RESEARCH PAPER

Unlocking Relief: The Role of Magnesium in Musculoskeletal Health – A Narrative Review

Dr. Richa Mahajan[1], Sakshi Vats[2], Mansi Jain[3], Divya[4], Dr. Meena Makhija[5*]

[1] Associate Professor, Department of Physiotherapy, Amar Jyoti Institute of Physiotherapy, University of Delhi, India
[2] PhD Scholar, Department of Allied Health Sciences, Galgotias University, Uttar Pradesh, India
[3] PhD Scholar, Department of Allied Health Sciences, Galgotias University, Uttar Pradesh, India
[4] Demonstrator, Department of Physiotherapy, School of Healthcare and Allied Sciences, GD Goenka University, Gurugram, India
[5]* Associate Professor, Department of Physiotherapy, ISIC-Institute of Rehabilitation Sciences, Vasant Kunj, New Delhi, India

*Corresponding Author: Dr Meena Makhija (BPT, MPT, PhD), Associate Professor, ISIC- Institute of Rehabilitation Sciences, Vasant Kunj, New Delhi, India. Email- makhijameena81@gmail.com

ABSTRACT

Musculoskeletal disorders are among the most prevalent causes of chronic pain and disability in clinical practice. Despite a wide therapeutic options, their management remains challenging, and nutritional factors particularly magnesium adequacy are often overlooked. Magnesium participates in many enzymatic reactions, governs calcium flux in muscle and bone cells, modulates parathyroid hormone activity, and suppresses pro-inflammatory signalling. Suboptimal intake is widespread across all age groups and is compounded by poor dietary quality, ageing-related absorption decline, and drug-induced renal losses. This narrative review synthesised evidence from MEDLINE, PubMed, Scopus, and Google Scholar on the role of magnesium in bone mineralisation, skeletal muscle physiology, cartilage integrity, and specific musculoskeletal conditions including osteoporosis, osteoarthritis, non-specific low back pain, myofascial trigger points, and fibromyalgia. The available literature consistently links higher magnesium intake with reduced fracture risk, greater muscle mass and power, lower systemic inflammation, and better knee cartilage morphology. Supplementation confers particular benefit in postmenopausal women and older adults with functional decline, while deficiency accelerates bone resorption, heightens neuromuscular excitability, and amplifies joint inflammation. These findings support routine assessment of magnesium adequacy in patients presenting with bone loss, chronic muscle pain, or inflammatory arthritis, and argue for larger randomised trials to define optimal dosing and target populations.

Keywords: magnesium; bone mineral density; musculoskeletal pain; osteoarthritis; muscle contraction; inflammation

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1. INTRODUCTION

Musculoskeltal problems are very commonly seen in the outpatient departments. Back pain, osteoarthritis,rheumatoid arthritis, osteoporosis-related fractures, and chronic muscle pain together account for an enormous share of global disability. There is a multifactorial basis in which genetics, lifestyle, and nutrition all play overlapping roles.[1,2] Among the nutritional factors, calcium and vitamin D have received the greatest research attention. However, this emphasis has often meant less focus on magnesium, a mineral that works quietly and extensively in the background of musculoskeletal physiology.

Magnesium is the fourth most common mineral in the human body and the second most abundant positively charged particle inside cells. Roughly 60% of the body's magnesium resides in bone, around 20% in skeletal muscle, and the remainder in soft tissues and extracellular fluid.[3] These distribution figures are not incidental they hint at just how central this mineral is to skeletal and muscular integrity. At the biochemical level, magnesium serves as an obligate cofactor for over many enzymatic reactions, ranging from the hydrolysis of ATP to the synthesis of nucleic acids and structural proteins.[4] It also plays a pivotal role in regulating calcium flux across muscle cell membranes, in modulating parathyroid hormone activity, and in governing whether the immune system tilts towards or away from a pro-inflammatory state.

Dietary magnesium insufficiency is widespread across North America, Europe, and South Asia, and worsens with age as renal efficiency declines, gut absorption becomes less reliable, and polypharmacy particularly with diuretics and proton pump inhibitors increases urinary losses.[5]

The present review aims to examine what current research reveals about the role of magnesium in musculoskeletal health. It considers each major domain in turn: bone mineralisation and

remodelling, skeletal muscle physiology, joint cartilage integrity, and painful conditions such as low back pain, myofascial trigger points, and fibromyalgia. This review also addresses issues of bioavailability, the practical implications of deficiency.

2. LITERATURE SEARCH STRATEGY

This narrative review was carried out through searches of four databases — MEDLINE, PubMed, Scopus, and Google Scholar — using combinations of the following terms: 'magnesium', 'musculoskeletal disorders', 'bone mineral density', 'osteoporosis', 'osteoarthritis', 'low back pain', 'trigger points', 'fibromyalgia', 'rheumatoid arthritis', 'muscle contraction', and 'pain relief'. No hard date cut-off was applied; studies span from 1992 to 2024, though the search was weighted towards publications from the past fifteen years (2009–2024). Earlier works were included where they provided seminal mechanistic insights or historical context that more recent papers built upon.

Studies were considered eligible if they were published in English, enrolled adult subjects (18 years or older), and reported data on magnesium status whether measured through dietary intake assessment, serum or erythrocyte concentration, or supplementation protocols alongside at least one musculoskeletal outcome. Randomised controlled trials, prospective cohort studies, and cross-sectional investigations were all included. Animal studies are referenced in a limited number of instances where they illuminate biological mechanisms not yet fully characterised in human research. No formal meta-analytic pooling was undertaken; findings are synthesised narratively with an emphasis on clinical relevance and consistency of evidence across study types.

3. OVERVIEW OF KEY STUDIES

Table 1. Selected studies examining magnesium intake or supplementation in relation to musculoskeletal outcomes.

Author	Objective	N	Design	Key Findings	Conclusion
Veronese et al. [6]	Examine how dietary magnesium relates to fracture incidence	3,765 adults (1,577 men; 2,071 women)	Prospective cohort	Top magnesium quintile: HR 0.47 (95% CI 0.21–1.00) in men; HR 0.38 (95% CI 0.17–0.82) in women; women meeting recommended intake had 27% fewer future fractures	Greater magnesium intake is protective against osteoporotic fracture, especially in women
Brilla & Haley [7]	Test whether supplemental magnesium augments strength-training gains	26	RCT	Magnesium group achieved significantly higher absolute and relative quadriceps torque than controls after 7-week training programme	Magnesium may enhance muscle adaptation, possibly through improved ribosomal protein synthesis
Welch et al. [8]	Assess associations between dietary magnesium and muscle mass, power, and CRP in women	2,570 women	Cross-sectional	Highest vs. lowest magnesium quintile: 2.6% more fat-free mass, 19.6 W/kg greater explosive power; associations 7× and 2.5× larger than those for protein; higher magnesium attenuated CRP–	Adequate magnesium may mitigate age-related muscle loss and support explosive performance across the female lifespan

Author	Objective	N	Design	Key Findings	Conclusion
				muscle mass inverse relationship	
Aydin et al. [9]	Evaluate biochemical bone turnover markers after 30-day oral magnesium in postmenopausal osteoporotic women	20 postmenopausal women	RCT	Significant fall in serum iPTH and urinary deoxypyridinoline; significant rise in serum osteocalcin in supplemented group	Oral magnesium shifts bone metabolism toward formation and away from resorption in postmenopausal women
Arias-Fernández et al. [10]	Prospectively track change in magnesium intake and physical performance over 5 years in older adults	863 (439 women; 424 men)	Prospective cohort	Higher intake in older women associated with better SPPB score (β 1.01; 95% CI 0.49–1.52; p-trend=0.001); no significant finding in men	Meeting magnesium dietary targets is linked to preserved functional capacity in older women
Veronese et al. [11]	Determine whether dietary magnesium correlates with MRI-derived knee cartilage volume and thickness	783	Cross-sectional	Each 100 mg/day increment in magnesium associated with significantly greater medial tibial and central medial femoral cartilage thickness and volume after confounder adjustment	Higher magnesium intake may preserve knee cartilage architecture, pointing to a chondroprotective role

4. HOW MAGNESIUM ACTS IN MUSCULOSKELETAL TISSUES

4.1 Bone Formation and Remodelling

Bone is not the static, inert scaffold it might appear but it is a metabolically dynamic tissue that spends an adult lifetime in a continuous cycle of breakdown and rebuilding, by osteoclasts and osteoblasts respectively. Magnesium is woven into this process at several levels simultaneously, which is part of what makes its deficiency so consequential for skeletal health.

At the structural level, magnesium ions are incorporated directly into the hydroxyapatite mineral lattice that gives bone its hardness. The size and solubility of hydroxyapatite crystals and therefore the mechanical properties of bone depend partly on magnesium content. When magnesium is low, crystals become larger and more brittle, a change that is associated with increased fracture susceptibility independent of bone mineral density.[4] This is a subtle but important point: two patients may have identical DEXA scores and yet very different fracture risk depending on crystal quality, and magnesium plays a role in determining that quality.

Parathyroid hormone (PTH) secretion and peripheral responsiveness to PTH are both impaired under conditions of hypomagnesaemia. PTH is a critical driver of renal calcium reabsorption and bone remodelling balance, so its dysregulation shifts the system towards net bone loss.[5]

At the cellular level, magnesium acts as a cofactor for alkaline phosphatase, the enzyme responsible for hydrolyzing pyrophosphate, thus permitting calcium phosphate deposition to proceed normally.[12] It also regulates expression of key matrix proteins, including osteocalcin and type I collagen, that provide the organic scaffold into which mineral is deposited.

4.2 Skeletal Muscle: Contraction, Relaxation, and Energy

Muscle contraction is triggered by calcium release from the sarcoplasmic reticulum, which binds to troponin C on the thin filament and initiates cross-bridge cycling between actin and myosin. Magnesium acts as a natural physiological brake on this process: at rest, intracellular magnesium concentrations exceed those of calcium, allowing magnesium to occupy the shared calcium/magnesium binding site on troponin C and thereby stabilise the relaxed state of the muscle fibre. When membrane depolarisation triggers rapid calcium release from the sarcoplasmic reticulum, calcium displaces magnesium at these troponin C sites, initiating cross-bridge cycling.[13] When intracellular magnesium is adequate, this competition keeps muscle excitability within normal bounds. When it falls short, the balance shifts in calcium's favour, and muscle fibres become prone to sustained or repetitive firing which correlate of cramps, spasms, and twitches.[14]

Beyond contractility, magnesium is essential for the ATP-dependent processes that underpin virtually every aspect of muscle cell maintenance. The sodium-potassium ATPase pump, which restores resting membrane potential after each action potential, requires Mg-ATP as its substrate. The calcium ATPase of the sarcoplasmic reticulum membrane, which actively sequesters calcium back into the reticulum after contraction to facilitate relaxation.[15] This combination accelerates fatigue, prolongs the calcium transient, and predisposes to prolonged muscle activation.

4.3 Inflammation and the Immune Environment of Joints and Muscles

Chronic inflammation is a shared final pathway across musculoskeletal conditions, from synovial inflammation in rheumatoid arthritis to low-grade mediator release in OA cartilage and central sensitisation in fibromyalgia. Magnesium moderates this inflammatory environment through several interconnected mechanisms.

At the molecular level, magnesium suppresses NF-κB, the master transcription factor driving expression of IL-6, TNF-α, and IL-1β.[16] These cytokines recruit immune cells into joint tissue, stimulate osteoclast activity, and sensitise nociceptors. Magnesium also enhances superoxide dismutase and glutathione peroxidase activity,[17] providing antioxidant support that is particularly relevant in OA and postmenopausal bone loss where oxidative burden is elevated. A meta-analysis by Veronese and colleagues confirmed that magnesium supplementation significantly lowers CRP in individuals with high baseline levels.[18]

5. CLINICAL CONSEQUENCES OF MAGNESIUM DEFICIENCY

Magnesium deficiency is often hard to detect. Symptoms are vague—fatigue, muscle tightness, sleep disturbance, and poor exercise recovery—so clinicians rarely test for it. Serum magnesium can be normal despite deficiency because it represents <1% of total body stores and is maintained at the expense of intracellular and bone reserves..[18]

Within bone, sustained inadequacy of magnesium tilts the remodelling balance towards resorption. Osteoclast activity remains relatively unchecked, while osteoblast function is impaired. The combined effect is progressive loss of trabecular and cortical bone, a process that over time markedly increases fracture risk.This consequence is particularly pronounced in postmenopausal women, who are already navigating accelerated bone turnover secondary to oestrogen withdrawal.[6,19] The compounding of hormonal and nutritional risk factors in this group is clinically underappreciated.There is an association between serum magnesium and osteoporosis and nocturnal cramps.[19,20]

At the level of joint inflammation, hypomagnesaemia creates a pro-inflammatory milieu. Elevated circulating levels of IL-6, TNF-α, and CRP have been documented in magnesium-deficient individuals, and this heightened inflammatory state accelerates the cartilage matrix degradation that characterises OA progression and the synovial pannus formation seen in rheumatoid arthritis.[17,21]

6. CLINICAL EVIDENCE BY CONDITION

6.1 Osteoporosis and Fracture Prevention

Perhaps the most striking evidence for magnesium in musculoskeletal medicine comes from fracture data. In a large prospective cohort, Veronese and colleagues followed 3,765 older adults living in the community. They found that those with the highest dietary magnesium intake had much lower fracture risk: hazard ratios of 0.47 (95% CI 0.21–1.00) in men and 0.38 (95% CI 0.17–0.82) in women, compared with those consuming the least. Put simply, women who consistently met their recommended magnesium intake were 27% less likely to experience a new fracture during follow-up. These are effect sizes that, if reproducible in randomised trials, would merit serious consideration of magnesium adequacy as a standard component of osteoporosis prevention programmes.[6,18]

In a separate randomised trial enrolling healthy periadolescent girls, Carpenter and colleagues demonstrated that magnesium oxide supplementation produced meaningful gains in integrated hip bone mineral content over the supplementation period, with good tolerability.[22]

6.2 Skeletal Muscle Massand Functional Capacity

Sarcopenia is the age-related decline in skeletal muscle mass and strength. It is among the most debilitating yet underdiagnosed conditions in older adults. Given magnesium's essential roles in muscle protein synthesis and contractility, it is unsurprising

that dietary magnesium has emerged as a predictor of muscle outcomes, though the strength of these associations is particularly noteworthy.

In a cross-sectional study of 2,570 women spanning a wide age range, Welch and colleagues found that those in the highest quintile of dietary magnesium intake had 2.6% greater fat-free mass, 0.4 kg/m² greater fat-free mass index, and 19.6 W/kg higher explosive leg power than those in the lowest quintile.[8] These associations were sevenfold and 2.5-fold greater, respectively, than those observed for protein. This finding challenges the conventional view of sarcopenia as primarily a problem of protein adequacy.The investigators also found that higher magnesium intake attenuated the inverse relationship between circulating high-sensitivity CRP and skeletal muscle mass, suggesting that part of magnesium's benefit on muscle operates through suppression of the inflammatory milieu that accelerates muscle wasting.[23]

Looking at functional endpoints rather than body composition measures, Arias-Fernández and colleagues followed 863 older Spanish adults prospectively over five years and found that women who increased their magnesium intake during follow-up had significantly better Short Physical Performance Battery (SPPB) scores — a composite measure of balance, gait speed, and chair-rise ability — than those whose intake stagnated or fell (β 1.01; 95% CI 0.49–1.52; p = 0.001).[10] No significant benefit was detected in older men, a sex difference the authors attributed to possible differences in baseline intake levels, magnesium metabolism, or hormonal context.

6.3 Osteoarthritis

Osteoarthritis is the commonest arthritis and a major cause of disability; its complex, multifactorial pathophysiology is managed mainly symptomatically. Emerging, still-preliminary evidence that magnesium may help preserve cartilage integrity is therefore of clinical interest.

Veronese and colleagues published a cross-sectional MRI study in which they quantified knee cartilage morphology with precision imaging in 783 participants.[11] They found that every 100 mg/day increment in habitual dietary magnesium was associated with significantly greater cartilage thickness and volume at the medial tibia and central medial femur — the anatomical regions where OA characteristically begins. These associations held after adjusting for body mass index, physical activity, vitamin D, and other potential confounders, suggesting a direct relationship rather than a proxy effect mediated by healthier overall diet.

The mechanistic underpinnings of this chondroprotective relationship are several. Chondrocytes are sensitive to both oxidative stress and inflammatory cytokine exposure, both of which magnesium helps to moderate.[24] Magnesium also regulates expression of matrix metalloproteinases, the enzymes that break down cartilage collagen, and of their inhibitors, suggesting that adequate magnesium may help maintain the anabolic-catabolic balance within cartilage that is disrupted in established OA.[25, 26] Subchondral bone is recognised as an active participant in OA progression rather than a passive bystander and magnesium's bone-protective effects may provide an additional layer of structural support for the overlying cartilage.

6.4 Non-Specific Low Back Pain

Non-specific low back pain is common and often driven by paraspinal muscle spasm; magnesium stabilises muscle membranes and antagonises NMDA-mediated spinal pain amplification, so it may help reduce spasm-related pain.A prospective randomised clinical trial by Bayram and colleagues directly addressed this question in patients presenting acutely with non-specific low back pain and found that adding oral magnesium to standard non-steroidal anti-inflammatory therapy did not significantly improve pain or functional outcomes compared with standard therapy alone.[27]

6.5 Myofascial Trigger Points

Myofascial trigger points are hyperirritable spots within taut muscle bands that produce predictable referred pain on palpation; they are common, under-researched, central to myofascial pain syndrome, and account for much regional or widespread chronic musculoskeletal pain.The pathophysiology of trigger points converges on a sustained energy crisis at the motor end-plate. The excessive acetylcholine release leads to persistent sarcomere shortening, local ischaemia, and the accumulation of nociceptive metabolites. Magnesium fits this picture in several ways: it normally modulates presynaptic acetylcholine release, competes with calcium at the actin-myosin interface to promote relaxation, and provides the ATP needed by the SERCA pump to restore calcium to the sarcoplasmic reticulum.[28] A muscle that is magnesium-deplete may therefore be predisposed to trigger point formation and maintenance, and one that is repleted may recover more readily.

Clinical intervention studies, though modest in number, support this reasoning. Ibrahim and colleagues demonstrated that iontophoresis of magnesium sulfate which is a non-invasive method of driving the mineral through the skin using a small electrical current .It reduced trapezius trigger point pain and pressure sensitivity significantly compared with direct current control (without active medication).[28] Refahee and colleagues took a more invasive approach and injected magnesium sulfate directly into masseter muscle trigger points in patients with temporomandibular myofascial pain, finding pain reduction and improved mouth opening.[29] Together these studies suggest that the route of magnesium delivery matters less than the act of delivering it to deficient muscle tissue.

6.6 Fibromyalgia and Chronic Widespread Pain

Fibromyalgia sits at a complicated intersection of peripheral muscle physiology, central pain processing, neuroendocrine dysregulation, and psychological factors. Reduced erythrocyte and intracellular magnesium concentrations have been documented in fibromyalgia patients in multiple studies, though whether this represents a cause, a consequence, or a bystander effect of the underlying pathophysiology is not yet fully settled.[30]

A literature review by Boulis and colleagues found that oral magnesium supplementation, most commonly administered as magnesium malate which is well tolerated and efficiently absorbed, was associated with modest but measurable reductions in tender point counts and pain severity among fibromyalgia patients across the trials they reviewed.[30]

7. DIETARY SOURCES AND BIOAVAILABILITY

Magnesium is plentiful in nuts, seeds, leafy greens, legumes, wholegrains, dark chocolate and fatty fish, yet habitual intakes often fall short because of processed diets and declining soil mineral content.Intestinal absorption of magnesium from food ranges from roughly 20% to 50% depending on several factors, including the form in which magnesium is present, the overall composition of the meal, and the individual's current magnesium status with fractional absorption increasing when stores are depleted and decreasing when they are replete.[3]Phytic acid and fibre in magnesium-rich foods can bind magnesium and reduce its absorption; high calcium intakes also compete at intestinal transporters, lowering net magnesium uptake.

Among supplemental forms, organic magnesium salts namely citrate, malate, glycinate, and taurate consistently demonstrate superior absorption compared with inorganic forms such as oxide or carbonate.[12]Magnesium oxide is poorly soluble at physiological pH and is less well absorbed despite high elemental content; magnesium citrate is water-soluble and produces larger, more consistent serum rises per gram, so formulation matters for therapeutic supplementation, especially in deficiency or GI compromise.

Several clinical scenarios common in musculoskeletal practice are also associated with heightened magnesium losses or impaired retention: type 2 diabetes, chronic kidney disease, proton pump inhibitor use, and loop or thiazide diuretic therapy.[18] Patients carrying these risk factors deserve particular attention to their magnesium status, not least because they are often the same patients presenting with bone loss, muscle weakness, and chronic pain.

8. DISCUSSION

The evidence synthesised here tells a coherent story. Magnesium is not merely a biochemical cofactor but is woven into musculoskeletal physiology at multiple levels, and its deficiency propagates consequences that are clinically visible: fractures, muscle dysfunction, joint deterioration, and chronic pain. These consequences accrue slowly, making them easy to attribute to ageing rather than to a correctable nutritional gap.

The fracture data are the most striking. A 62% reduction in fracture hazard among women with the highest dietary magnesium intake[6] exceeds the effect size of many pharmacological interventions. Although confounding cannot be dismissed, the finding is biologically plausible, consistent across populations, and supported by mechanistic and interventional evidence, warranting a properly powered randomised fracture-prevention trial.

The muscle data are equally compelling. Magnesium's associations with muscle mass and explosive power substantially outpace those for protein intake,[8] with direct implications for sarcopenia management. Overlooking magnesium while emphasising protein and exercise may partly explain the modest functional outcomes seen in nutrition-and-exercise programmes for older adults.

Magnesium's anti-inflammatory actions provide a unifying mechanism across OA, rheumatoid arthritis, fibromyalgia, and low back pain. Suppression of NF-κB signalling, reduced circulating CRP, and enhanced antioxidant enzyme activity[31] collectively dampen the inflammatory cascade driving structural damage and pain sensitisation.

The literature remains heterogeneous and most interventional trials are small, short, and lacking hard clinical endpoints, so definitive dosing recommendations await larger trials. On safety, oral magnesium is well tolerated; osmotic diarrhoea is manageable, and significant hypermagnesaemia is rare in normal renal function, though caution is needed in chronic kidney disease.[32]

9. CONCLUSION

This review makes a case for reconsidering the place of magnesium in musculoskeletal medicine. From bone mineralisation to muscle contraction, from arthritic joint inflammation to pain modulation in fibromyalgia, magnesium operates across all these domains and its deficiency leaves traces in each.

For the practising clinician, the message is straightforward: magnesium adequacy should be assessed in any patient presenting with bone loss, chronic muscle pain, or inflammatory joint disease. The first intervention should be dietary, prioritising nuts, leafy vegetables, legumes, and wholegrains. Where diet proves insufficient, supplementation with magnesium citrate or malate is a safe and inexpensive option provided renal function is adequate.

Larger trials with hard clinical endpoints including fracture incidence, bone mineral density, handgrip

strength, and validated functional scores are still needed before definitive recommendations can be made.

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