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Magnesium Homeostasis and Implications for Human Health: A Review of the Literature

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Abstract

Magnesium ions (Mg^{2+}) is a vital ion for human health, involved in numerous bodily functions, including energy production and protein synthesis. Despite its significance, serum magnesium levels are often overlooked in clinical settings, even though they can be disrupted in various health conditions. This review delves into the role of magnesium in human health and disease, highlighting its importance in key organs like the brain, heart, and muscles. Magnesium supplementation has shown promise in treating conditions such as migraine, depression, heart disease, and asthma. Recent discoveries have unveiled genetic factors, including mutations in specific genes, that can lead to magnesium deficiency. Additionally, certain medications, such as diuretics and proton pump inhibitors, can contribute to low magnesium levels. This review explores the mechanisms of magnesium regulation in the intestines, kidneys, and bones.

Keywords
Magnesium, physiology

Introduction

Magnesium ions (Mg^{2+}) is essential for the structural integrity and catalytic activity of DNA and RNA polymerase[1]. DNA polymerases contain two Mg^{2+} binding sites, believed to facilitate conformational changes in the enzyme during its catalytic action[2]. Studies have shown that the release of one Mg^{2+} ion is necessary to open the catalytic site for incorporating new nucleotides[3]. Additionally, Mg^{2+} is crucial for cellular DNA repair mechanisms, including nucleotide excision repair (NER), base excision repair (BER), and mismatch repair (MMR). It serves as a cofactor for nearly all enzymes involved in NER[4]. In BER, Mg^{2+} is indispensable for the function of endonucleases that cleave damaged DNA and for DNA polymerases and ligases that fill and seal the resulting gap[5]. In MMR, Mg^{2+} availability affects the activity of several Mg^{2+}- and ATP-dependent enzymes. Other Mg^{2+}-dependent enzymes include topoisomerases, helicases, exonucleases, protein kinases, cyclases, and ATPases, underscoring Mg^{2+}'s vital role in DNA replication, RNA transcription, amino acid synthesis, and protein production. Collectively, Mg^{2+} is a cornerstone in maintaining genomic and genetic stability, with its deficiency linked to cancer risk[6].

Beyond its role in nucleic acid and protein metabolism, Mg^{2+} is a critical regulator of glycolysis, serving as a cofactor for adenine nucleotides. Mg-ATP is essential for key glycolytic enzymes such as hexokinase, phosphofructokinase, aldolase, phosphoglycerate kinase, and pyruvate kinase. This makes Mg^{2+} availability pivotal for glucose metabolism, potentially explaining its association with type 2 diabetes mellitus[7].

Furthermore, Mg^{2+}'s importance extends across cellular functions. As a necessary cofactor for kinases, ATPases, guanylyl cyclases, and adenylyl cyclases, Mg^{2+} is involved in virtually every cellular process, emphasizing its fundamental role in cell physiology.

Analysis of the Literature

This part of the review focuses on the organ-specific functions of Mg^{2+} and provides an overview of diseases in which Mg^{2+} may play a role, including diabetes, osteoporosis, Parkinson's disease, coronary artery disease and immunity.

Diabetes

Magnesium ions (Mg^{2+}) appears to affect insulin secretion in the islets of Langerhans, but experimental results are inconsistent, with some studies reporting stimulation and others inhibition. For instance, Milner and Hales[24] demonstrated that Mg^{2+} suppressed insulin secretion in an ex vivo rabbit pancreas model. These conflicting findings may be explained by differences in Ca^{2+} availability across studies. As Mg^{2+} likely influences insulin secretion

through its antagonistic relationship with Ca^{2+}, the effect may rely more on the cytosolic Ca^{2+}/Mg^{2+} ratio than on Mg^{2+} levels alone[30].

Patients with type 2 diabetes mellitus frequently exhibit low serum Mg^{2+} levels[8], which are associated with poorer disease outcomes and may even increase mortality risk[9]. Hypomagnesemia is thought to contribute to the development of type 2 diabetes by promoting insulin resistance. Insulin receptors (IR), part of the tyrosine kinase receptor family, require the binding of two Mg^{2+} ions for their kinase activity[10]. Activation of the IR triggers a complex intracellular signaling cascade mediated by insulin receptor substrate proteins. In Mg^{2+}-deficient conditions, this activation is impaired, leading to reduced signal transduction and contributing to insulin resistance. Studies on hypomagnesemic rats support this, showing decreased IR phosphorylation, though differences between specific organs were observed.

In addition, Mg^{2+} deficiency may exacerbate insulin resistance through increased expression of factors such as IL-1, IL-6, IL-8, TNF-α, norepinephrine, epinephrine, and reactive oxygen species (ROS)[11]. Genetic variations also play a role; certain SNPs in the TRPM6 gene are linked to a higher risk of developing type 2 diabetes. These SNPs prevent TRPM6 from being activated by insulin, further implicating Mg^{2+} in diabetes progression[12].

Clinical studies on Mg^{2+} supplementation in type 2 diabetes patients have yielded mixed results. While some have reported significant reductions in glycated hemoglobin (HbA1c) levels and fasting glucose concentrations, others have not observed notable improvements in glycemic control. Despite these inconsistencies, Mg^{2+} supplementation remains a potentially promising approach to improving glycemic control in diabetes patients[13].

Osteoporosis

The structure of bone hydroxyapatite is primarily composed of inorganic Phosphate and Ca^{2+} ions, with Mg^{2+} ions binding to the surface of the crystals. Mg^{2+} enhances mineral solubility, influencing both the size and formation of the crystals. In bones lacking sufficient Mg^{2+}, the crystals tend to be larger, making the bone more brittle and prone to fractures. Additionally, Mg^{2+} promotes osteoblast proliferation, meaning that a deficiency in Mg^{2+} can lead to reduced bone formation[25].

Numerous studies have linked low serum Mg^{2+} levels to osteoporosis, with most research focusing on postmenopausal women[14]. However, some evidence suggests reduced bone Mg^{2+} content in older individuals, even when serum Mg^{2+} levels are normal. In such cases, increased Mg^{2+} retention during a loading/tolerance test has been observed.

A few small-scale studies have investigated the impact of oral Mg^{2+} supplementation (200–750 mg/day) on bone mineral density (BMD) in osteoporosis patients. One early study in

1991 reported an 11% increase in BMD after 12 months of daily 600 mg Mg supplementation, although the concurrent use of other supplements, including 500 mg/day of calcium, complicates the attribution of this effect solely to Mg^{2+}.

Subsequent studies across various populations have consistently found that Mg^{2+} supplementation can enhance BMD, albeit modestly (1–3%). However, the small sample sizes of these studies limit the strength of their conclusions. Larger-scale investigations are needed to confirm the potential of Mg^{2+} supplementation as a treatment for osteoporosis[15, 16].

Parkinson's Disease

Low serum Mg^{2+} levels are linked to a wide range of neurological conditions, including migraine, depression, epilepsy, schizophrenia, bipolar disorder, neuroses, addiction, stress, and Alzheimer's disease. Neuronal Mg^{2+} concentrations play a crucial role in regulating the excitability of N-methyl-d-aspartate (NMDA) receptors, which are essential for excitatory synaptic transmission, neuronal plasticity, and excitotoxicity. These receptors are pivotal in developmental plasticity, learning, and memory.

While Mg^{2+} deficiency is suggested to contribute to the etiology of these neurological disorders, current evidence is primarily epidemiological in nature[29]. Parkinson's disease is marked by the degeneration of dopaminergic neurons and is associated with reduced Mg^{2+} levels in the cortex, white matter, basal ganglia, and brain stem. Notably, rats with prolonged low Mg^{2+} intake demonstrate a significant loss of dopaminergic neurons, mirroring aspects of the disease.

At the cellular level, Parkinson's disease is often modeled using differentiated PC12 cells and the neurotoxin 1-methyl-4-phenylpyridinium ion (MPP+). In such models, decreased mitochondrial Mg^{2+} concentrations have been observed, as shown using the mitochondrial KMG-301 fluorescent Mg^{2+} probe[17]. Additionally, the Mg^{2+} transporter SLC41A1, located on the PARK16 locus associated with Parkinson's, has been implicated in the disease.

Research on the SLC41A1-pA350V single nucleotide polymorphism (SNP) linked to Parkinson's has revealed a gain-of-function effect. These findings suggest that Mg^{2+} supplementation could offer therapeutic benefits for individuals with Parkinson's disease[18].

Coronary Artery Disease

Over the past two decades, numerous studies have shown that low serum Mg^{2+} levels and inadequate Mg^{2+} intake are linked to a higher risk of coronary artery disease (CAD),

atherosclerosis, and metabolic syndrome. Additionally, low Mg^{2+} levels in CAD patients have been associated with increased mortality risk[19, 20].

Ions Mg^{2+} supplementation may benefit CAD patients in several ways. Its potent anti-inflammatory effects contribute to improved lipid profiles, reduced free oxygen radical production, and enhanced endothelial function. Mg^{2+} also inhibits blood clot formation by reducing platelet aggregation and acts as a strong vasodilator, making it a key factor in both the prevention and management of CAD[21].

Ions Mg^{2+} plays a crucial role in vascular health. Low serum Mg^{2+} levels have been linked to increased carotid intima-media thickness and a higher risk of sudden cardiac death. Clinical studies provide further support for its benefits: in a randomized, double-blind, placebo-controlled trial involving 50 CAD patients, oral Mg^{2+} supplementation improved endothelial function.

In another trial with 42 CAD patients, Mg^{2+} reduced platelet-induced thrombosis[22]. Moreover, a six-month Mg^{2+} supplementation regimen enhanced maximal oxygen uptake (Vo_2 max) and left ventricular ejection fraction (LVEF) in 53 CAD patients[23]. These findings underscore the importance of monitoring Mg^{2+} levels in CAD patients and highlight its potential as a therapeutic agent to improve patient outcomes and quality of life.

Magnesium in Immunity

Magnesium ions (Mg^{2+}) are known to have anti-inflammatory properties. They work by decreasing the production and release of substances like substance P, which promote inflammation. Additionally, Mg^{2+} can affect the immune system by controlling the growth and development of lymphocytes[29].

Research has shown that a lack of magnesium can lead to chronic inflammation. This is because low magnesium levels can trigger the release of inflammatory substances like interleukin-1 (IL-1) and tumor necrosis factor (TNF)[26]. Additionally, magnesium deficiency can activate various cellular processes that contribute to inflammation, such as phagocytosis, calcium influx, and the activation of specific signaling pathways.

Magnesium is crucial for many bodily functions, including those related to blood vessels and inflammation. Studies have demonstrated that insufficient magnesium can promote blood clot formation, impair blood vessel health, and hinder the growth and movement of endothelial cells. Furthermore, low magnesium levels can exacerbate inflammation by activating the IL-33/ST2 pathway, a key inflammatory pathway[28].

Endothelial dysfunction, a condition often associated with low magnesium levels, can lead to the release of inflammatory substances. Magnesium sulfate, a compound containing

magnesium, has been shown to possess anti-inflammatory properties[27]. It can reduce the production of inflammatory mediators and block specific ion channels in immune cells.

Human studies have linked low blood magnesium levels and inadequate dietary magnesium intake to systemic inflammation. Individuals with lower magnesium consumption tend to have higher levels of inflammation and are more likely to develop metabolic syndrome[28].

Conclusion

In recent years, Mg^{2+} has emerged as a potential treatment for various significant health conditions, including preeclampsia, stroke, heart attacks, and asthma, as demonstrated by several large-scale clinical trials. This growing interest has captured the attention of neurologists, cardiologists, and pulmonologists. Despite its importance, Mg^{2+} levels are often overlooked in routine clinical practice, even though a substantial number of critically ill patients exhibit Mg^{2+} deficiency.

Routine measurement of serum Mg^{2+}, alongside other electrolytes like sodium, potassium, and calcium, could be beneficial for early detection of imbalances and prompt intervention. Mg^{2+} disturbances can manifest as muscle cramps, irregular heart rhythms, and seizures, highlighting the need for consideration of Mg^{2+} levels in patients presenting with these symptoms. However, the precise molecular mechanisms underlying the effects of Mg^{2+} in the brain, heart, and lungs remain largely unexplored. Unraveling these mechanisms could further expand the therapeutic potential of Mg^{2+} in clinical settings.

Genetic and drug-induced disorders of Mg^{2+} homeostasis have significantly advanced our understanding of Mg^{2+} reabsorption in the kidneys and intestines. These studies exemplify the powerful synergy between clinical and fundamental research. For instance, insights into the role of EGF in renal Mg^{2+} handling have led to the standardization of Mg^{2+} measurements in patients receiving EGFR blockers, enabling early detection of imbalances and adjustments in treatment strategies.

By fostering similar collaborations between clinical and fundamental researchers in the fields of brain, heart, and lung Mg^{2+} research, we may be able to elucidate the unexplained role of Mg^{2+} in conditions like migraine, depression, epilepsy, COPD, and hypertension. In the past, Mg^{2+} research has primarily focused on the kidneys and intestines. By expanding our focus to include the heart, brain, and lungs, and by integrating both clinical and fundamental perspectives, we can ensure that Mg^{2+} is no longer overlooked as a crucial cation.

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