a longer 8 month observation time of healthy ageing adults found no benefit from creatine supplementation irrespective of dosing timing (51). Another clinical trial studied seventy people (both men and women) with mean age of 58 and assessed other bone geometry parameters with creatine supplementation significantly increasing total bone area in the distal tibia and tibial shaft compared to placebo (52). The big limitation of the previously mentioned trials was the low sample size allowing for spuriously significant results and limiting the detection of significant differences at the same time. Notably, few of the available randomized trials systematically assessed biochemical markers of bone turnover, limiting insight into whether creatine supplementation exerts subtle effects on bone remodeling processes that may not be captured by DXA-derived endpoints (36). Two major meta analyses were studied to report pooled results. A paper by Forbes at al (53) included 5 trials with a total of 193 participants above the age of 50 or post-menopausal. It found no significant effects of creatine vs placebo with resistance training in terms of whole body, hip, femoral neck and lumbar spine bone mineral density. A recent meta analysis by Sharifian et al (54) included a total of 1093 adults at the age of 55 or older. It analyzed randomised trials with intervention lasting at least two weeks where a group with creatine supplementation was compared to placebo with both groups on the same training regimen. The study showed no significant difference in bone mineral density between groups.

The authors stress that heterogeneity in intervention periods, creatine dosing regiments as well as training programmes in the studies pooled for the meta analysis necessitate further research into the subject. Current evidence does not support creatine supplementation as an effective strategy to increase areal bone mineral density in older adults. However, when combined with resistance training, creatine may help preserve or improve bone geometric properties associated with hip strength, suggesting a potential indirect role in fracture risk reduction that warrants further long-term, adequately powered trials.

4. Discussion

Across the available randomized trials and meta-analyses, the most consistent and clinically meaningful signal is that creatine supplementation potentiates adaptations to resistance training in older adults, improving lean mass and dynamic strength and translating to better performance in functional tasks such as sit-to-stand and chair-rise assessment which are closely aligned with contemporary sarcopenia frameworks and with disability risk. The mechanistic rationale for these effects is coherent: creatine increases intramuscular creatine/phosphocreatine availability, supporting rapid ATP resynthesis during repeated high-intensity contractions, thereby enabling higher training volume or quality and facilitating downstream anabolic remodeling responses. In contrast, the bone literature is less uniform: pooled effects on areal BMD are typically null even with prolonged supplementation, yet selected trials suggest preservation or improvement of hip structural geometry parameters that may contribute to mechanical strength independently of DXA-derived BMD. This divergence implies that creatine's skeletal effects may be modest, indirect (via muscle-mediated loading and falls prevention), or better captured by structural/strength surrogates than by areal BMD alone. Key limitations of the evidence base include heterogeneity in dosing regimens (loading vs no loading, maintenance dose), training protocols, baseline creatine status (dietary pattern, muscle mass), and participant characteristics (generally healthy older adults vs those meeting formal sarcopenia criteria), as well as inconsistent assessment of bone turnover biomarkers and fracture-relevant outcomes. Safety considerations remain important in geriatric practice: creatine can increase serum creatinine and artifactually lower creatinine-based eGFR without implying renal injury, emphasizing the need for careful interpretation of kidney function markers in older adults and in those with comorbidity.

5. Conclusions

Creatine supplementation, particularly when combined with progressive resistance training, is supported by the best available evidence as an effective adjunct to improve lean mass, muscular strength, and physical function in older adults—outcomes that are directly relevant to the prevention and management of sarcopenia and functional decline. In contrast, current data do not demonstrate a consistent benefit for increasing areal bone mineral density; nevertheless, preliminary signals for favorable effects on hip structural geometry and the strong muscle-mediated pathway to falls reduction justify further investigation of creatine within integrated osteosarcopenia prevention strategies. Future trials should prioritize well-characterized older populations (including those with confirmed sarcopenia and/or osteopenia), standardized functional endpoints (e.g., chair-rise, gait speed), contemporary imaging beyond DXA where feasible (e.g., structural geometry or pQCT), and clinically relevant outcomes such as falls and fractures, while employing dosing regimens that are practical and generalizable across real-world settings.

Disclosure

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Declaration of the Use of Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work, the author(s) used ChatGPT 5.2 for the purpose of stylistic review. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the substantive content of the publication.

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