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Review Article

Dietary magnesium in health: Mechanisms, evidence, and clinical implications

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Abstract

Magnesium is an essential mineral cofactor in over 300 enzymatic processes, integral to cellular energy metabolism, nucleic acid synthesis, ion transport, neuromuscular function, vascular regulation, and bone homeostasis. Despite its importance, suboptimal dietary magnesium intake is widespread and has been associated with increased risks of metabolic syndrome, hypertension, cardiovascular disease, type 2 diabetes, osteoporosis, migraine, and inflammation. In interventional trials, magnesium supplementation has shown robust benefit for migraine prophylaxis and reduction in hospitalization during pregnancy, with moderately convincing evidence for improvements in glycemic control, inflammation, and vascular function. This article comprehensively reviews magnesium absorption and homeostasis, dietary sources and intake recommendations, the epidemiologic and clinical trial evidence linking magnesium with health outcomes, clinical manifestation of deficiency, diagnostic challenges, and therapeutic guidance. Gaps in knowledge and directions for future research are highlighted.

Keywords: Magnesium, Micronutrient, Diet, Deficiency, Cardiovascular disease, Metabolic syndrome, Osteoporosis, Neurological disease

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1. Introduction

Magnesium (Mg^{2+}) is the second most abundant intracellular cation after potassium and the fourth most abundant mineral in the body. Its ubiquitous involvement in physiological processes underscores its fundamental role in health.[1] Yet multiple surveys indicate that a substantial proportion of populations in Europe, North America, and Asia fail to meet recommended magnesium intakes, rendering magnesium insufficiency an underrecognized contributor to chronic disease.[2]

This review aims to synthesize current knowledge on the role of dietary magnesium in health, bridging mechanistic insights and clinical evidence, and to offer pragmatic perspectives for clinicians and researchers.

2. Physiology of Magnesium: Absorption, Distribution, and Homeostasis

2.1. Intestinal absorption and transport

Dietary magnesium is absorbed predominantly in the small intestine via two complementary pathways: a passive paracellular route across tight junctions, and an active transcellular route mediated by ion channels. The paracellular pathway is concentration-driven and non-saturable; the transcellular route involves the epithelial channels TRPM6 and TRPM7, as well as other transporters (e.g. SLC41A1).[3] In the colonic epithelium, TRPM6 expression also contributes to magnesium uptake under low-intake conditions.[4]

EGF signaling has been shown to regulate TRPM6 trafficking and function in intestinal cells, revealing cross-talk between growth factors and magnesium uptake.[3]

The fractional absorption of magnesium ranges from ~30% to 60%, depending on dietary load, presence of competing ions (e.g. calcium, phosphate), and intestinal

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health.[5] In conditions of low magnesium intake, the body adapts by increasing the efficiency of absorption.

3. Renal Handling and Excretion

Renal excretion is the key regulator of systemic magnesium balance. After glomerular filtration of magnesium, ~95% is reabsorbed along the nephron, predominantly in the thick ascending limb (TAL) (~60–70%) via passive paracellular mechanisms driven by the lumen-positive transepithelial potential (with claudin-16 and claudin-19 facilitating cation selectivity).[6] Additional reabsorption occurs in the proximal tubule and distal convoluted tubule (DCT). In the DCT, active transcellular reabsorption is mediated by TRPM6 located on the apical membrane; the basolateral extrusion mechanism is not yet fully characterized.[6]

Genetic or acquired dysregulation of TRPM6 is implicated in disorders of magnesium balance (Mg^{2+} homeostasis: the balancing act of TRPM6)[4] (TRPM6 forms the Mg^{2+} influx channel).[3] In murine models, intestinal and renal deletion of TRPM6 leads to profound hypomagnesemia with severe developmental consequences (Epithelial magnesium transport by TRPM6).[7]

Regulatory hormones (e.g. insulin, parathyroid hormone, aldosterone) and dietary factors modulate TRPM6 expression and function.[8]

4. Distribution and Intracellular Functions

Approximately 99% of total body magnesium resides intracellularly: ~50–60% in bone matrix, and the remainder in soft tissues such as muscle and other organs; <1% is in serum and extracellular fluid.[2] Because serum magnesium represents a small fraction of total stores and is tightly regulated, it is a relatively insensitive marker of magnesium sufficiency.

Within cells, magnesium serves numerous roles:

1. ATP binding and energy metabolism: Mg^{2+} stabilizes ATP (as Mg–ATP), the biologically active form in enzymatic reactions.
2. Nucleic acid and protein synthesis: Acts as a cofactor for polymerases and ribosomal functions.
3. Ion channel modulation and signaling: Regulates calcium, potassium, and sodium channels; acts as a natural calcium antagonist in vascular smooth muscle.
4. Signal transduction: Involved in second messenger systems (e.g. cAMP).
5. Mitochondrial function, oxidative stress, and apoptosis regulation.
6. Bone health: Influences osteoblast/osteoclast balance, parathyroid hormone (PTH) secretion, and active vitamin D metabolism.

These varied functions create a scenario in which mild magnesium insufficiency may subtly impair multiple physiological systems before overt deficiency becomes clinically manifest.

5. Dietary Sources, Recommended Intake, and Factors Affecting Bioavailability

5.1. Dietary Sources

Foods rich in magnesium include:

1. Nuts and seeds (almonds, cashews, pumpkin seeds, chia seeds)
2. Whole grains (brown rice, oats, whole wheat)
3. Legumes (beans, lentils)
4. Green leafy vegetables (spinach, Swiss chard)
5. Tofu, soy products
6. Seafood (e.g. salmon, mackerel)
7. Dark chocolate

Table 1: Below is a representative table of magnesium content in selected foods:

Table 1: Approximate magnesium content of selected foods

Food Item	Approximate Mg (mg per 100 g)	Notes / Comments
Pumpkin seeds / chia seeds	400–560	Among highest in common foods
Almonds (dry roasted)	~280	High density source
Whole grain cereals	~200–240	Varies with grain and processing
Dark chocolate (70%)	~230	Also contains flavonoids
Peanuts (oil roasted)	~100–110	Moderate source
Cooked spinach	~60–70	Also rich in other micronutrients
Tofu	~50–60	Plant protein source
Salmon	~30–40	Lower but contributes alongside protein
Banana	~25–30	Fruit source

Magnesium in drinking water (especially “hard water”) may contribute measurably in some regions.

6. Recommended Intake and Observed Gaps

Various national bodies recommend magnesium intakes in the range of ~310–420 mg/day for adults, depending on age and sex, with higher needs in pregnancy and lactation. However, dietary surveys frequently report mean intakes below these levels in many populations.[2]

A large dose–response meta-analysis of prospective cohort studies (over 1 million participants) found that each incremental 100 mg/day increase in dietary magnesium was associated with a 7% lower stroke risk, 22% lower heart

failure risk, 19% lower incidence of type 2 diabetes, and 10% reduction in all-cause mortality.[9]

Several dietary and physiologic factors can adversely affect magnesium absorption or increase loss:

1. High dietary calcium, phosphorus, or zinc
2. Phytate, oxalate, and excessive dietary fiber
3. High doses of other minerals
4. Drugs: diuretics, proton-pump inhibitors, aminoglycosides, cisplatin
5. Gastrointestinal disorders: malabsorption, inflammatory bowel disease, resection
6. Chronic diarrhea or vomiting
7. Alcoholism and renal magnesium wasting[10]

Additionally, magnesium bioavailability is influenced by the chemical form (organic vs. inorganic salt), the dose (smaller fractionated doses absorb better), and the food matrix (e.g. presence of enhancing fibers, medium-chain triglycerides).[5]

7. Evidence Linking Dietary Magnesium and Health Outcomes

The health effects of magnesium are evaluated through epidemiologic observational studies, clinical trials, and meta-analyses.

8. Umbrella Review of Magnesium and Health Outcomes

An umbrella review integrating multiple meta-analyses of observational and RCT data evaluated 55 independent health outcomes across 16 meta-analyses.[11] Among RCTs, strong evidence was found for reduction in hospitalization in pregnancy and decreased frequency/intensity of migraine; in observational studies, highly suggestive evidence supported a lower incidence of type 2 diabetes, and suggestive evidence for reduced stroke risk.

This review underscores that while magnesium is broadly implicated in health, the strongest RCT-level evidence is concentrated in a few domains (migraine, pregnancy).[11]

9. Cardio Metabolic and Cardiovascular Health

A detailed review (Role of dietary magnesium in cardiovascular disease prevention) concluded that higher magnesium intake is inversely associated with metabolic syndrome, hypertension, type 2 diabetes, and cardiovascular disease (CVD). Mechanistic pathways include modulation of insulin signaling, improved lipid profiles, reduced inflammation, suppression of oxidative stress, and vascular smooth muscle relaxation (via calcium antagonism).[12]

In controlled trials, long-term magnesium supplementation has been shown to reduce arterial stiffness—a surrogate CVD risk marker—in overweight and obese adults (350 mg/d for 24 weeks).[13] However, improvements in direct endothelial function indices (e.g. flow-mediated dilation, FMD) have been inconsistent: a meta-analysis of RCTs found no significant overall effect of oral magnesium on FMD or pulse wave velocity (PWV), though subgroup analyses showed benefit in longer-duration trials (>6 months), in older or overweight individuals.[14]

The more recent review affirmed that lower magnesium status is associated with hypertension, coronary calcification, stroke, atrial fibrillation, heart failure, and increased cardiac mortality.[8] Controlled metabolic unit studies indicate that even modest magnesium deficiency evokes reversible metabolic perturbations supportive of a contributory role in CVD pathogenesis.

Meta-analyses focusing on blood pressure suggest that magnesium supplementation yields modest reductions in systolic and diastolic blood pressures, particularly at doses ≥300 mg/day and treatment durations ≥12 weeks (e.g. in T2DM patients).[15]

10. Glucose Metabolism, Insulin Resistance, and Type 2 Diabetes

Magnesium plays a direct role in insulin receptor phosphorylation and post-receptor signaling, and magnesium deficiency is hypothesized to induce insulin resistance. Observational studies consistently show inverse associations between magnesium intake and incident type 2 diabetes; in the umbrella review, this was among the strongest observational associations.[11] The dose–response meta-analysis mentioned earlier observed that each 100 mg/day increment in dietary Mg was associated with a 19% lower incidence of diabetes.[9]

In a dedicated meta-analysis of RCTs in T2DM patients (16 trials), magnesium supplementation (versus control) significantly raised serum magnesium (mean difference 0.15 mg/dL; 95% CI 0.06–0.23) and increased urinary magnesium excretion (WMD 1.99 mg/dL).[10] Some trials also report improvements in HbA1c and insulin resistance metrics, though heterogeneity limits definitive conclusions.

11. Inflammation, Immune Function, and Other Diseases

Magnesium's anti-inflammatory and immunomodulatory roles are increasingly studied. A meta-analysis of 17 RCTs (889 participants) demonstrated that magnesium supplementation significantly reduced serum C-reactive protein (CRP) and increased nitric oxide (NO) levels; some trials also reported reductions in fibrinogen, IL-1, and TNF-family proteins.[16] These findings provide mechanistic plausibility for broader health effects.

Animal and in-vitro models of magnesium deficiency exhibit systemic "neurogenic inflammation," mediated via substance-P release and activation of prooxidant pathways that impact cardiovascular and intestinal tissues.[17]

Magnesium has been studied in infection and immune contexts: a randomized trial in COVID-19 patients (300 mg magnesium daily) reported fewer patients requiring oxygen therapy and improved oxygen saturation (though no significant changes in hs-CRP or TNF-α).[18]

Other conditions explore magnesium's role:

12. Bone Health and Osteoporosis

Magnesium exerts multiple effects on bone: it influences PTH secretion, vitamin D activation, osteoblast/osteoclast balance, and the bone matrix microenvironment. Chronic magnesium deficiency in animal models leads to bone loss, reduced bone formation, and structural defects.[19]

Epidemiologic studies support associations between higher magnesium intake and higher bone mineral density, particularly in older adults and postmenopausal women.[2] Interventional RCT evidence is less robust, and confounding by calcium and vitamin D complicates interpretation.

13. Neurological Health: Migraine, Cognitive Decline, and Neuromuscular Function

Magnesium influences neuronal excitability, synaptic plasticity, and modulates NMDA receptor activity. The umbrella review found strong evidence supporting magnesium supplementation in reducing frequency and intensity of migraine attacks. Clinical trials of magnesium (e.g. oral or intravenous) for migraine prophylaxis show modest benefit over placebo.

Emerging observational data suggest higher dietary magnesium correlates with larger brain volumes and lower white matter lesions, particularly in aging adults, hinting at neuroprotective effects.[20] However, causality remains unproven.

In neuromuscular physiology, magnesium deficiency classically presents with tetany, muscle cramps, tremors, and hyperexcitability.[21]

14. Clinical Manifestations, Diagnostics, and Management

14.1. Clinical manifestations of magnesium deficiency

Hypomagnesemia may present acutely or chronically.[22]

1. **Acute/severe deficiency** (serum Mg < 1.5 mEq/L): neuromuscular hyperexcitability (tetany, tremors, muscle cramps, fasciculations), seizures, arrhythmias (e.g. torsades de pointes), hypokalemia and hypocalcemia resistant to correction, prolongation of QT interval.[22]

2. **Chronic or subclinical deficiency**: may contribute to hypertension, insulin resistance, vascular dysfunction, metabolic syndrome, osteoporosis, migraine, mood disturbances, and increased oxidative stress.[22]

Hypomagnesemia remains underdiagnosed in clinical settings. In hospitalized populations, ~10% of patients are hypomagnesemic on admission.[22]

It's emphasized that magnesium depletion often goes unrecognized because clinicians focus on sodium, potassium, and calcium; newer measurement techniques may help improve recognition.[23]

15. Diagnostics and Biomarkers of Magnesium Status

Because serum (total) magnesium constitutes only ~1% of the body's stores and is tightly regulated, a "normal" serum magnesium does not preclude intracellular deficiency.[2]

Diagnostic approaches include:

1. **Serum total Mg** – widely available but insensitive for mild deficiency.
2. **Ionized (free) magnesium** – more physiologically relevant but less commonly available.
3. **Intracellular magnesium assays**: erythrocyte magnesium, lymphocyte magnesium, or mononuclear cell magnesium.
4. **Magnesium loading (retention) test**: administer a known magnesium load and measure urinary excretion; retention suggests deficiency.

Currently, no consensus "gold standard" exists, and many clinicians rely on risk factor assessment, symptoms, and incremental supplementation[14]

16. Management Principles and Supplementation

16.1. Dietary optimization

First-line strategy is to optimize dietary magnesium intake via whole-food sources. Encouraging consumption of magnesium-rich foods and reducing factors impairing absorption (e.g. phytate, excessive competing minerals) is foundational.

17. Oral Magnesium Supplementation

When dietary measures are insufficient or in patients with symptoms or risk factors, oral magnesium supplementation may be considered. Key considerations:

1. Formulations: organic salts (citrate, malate, glycinate) may offer better bioavailability and tolerability than inorganic forms (oxide) in some studies, though evidence is mixed.
2. Dosing: lower divided doses (e.g. 100–200 mg multiple times daily) are absorbed more efficiently than single large bolus.
3. Duration: benefits on vascular and metabolic endpoints often require prolonged supplementation (≥6 months)[14]
4. Monitoring: periodically monitor serum Mg, renal function, and relevant clinical markers (e.g. glycemic indices, BP).

5. Safety: in patients with normal renal function, moderate supplementation is generally safe. In renal insufficiency, risk of hypermagnesemia must be considered. Gastrointestinal side effects (e.g. diarrhea) are dose-limiting.

In prehypertensive individuals, a 4-month RCT using 360 mg magnesium lactate increased expression of TRPM6 and TRPM7 transcripts in leukocytes, suggesting molecular modulation (Effect on TRPM6/7 transcription).[24]

18. Parenteral Magnesium (Severe Deficiency)

In acute symptomatic hypomagnesemia (e.g. arrhythmias, seizures), intravenous magnesium is indicated, commonly at 24–48 mEq/day for several days, with electrolyte monitoring.[25]

Special Populations

1. **Pregnancy**: magnesium supplementation reduces the risk of hospitalization (e.g. preeclampsia, eclampsia), supported by RCT evidence.[11]
2. **Migraine**: strong RCT-level evidence supports magnesium for prophylaxis; clinicians may use oral (or IV in acute) magnesium.
3. **Chronic disease** (e.g. T2DM, CVD): supplementation may supplement other therapies but should not replace standard care.
4. **Renal disease**: dose adjustment or avoidance may be necessary in patients with reduced excretion capability.

19. Discussion

19.1. Summary and Future Directions

19.1.1. Strengths and limitations of existing evidence

The body of evidence linking dietary magnesium to health outcomes is biologically plausible, consistent across observational studies, and supported by modest RCT data in specific domains (e.g. migraine, pregnancy). Yet limitations remain: heterogeneity of trials, short durations, variable magnesium formulations, and reliance on serum magnesium as a surrogate.

The umbrella review provides a useful high-level overview, indicating that only a subset of magnesium associations enjoys strong RCT confirmation.[11] For most other outcomes, the evidence is suggestive or moderate.

Gaps and Future Research Directions

1. *Better biomarkers of total body magnesium status* — improved accessible intracellular measures or imaging modalities.
2. *Long-term randomized trials* in populations at risk (e.g. metabolic syndrome, elderly, low-intake regions), comparing doses/forms, and assessing hard endpoints (CVD events, fractures, diabetes onset, mortality).
3. *Mechanistic human studies* to delineate pathways (e.g. TRPM6 regulation, mitochondrial and autophagy effects, neuroimmune crosstalk).
4. *Precision nutrition approaches* — stratification by genetic polymorphisms in magnesium transporters (e.g. TRPM6), comorbidities, and interactions with other micronutrients.
5. *Public health strategies* — food fortification or targeted supplementation policies in at-risk populations.
6. *Integration into clinical guidelines* — standardized recommendations for screening, supplementation thresholds, and monitoring protocols.

20. Conclusion

Dietary magnesium is a foundational micronutrient with broad pleiotropic roles spanning energy metabolism, vascular regulation, neuromuscular excitability, and bone health. The prevalence of magnesium insufficiency in modern diets suggests that optimizing magnesium intake may represent a low-cost, low-risk adjunctive strategy in preventive and therapeutic medicine.

While RCT evidence is strongest in migraine prophylaxis and pregnancy outcomes, the totality of observational and mechanistic data generates compelling hypotheses that magnesium supplementation could favorably influence metabolic, cardiovascular, inflammatory, and bone health endpoints. Clinicians should maintain a high index of suspicion for magnesium deficiency in relevant clinical settings and consider supplementation when appropriate, especially in at-risk individuals. Future rigorous trials and improved diagnostic tools are needed to fully delineate magnesium's therapeutic potential.

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22. Conflict of Interest

None.

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