

Harnessing Magnesium: A Key to Cardiovascular and Metabolic Wellness

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ABSTRACT

Magnesium is an essential mineral involved in over 300 enzymatic reactions critical for maintaining cellular and systemic health. This review highlights its multifaceted roles, particularly in cardiovascular and metabolic health. Magnesium acts as a natural calcium antagonist, regulating vascular tone by inhibiting calcium influx into smooth muscle cells, promoting vasodilation, and reducing blood pressure. It supports endothelial function by enhancing nitric oxide bioavailability, reducing oxidative stress, and suppressing inflammation. Magnesium also modulates the renin-angiotensin-aldosterone system, mitigating hypertension by attenuating vasoconstriction and sodium retention. In glucose metabolism, magnesium improves insulin sensitivity and facilitates cellular glucose uptake, reducing the risk of type 2 diabetes and associated cardiovascular complications. Additionally, magnesium's antioxidative properties neutralize reactive oxygen species, curbing oxidative damage and chronic inflammation. Specific populations, such as the elderly and pregnant women, benefit significantly from magnesium supplementation to manage hypertension and preeclampsia. Moreover, magnesium's interaction with platelet-activating factors highlights its role in reducing thrombotic risk and systemic inflammation. Emerging evidence underscores magnesium's potential as a therapeutic strategy for hypertension, diabetes, and chronic inflammatory conditions. This review emphasizes the importance of maintaining optimal magnesium levels to enhance cardiovascular and metabolic health, advocating for its broader clinical application in disease prevention and management.

Keywords: Advanced CKD, Blood Pressure, Chronic kidney disease, Chlorthalidone, Hypertension

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INTRODUCTION

Magnesium, a vital divalent cation, is indispensable for numerous physiological processes, including energy metabolism, enzyme function, and cellular signaling. Among its many roles, magnesium's involvement in vascular health has garnered significant attention, particularly in the context of hypertension, a major global health challenge and leading risk factor for cardiovascular disease.¹

Magnesium deficiency is prevalent worldwide and has been linked to several cardiovascular pathologies, including hypertension, vascular calcification, and endothelial dysfunction.² Its role extends to modulating the renin-angiotensin-aldosterone system (RAAS), suppressing catecholamine-induced vasoconstriction, and preventing vascular calcification, all of which are crucial for maintaining optimal vascular tone.^{3,4} Furthermore, magnesium acts as a cofactor for

enzymes involved in nitric oxide synthesis, enhancing endothelial-dependent vasodilation.⁵ Emerging evidence highlights magnesium's influence on reducing oxidative stress, inflammation, and vascular aging, further emphasizing its protective effects on vascular health.⁶

Given the increasing prevalence of hypertension and its associated complications, understanding magnesium's multifaceted role in vascular regulation is essential. This review aims to explore the relationship between magnesium and hypertension, focusing on its mechanisms of action in regulating vascular tone, including its roles as a calcium antagonist, modulator of endothelial function, inhibitor of vascular calcification, and regulator of RAAS and catecholamine activity. Recent research findings are discussed to underscore the potential of magnesium as a therapeutic target in hypertension management and cardiovascular health.

PHYSIOLOGICAL ROLES OF MAGNESIUM

- 1. Energy Production and Metabolism:** Magnesium is a cofactor for adenosine triphosphate (ATP) synthesis in mitochondria, the energy powerhouse of cells. ATP is the primary energy currency

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of cells, and magnesium's presence is essential for its formation. Also, many enzymes involved in glycolysis, the Krebs cycle (citric acid cycle), and oxidative phosphorylation require magnesium for their activity. These processes are central to energy metabolism and cellular respiration.^{7,8}

2. **Muscle Function:** Magnesium plays a crucial role in muscle contraction by regulating the influx of calcium ions into muscle cells. It acts as a natural calcium channel blocker, preventing excessive calcium entry that can lead to prolonged muscle contraction and cramping. After contraction, magnesium facilitates muscle relaxation by promoting calcium efflux from muscle cells and by inhibiting myosin ATPase activity, essential for muscle relaxation.^{9,10}
3. **Neurological Function:** Magnesium is involved in regulating neurotransmitter release and neuromuscular transmission. It modulates the activity of N-methyl-D-aspartate (NMDA) receptors, which are critical for synaptic plasticity, learning, and memory. Adequate magnesium levels support cognitive function, mood stability, and overall brain health. Research suggests that magnesium deficiency may contribute to neurological disorders like migraines, depression, and anxiety.^{11,12}
4. **Bone Health:** It promotes calcium absorption in the gut and deposition into bones, contributing to bone density and strength. Magnesium is involved in bone mineralization processes, where it interacts with proteins like osteocalcin and influences the formation of hydroxyapatite crystals, the mineral component of bones.^{13,14}
5. **Cardiovascular Function:** Magnesium acts as a vasodilator by relaxing blood vessels and reducing peripheral resistance, which helps maintain normal blood pressure. It regulates the function of cardiac muscle cells and contributes to maintaining a regular heart rhythm (sinus rhythm).¹⁵
6. **Glucose and Insulin Metabolism:** Magnesium plays a role in insulin signaling pathways, influencing glucose uptake into cells and insulin sensitivity. Low magnesium levels have been linked to insulin resistance and impaired glucose tolerance, contributing to the development of type 2 diabetes.¹⁶
7. **Antioxidant Defense:** Magnesium acts as a cofactor for antioxidant enzymes like superoxide dismutase (SOD), glutathione peroxidase, and catalase which help neutralize free radicals and oxidative stress.^{17,18}

MAGNESIUM AND HYPERTENSION: DECODING MECHANISMS FOR INNOVATIVE THERAPEUTIC STRATEGIES

Magnesium as a Natural Calcium Blocker in Modulating Vascular Tone

Magnesium plays a pivotal role in maintaining vascular tone through its action as a natural calcium antagonist. It inhibits calcium influx into vascular smooth muscle cells by blocking voltage-gated calcium channels, preventing calcium-dependent smooth muscle contraction and promoting vasodilation. Consequently, peripheral vascular resistance decreases, contributing to lower blood pressure. Magnesium also enhances endothelial nitric oxide (NO) production,

a critical mediator of vasodilation. This dual action of magnesium—direct inhibition of calcium entry and indirect promotion of NO bioavailability—ensures optimal vascular function. Magnesium deficiency, conversely, leads to unopposed calcium-mediated vasoconstriction and endothelial dysfunction, resulting in elevated blood pressure and increased cardiovascular risk.¹⁹

Kasset *et al.* demonstrated improved arterial stiffness and enhanced vasodilation in hypertensive patients by magnesium supplementation. Dominguez *et al.* (2021) emphasized magnesium's critical role in mitigating hypertension, particularly in older adults, through its impact on vascular tone.²⁰

Magnesium as a Key Modulator of Endothelial Function

Magnesium supports endothelial health by enhancing nitric oxide (NO) production by endothelial cells, thereby reducing vascular resistance and promoting blood flow. Magnesium also suppresses endothelial oxidative stress by scavenging free radicals and modulating antioxidant enzyme activity. This protective effect preserves NO bioavailability, ensuring sustained vasodilation, reduces endothelial inflammation by inhibiting the expression of pro-inflammatory cytokines and adhesion molecules. Magnesium deficiency is associated with endothelial dysfunction, characterized by reduced NO production, heightened oxidative stress, and inflammation, all contributing to increased vascular tone and hypertension. Clinical studies have highlighted magnesium's ability to restore endothelial function. For example, Zeng *et al.* demonstrated that magnesium supplementation improves endothelial-dependent vasodilation in hypertensive individuals.^{20,21}

Magnesium and Its Influence on Renin-Angiotensin-Aldosterone System

Magnesium deficiency is associated with hyperactivation of the RAAS, leading to elevated levels of renin, angiotensin II (a potent vasoconstrictor), and aldosterone, increasing vascular resistance, sodium retention, which contributes to hypertension. Magnesium counteracts this effect by inhibiting calcium entry into these cells, thereby reducing angiotensin II-induced vasoconstriction and suppressing aldosterone secretion, which helps mitigate sodium retention and fluid overload, further reducing blood pressure.²²

Zheltova *et al.* (2016) demonstrated that magnesium deficiency exacerbates RAAS activation, while adequate magnesium levels attenuate its effects, contributing to improved blood pressure control and vascular function.²³

Magnesium and Its Role in Catecholamine-Mediated Vascular Tone Regulation

Magnesium functions as a natural antagonist to catecholamine-induced vascular effects: vasoconstriction, increased peripheral resistance. It inhibits the release of catecholamines from nerve terminals and adrenal chromaffin cells by blocking calcium-dependent exocytosis. Additionally, magnesium suppresses adrenergic receptor sensitivity on vascular smooth muscle cells, reducing the vasoconstrictive response to catecholamines. Magnesium deficiency is associated with heightened catecholamine activity, leading to increased vascular tone, oxidative stress, and endothelial dysfunction.²⁴

A clinical study by Sueta *et al.* showed that magnesium improved sympathetic regulation and reduced catecholamine-induced vascular reactivity in hypertensive patients. These findings highlight magnesium's protective role in mitigating catecholamine-mediated vasoconstriction, emphasizing its therapeutic potential in managing hypertension and vascular dysfunction.²⁵

Magnesium's Role in Vascular Calcification and Tone Regulation

Vascular calcification involves the deposition of calcium phosphate crystals within the vascular walls, leading to reduced arterial elasticity and increased peripheral resistance, which elevates blood pressure. Magnesium inhibits vascular calcification by several mechanisms. First, it directly binds phosphate, reducing the formation of calcium-phosphate complexes. Second, magnesium antagonizes the effects of calcium on vascular smooth muscle cells (VSMCs), preventing their transdifferentiation into osteoblast-like cells, a critical step in calcification. Third, magnesium promotes the expression of calcification inhibitors such as matrix Gla-protein (MGP), further safeguarding against arterial stiffening.²⁶

Clinical studies by Mazidi *et al.* (2018) demonstrated that higher magnesium intake correlates with reduced vascular calcification and improved arterial compliance, thereby enhancing blood pressure regulation.²⁷

Magnesium's Role in Insulin Function, Diabetes, and Cardiometabolic Health

Magnesium plays a crucial role in insulin action and its deficiency is often linked to insulin resistance, a hallmark of type 2 diabetes and cardiometabolic syndrome. Magnesium is involved in over 300 enzymatic reactions, regulating glucose metabolism and insulin signaling. It facilitates insulin receptor activation, enhances tyrosine kinase activity, and promotes downstream signaling pathways essential for glucose uptake in cells. Magnesium also regulates the activity of GLUT-4, the insulin-responsive glucose transporter, improving cellular glucose utilization. Deficiency in magnesium impairs these processes, contributing to insulin resistance and hyperglycemia.^{28,29}

Clinical studies suggest that increasing magnesium intake may improve insulin sensitivity and reduce the risk of cardiovascular complications by promoting better metabolic control.³⁰

Magnesium's Impact on Oxidative Stress and Persistent Inflammation

Magnesium acts as a cofactor for enzymes involved in antioxidant defense systems, such as superoxide dismutase (SOD) and glutathione peroxidase (GPx), helping to neutralize free radicals. Magnesium deficiency has been linked to increased oxidative stress, which occurs when the balance between reactive oxygen species (ROS) and antioxidants is disrupted, leading to cellular damage.³¹ In chronic inflammation, magnesium acts as a natural inhibitor of pro-inflammatory cytokines like TNF- α , IL-6, and CRP, which are involved in the pathogenesis of many chronic diseases. Insufficient magnesium levels can thus promote a persistent inflammatory state, contributing to disease progression and complications.^{32,33} Studies suggest that maintaining optimal magnesium levels may

reduce chronic inflammation and oxidative damage, potentially lowering the risk of associated diseases.³⁴

Magnesium Deficiency and Hypertension in the Elderly

In older adults, magnesium deficiency may exacerbate hypertension due to reduced magnesium intake, impaired renal function, and the increased use of medications that deplete magnesium. Studies suggest that magnesium supplementation can help lower both systolic and diastolic blood pressure, especially in individuals with magnesium deficiency.³⁵ Magnesium effects are believed to stem from its ability to regulate sodium and potassium balance, influence the renin-angiotensin-aldosterone system, and reduce inflammation and oxidative stress, which are often elevated in the elderly with hypertension. Addressing magnesium deficiency in the elderly may thus be an effective strategy for managing age-related hypertension and preventing complications.³⁶⁻³⁸

The Role of Magnesium in Managing Pre-eclampsia

Magnesium sulfate is commonly used in clinical settings to prevent and treat severe preeclampsia and eclampsia, as it has both anticonvulsant and vasodilatory effects. Magnesium sulfate is believed to relax smooth muscles, improving blood flow and reducing vascular resistance. Magnesium is also thought to inhibit the release of pro-inflammatory cytokines and reduce oxidative stress, contributing to endothelial dysfunction of preeclampsia.³⁹ Furthermore, magnesium helps modulate calcium levels, involved in the contraction of blood vessels. This regulation may help in preventing the complications of severe preeclampsia, such as stroke and organ damage. Studies have demonstrated that magnesium supplementation may also help prevent the progression of preeclampsia in high-risk pregnant women.^{40,41} However, the optimal dosage and duration of magnesium therapy in preeclampsia remain areas of active research.

Crosstalk between Mg and PAF

PAF is a potent lipid mediator involved in platelet aggregation, endothelial dysfunction, and the promotion of inflammatory pathways. It has been implicated in several cardiovascular diseases, including atherosclerosis and thrombosis.⁴² Magnesium, as a natural calcium antagonist, influences platelet aggregation and activation by modulating intracellular calcium levels. It inhibits PAF-induced platelet aggregation, thus reducing the thrombotic response. Magnesium also suppresses the release of PAF from platelets and endothelial cells. Moreover, magnesium deficiency can increase the expression of PAF receptors on platelets, enhancing their reactivity and promoting clot formation. This creates a positive feedback loop where low magnesium levels exacerbate platelet aggregation, endothelial dysfunction, and systemic inflammation. Conversely, adequate magnesium levels help balance this interaction, reducing the effects of PAF and mitigating its pro-thrombotic and pro-inflammatory actions.^{43,44}

DISCUSSION

Magnesium is a critical element involved in numerous physiological processes that underscore its essential role in maintaining health and managing various diseases. Its function as a natural calcium channel blocker allows it to regulate vascular tone by inhibiting calcium influx into vascular smooth muscle cells, promoting

vasodilation, and reducing peripheral resistance.²⁰ This property is particularly significant in hypertension management, as magnesium supplementation has been shown to improve arterial stiffness and enhance endothelial nitric oxide (NO) production, a critical mediator of vascular health.⁴⁵ Magnesium deficiency, conversely, leads to calcium-mediated vasoconstriction and endothelial dysfunction, exacerbating cardiovascular risks.²

In addition to its vascular benefits, magnesium influences the renin-angiotensin-aldosterone system (RAAS), reducing angiotensin II-induced vasoconstriction and aldosterone-mediated sodium retention. This dual action helps mitigate hypertension, with studies demonstrating magnesium's ability to attenuate RAAS hyperactivation.⁴⁶ Furthermore, magnesium's role in endothelial function is multifaceted, as it enhances NO bioavailability, reduces oxidative stress by acting as a cofactor for antioxidant enzymes, and suppresses pro-inflammatory cytokines such as TNF- α and IL-6. These mechanisms collectively preserve vascular integrity and reduce inflammation, as supported by studies showing improved endothelial-dependent vasodilation with magnesium supplementation.⁵

Magnesium also plays a pivotal role in glucose metabolism by facilitating insulin receptor activation and enhancing GLUT-4 activity, thereby promoting cellular glucose uptake.⁴⁷ Clinical studies have established a strong link between magnesium deficiency and insulin resistance, which is a hallmark of type 2 diabetes.⁴⁸ Magnesium supplementation has been shown to improve insulin sensitivity and reduce the risk of cardiovascular complications associated with diabetes.⁴⁹ Additionally, its antioxidative properties help neutralize reactive oxygen species (ROS) and inhibit pro-inflammatory pathways, mitigating chronic inflammation that contributes to the progression of metabolic disorders.^{50,51}

The role of magnesium extends to specific populations, including the elderly and pregnant women. In older adults, magnesium deficiency exacerbates hypertension due to reduced intake, impaired renal function, and the use of magnesium-depleting medications. Addressing this deficiency through supplementation has demonstrated efficacy in lowering systolic and diastolic blood pressure, particularly in magnesium-deficient individuals.⁴⁹ In pregnancy, magnesium sulfate has proven effective in preventing and treating severe preeclampsia and eclampsia by relaxing smooth muscles, reducing vascular resistance, and mitigating endothelial dysfunction.⁵¹ The interaction of magnesium with platelet-activating factor (PAF) further underscores its cardiovascular benefits, as it inhibits PAF-induced platelet aggregation, reduces thrombotic responses, and balances systemic inflammation.⁵⁰

In summary, magnesium's multifaceted roles in regulating vascular tone, glucose metabolism, antioxidant defense, and inflammation highlight its therapeutic potential.⁵¹⁻⁵² Its deficiency is associated with numerous pathological states, emphasizing the importance of maintaining optimal magnesium levels for cardiovascular and metabolic health.

CONCLUSION

Despite robust evidence supporting magnesium's role in vascular health and hypertension, gaps remain in understanding its precise mechanisms, optimal supplementation strategies, and long-term

safety. Current studies often lack large sample sizes, standardized dosages, and longitudinal follow-ups. The interplay between magnesium and other nutrients, genetic predispositions, and metabolic pathways warrants further exploration. Future research should focus on personalized approaches, clarifying its impact on diverse populations and comorbidities. Magnesium's established roles as a calcium antagonist, endothelial protector, and anti-inflammatory agent highlight its therapeutic potential. Addressing research gaps could unlock its full benefits for hypertension and cardiovascular health.

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