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Review Article

Magnesium: An Essential Mineral for Optimal Wellness

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Abstract

Magnesium (Mg) is the second most abundant intracellular cation and the fourth most abundant mineral in the human body. Mg is involved in multiple biochemical reactions, and its numerous activities are beneficial to our bodies. This review outlines the health significance of Mg in its physiologically beneficial role in function, the sources of dietary Mg along with symptoms of Mg deficiency and the health problems that come from it. Mg is a cofactor in various (more than 300) enzymes and essential for the synthesis of certain neurotransmitters, muscle cells' capacity to contract and relax, and brain functionality. The proper levels of Mg in cells are achieved through membrane channels and transporters (e.g., TRPM7, MagT1, SLC41A1). These include green leafy vegetables, nuts, seeds, legumes, and whole grains as good sources for Mg. Low levels of such an essential substance in the body can heighten susceptibility to chronic diseases such as metabolic syndrome, Type 2 diabetes, obesity, and cardiovascular morbidity. And inadequate Mg can manifest in symptoms like muscle weakness, fatigue, and cardiac arrhythmias. Not only that, but adequate Mg is needed to maintain bone density and reduce susceptibility to osteoporosis. A sufficient intake of Mg will help to mitigate health problems caused by a deficit of Mg and reduce the incidence of chronic diseases. Healthcare providers need to educate patients on consuming Mg-rich foods and, when indicated, when Mg supplementation is indicated, especially with high-risk individuals and/or those with chronic conditions.

Keywords: Magnesium, chronic diseases, deficiency, mineral.

INTRODUCTION

As an essential mineral, magnesium (Mg) is the fourth most commonly occurring cation in the human body and plays a crucial role due to its multifaceted physiological and biochemical functions [1]. Magnesium is amongst the most prevalent intracellular cations. Second only to potassium, Mg is essential to cellular functions [2]. Approximately 50-60% of the body's Mg is found in the bones, while the remaining 40-50% of Mg is stored in the soft tissue and the intracellular compartment [3]. Magnesium stored in the tissues and intracellular compartment is critical for regulating the activity of enzymes; energy metabolism; and moving ions through cell membranes [4]. Even though Mg has many essential biological functions, Mg deficiency is an issue all over the world and is a growing public health issue. Not getting enough Mg through diet, poor stomach absorption of Mg, too much Mg loss through urine, and losses through excessive sweat due to physical activity are some of the ways low Mg will cause a magnesium deficiency [5, 6]. More recent dietary trends, which include an increased intake of processed and refined foods, have played a significant role in the decrease of Mg in our diets, therefore resulting in a higher incidence of Mg deficiency syndromes [7].

Normal cellular function requires Mg. Magnesium is a cofactor for more than 300 enzymes. Enzymes that function in geological and ludimia-synthesis, and more generally to power the body's energy metabolism, function through Mg [8]. Magnesium functions with ATP, the chemical energy used by cells to produce energy. At the outset, magnesium and ATP are connected as a full complex (Mg-ATP). In cases where the amount of magnesium in a cell is insufficient, there will be a negative impact on the ability of the cells to utilize ATP and to transfer energy [4]. As a result, magnesium deficiency can negatively impact the generation of energy within cells, resulting in fatigue, muscle weakness, and decreased performance [9]. In addition to metabolic activities, magnesium also has a significant role in neuromuscular function and the cardiovascular system [10, 11]. It acts as an ion channel regulator, stabilizer of cell membranes, and controls the flow of calcium, potassium, sodium, and other ions across the cell membrane [2]. As a result, magnesium plays a key role in nerve impulse transmission, muscle

contraction/relaxation, and maintaining a regular heartbeat [12]. Within the nervous system, magnesium is active in neurotransmitter release and synaptic plasticity, which are necessary for healthy cognitive and emotional well-being [13]. Disruption of magnesium balance has been linked to neurological disorders, changes in mood (depression/anxiety), and increased stress responses [14].

While helping maintain strong bones through proper mineral composition and healthy calcium metabolism, magnesium also activates Vitamin D, regulating calcium absorption and how much bone is broken down and rebuilt (bone turnover). A low level of Magnesium can disrupt the natural equilibrium between bone growth and bone loss, putting patients at risk for osteoporosis and fractures. In addition to bone health, Magnesium is critical to maintaining proper immune function and modulating inflammatory responses, establishing magnesium as a key mineral in supporting systemic health and function. The body maintains its concentration of magnesium using a variety of mechanisms to both absorb magnesium through the intestines and excrete or store it through the kidneys. Additionally, several transport proteins/channels assist with the movement of magnesium into and out of cells, including transient receptor potential melastatin 6/7 (TRPM6/7), magnesium transporter 1 (MagT1), and solute carrier family 41 member 1 (SLC41A1). A problem with any of these transport mechanisms hinders the proper absorption and distribution of magnesium in the body, greatly increasing the chance of developing a magnesium deficiency. Magnesium comes from a variety of food sources, including green leafy vegetables, legumes, nuts, seeds, and whole grains. In spite of this wide choice of dietary sources, research suggests that many people do not consume enough magnesium. Low magnesium intake can lead to an increased risk for several chronic conditions, including metabolic syndrome, Type 2 diabetes, obesity, hypertension, and cardiovascular disease, making magnesium an increasingly important nutrient to consider in both the clinical and research settings. This review will outline the biological functions of magnesium in humans, as well as the regulatory mechanisms that are responsible for maintaining normal levels of magnesium in our bodies. Additionally, the review will discuss the different food sources of magnesium, plus the health-related complications resulting from insufficient magnesium intake, thereby providing a comprehensive overview of the relationship between magnesium and the health promotion of humans.

Distribution of Magnesium:

An adult has approximately 24 g of magnesium in his or her body and almost 99% of it is found inside the cells, mostly in the bones, muscles, and non-muscular soft tissues. The other 1% is found outside of the cells. Of the total amount of magnesium stored in the body, approximately half to a little over half is stored as part of the bone and the other half to a little less than half is found in soft tissues and other intracellular compartments [15]. Bone acts as a large reservoir for magnesium because it can easily release magnesium and help stabilize any sudden changes in the amount of magnesium in the bloodstream. About 33% of the magnesium stored in bone is readily available for exchange and helps to keep the level of magnesium in the bloodstream (extracellular) stable; this is called the buffering effect [1]. The intracellular concentration of magnesium is between 5 and 20 mmol/l and 1-5% of this magnesium is ionized, while the rest is bound to proteins, other negatively charged molecules, and ATP [16]. The amount of extracellular magnesium is about 1% of the total amount of magnesium in the body and is found primarily in serum and red blood cells. The level of magnesium in serum consists of three separate fractions, i.e., the ionized, protein-bound, and complex fractions. The normal serum magnesium level is between 1.7 and 2.6 mg/dl [17].

Homeostasis of Magnesium:

Magnesium that is absorbed through the intestine is balanced by the excretion of Mg through the kidneys, and any acute shortages of Mg are compensated for through the release of Mg from the bones to maintain a normal serum level of Mg [18]. Therefore, the intestine, kidney, and bone make up three primary organs that regulate and maintain Mg homeostasis.

Most of the Mg the body absorbs occurs in the jejunum and ileum part of the small intestine via two mechanisms: Passive paracellular transport and active transcellular transport. Passive transport occurs through the tight junctions of enterocytes, while active transport is done by means of specific Mg transporters called transient receptor potential melastatin channels 6 (TRPM6) and TRPM7, which help Mg move into the enterocytes [19]. The Mg absorption process is also influenced by vitamin D status, total dietary intake, and other nutrients present [20].

Once Mg has been absorbed into the blood stream, it is moved throughout the body, with about 99% of all magnesium stored in the bone and soft tissues, 60% in bone tissue, and 40% in soft tissue. The rest is found in just 1% of the extracellular space, but this small amount of magnesium serves many important functions within the body [2]. The kidneys are very important regulators of magnesium balance for the body. After filtration, approximately 20 to 30 percent of the filtered magnesium is reabsorbed into the proximal tubule, 60 to 70 percent is reabsorbed in the thick ascending limb of the loop of Henle, and approximately 5 to 10 percent is reabsorbed in the distal convoluted tubule [17]. Therefore, the quantity of magnesium excreted from the kidneys is precisely regulated according to the body's needs. Parathyroid hormone (PTH) and calcitonin are reported as the principal players of magnesium homeostasis. PTH promotes the renal reabsorption and

calcitonin promotes magnesium retention. In the other hand vitamin D enhance the intestinal absorption of magnesium [21].

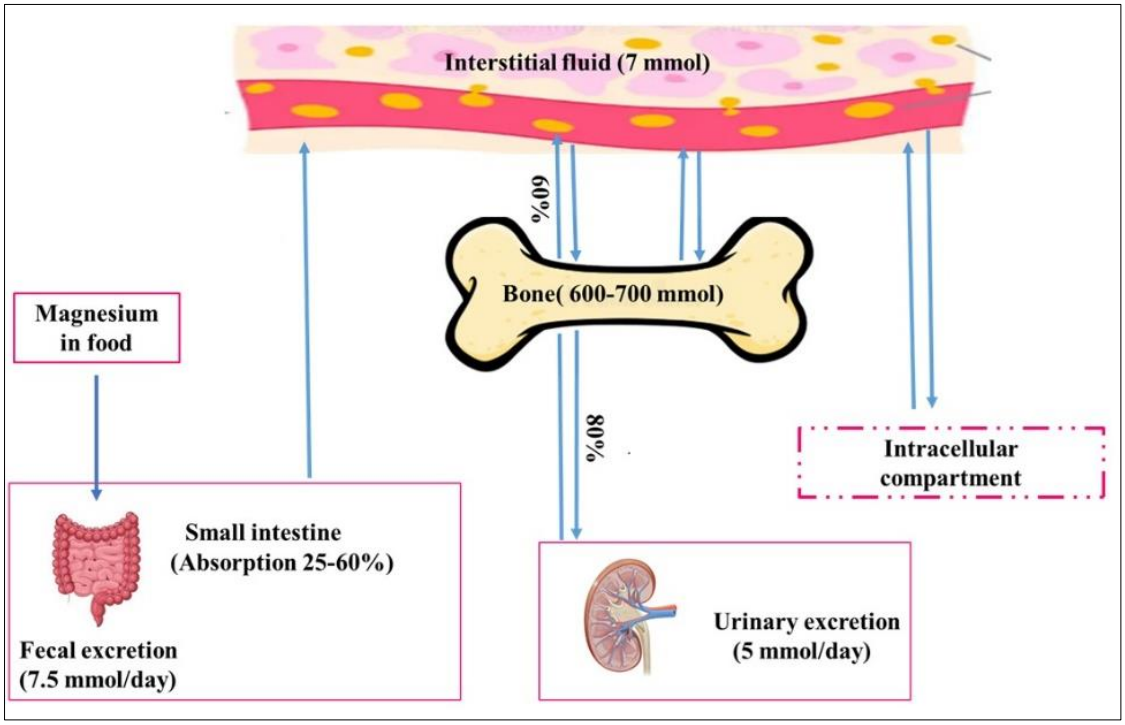

Figure 1: Absorption and excretion of magnesium

Recommended Daily Allowance (RDA) of magnesium:

The RDA for magnesium varies depending on the individual's gender, age, and life cycle. Adult males are recommended to get 400-420 mg of magnesium daily, while adult females are recommended to get 310-320 mg daily [22]. Adolescents, pregnant and breastfeeding women, and those who perform high levels of aerobic exercise are recommended to have higher amounts of magnesium intake [23]. A deficiency of magnesium can lead to various health problems including muscle cramps, tiredness/fatigue, weakness, cardiovascular issues and possible long-term effects. Studies have shown that people with long-term magnesium deficiencies are at greater risk of developing cardiovascular diseases, having Type 2 Diabetes, migraines and osteoporosis [24].

Causes of magnesium deficiency:

Magnesium is a mineral that plays an important role in numerous body functions. Insufficient Mg intake has become a global issue and is now the primary cause of Mg deficiency. Most people are consuming less than the recommended daily intake of Mg [25]. Increasingly refined food choices, a lack of Mg-rich leafy greens in the diet, food preparation and cooking methods that decrease Mg levels, as well as foods rich in phytic acid, pathogenic gut organisms, and glyphosate exposure all contribute to lower dietary magnesium levels and decreased absorption of Mg from the gastrointestinal tract. [26] Other factors that may lead to Mg deficiency are excessive alcohol use; inherited disorders that affect renal transport of Mg; endocrine disorders (type 2 diabetes); and use of some medications that can interfere with Mg absorption, including diuretics, proton pump inhibitors, cardiac glycosides, calcineurin inhibitors, aminoglycoside antibiotics, and amphotericin B [27].

Magnesium, Obesity and Metabolic Syndrome

Metabolic syndrome (MS), a well-characterized comorbidity of obesity, recently have become a major public health concern in the world. The obesity and metabolic syndrome (MS) are interrelated with each other and are characterized by the excessive accumulation of fat [28, 29]. Obesity implies just the excess accumulation of fat, however MS is a condition of excess fat accumulation along with the low-grade inflammation, leading to central obesity, hypertension, dyslipidemia and insulin resistance [29].

The term "obesity" refers to an individual having an abnormally high amount of body fat. Metabolic Syndrome, also known as obesity related comorbidity, is gaining prominence as a global public health problem [30]. There is a strong correlation between MS and obesity, as both conditions exhibit excessive body fat accumulation. While the term "obesity" generally refers to an individual storing an excessive amount of fat, the MS requires additional criteria that indicate a low-grade inflammatory response

associated with this storage of fat [31, 32]. These inflammatory responses will usually be characterized by having elevated blood pressure, dyslipidemia, and decreased insulin sensitivity in the body [33].

The complexity of obesity and the lack of clarity surrounding its pathophysiology is matched only by the complexity of its causes. While there is much debate regarding the exact role played by different factors, there is no doubt that the environment, sedentary lifestyles, and unhealthy dietary patterns are the primary contributors to the development of obesity [29]. The result of this combination of risk factors is that many obese individuals suffer from Mg deficiency as documented by numerous studies [34, 35, 36]. Obese individuals demonstrate highly elevated rates of hepatic glucose metabolism; as such, many of the enzymes associated with the metabolic processes involved in the breakdown of glucose are dependent on Mg [37]. Furthermore, Mg is an essential nutrient needed to convert vitamin B1 to the bioactive form thiamine diphosphate (TDP), an important coenzyme in oxidative metabolism [38]. A decrease in the amount of intracellular Mg can lead to decreased activity of TDP-dependent pyruvate dehydrogenase (an enzyme involved in the conversion of pyruvate to acetyl CoA), thereby driving glucose metabolism toward the oxidative phase of the pentose phosphate pathway, increasing the amounts of NADPH produced [37]. Increased levels of NADPH support the anabolism of fatty acids, leading to the reformation of triglycerides and increased secretion of very-low-density lipoproteins [39]. Ultimately, this results in an increase of triglyceride formation and storage in adipocytes, thereby worsening obesity and increasing the risk of comorbid conditions associated with obesity.

The main characteristics of MS include central obesity, long-term low-grade inflammation, high blood sugar, insulin resistance, and combinations of high blood pressure, high triglycerides, and low HDL cholesterol [40]. Low dietary Mg consumption has been linked to an increased risk of developing MS. Mg consumption and MS were found to be inversely correlated in a meta-analysis by Youngyo Kim *et al.,* [41]. In a similar vein, individuals who took more magnesium had a lower likelihood of acquiring MS, in ten-year research evaluating the association between magnesium consumption and MS-related risk [42]. Additionally, an analysis of China Health and Nutrition Survey (CHNS) participants revealed a strong correlation between magnesium consumption and MS, as well as all of its risk factors [43].

Another study conducted in Siberia by Zorica Rasic et al. shown that Mg supplementation may be a useful strategy for preventing MS in regions with low Mg levels in drinking water [44]. It has been estimated that, MS increases the risk of myocardial infarction by three to four times and stroke by two to four times [29]. When these two disorders are combined, morbidity and mortality increase dramatically. A statistically significant reduction in risk factors for cardiovascular disease (CVD), type 2 diabetes mellitus, and overall mortality is associated with maintaining an appropriate intake of magnesium. Additionally, individuals who have enough magnesium in their blood are less likely to develop cardiovascular disease [45].

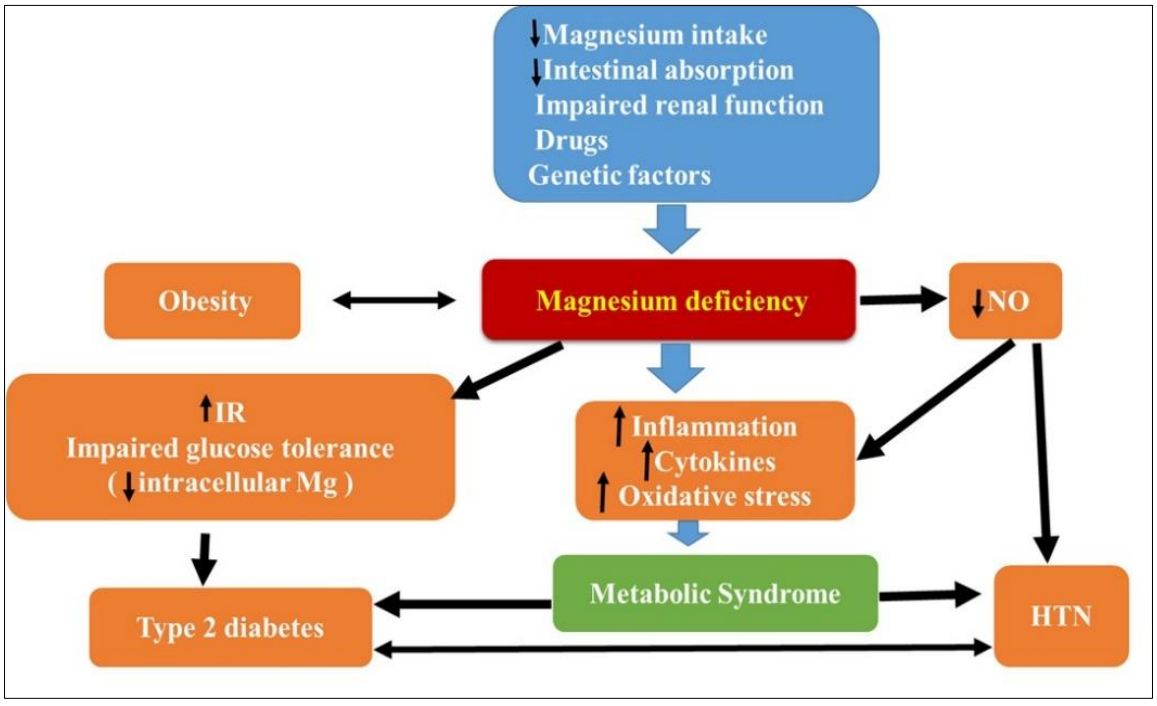

Figure 2: Illustrating the consequences of Mg deficiency

Magnesium and type 2 diabetes mellitus

A chronic deficiency of the mineral Mg has been documented to be a contributing factor to the onset of type 2 diabetes mellitus (T2DM) [46]. The incidence of Mg deficiency is especially high in T2DM individuals whose levels are poorly controlled, have been diagnosed with T2DM for long periods, and/or have chronic microvascular or macrovascular complications associated with T2DM [45]. Increased insulin resistance and a disrupted tricarboxylic acid cycle, which plays a critical role in energy metabolism, can result from chronic insufficiency in magnesium [6]. Therefore, this condition is thought to be associated with an increased risk of developing T2DM. A reduced intracellular Mg concentration has been found in T2DM patients, in comparison to nondiabetic individuals. In a meta-analysis of 286,668 individuals, the results concluded that people who maintain their daily magnesium consumption at or above 100 mg experience a decrease in their risk for developing T2DM by 15% [47]. The mechanisms through which Mg deficiency occurs in those with T2DM remain not completely known, but the storage of Mg in the cells may be inhibited due to insufficient cellular Mg uptake or complications related to the production and use of ATP. An increased loss of Mg through urine, due to altered renal function, may play a crucial role in the development of Mg deficiencies in patients with T2DM [48].

Magnesium and cardiovascular disorders

Magnesium deficiency manifests in two forms: hypomagnesemia and chronic latent magnesium deficiency. Hypomagnesemia is easily recognized, resulting in decreased serum magnesium levels (less than 1.5–1.8 mg/dL) [49]. On the other hand, chronic latent deficiency may remain stable despite normal serum concentrations, complicating its detection. This form requires a magnesium tolerance assessment using intravenous infusion and urine testing to identify it [6]. However, it is complex and also not always available in the late 1990s and early 2000s, it became evident that insufficient dietary magnesium could result in hypomagnesemia, which may have an impact on cardiovascular function [7]. Prior studies have shown that Mg presence in blood has a negative association with the occurrence of several cardiovascular diseases, including hypertension, stroke, ischemic heart disease, abnormal lipid profiles, heart failure, and overall cardiovascular morbidity and mortality [50]. Mg has been shown to have important functions in cardiovascular system like facilitating flow of ions across cell membranes, endothelium-dependent vasodilation, lowering blood pressure, mitigating inflammation, improving insulin sensitivity, and glucose metabolism. As well, magnesium partially regulates ionic channels for calcium, sodium, and potassium [51]. Mg also shows antiplatelet and anticoagulant action that serve as endogenous calcium inhibitors [52]. The transient and sustained calcium channel antagonism with Mg is particularly protective against activation. Furthermore, magnesium is involved in coronary vessel spasm reduction and regulating vascular muscle tone and systemic arterial blood pressure. [53] Hypomagnesemia impairs these reactions and causes increased concentration of sodium and calcium in the cytoplasm. Intracellular levels of calcium also have been associated with ischemia in the heart. In response to physiological signals, Mg enters the cell through membrane-integrated Mg transporter proteins [54, 55]. Research indicates that Mg plays a role in numerous cellular functions, notably within mitochondria, which act as the main intracellular reservoir for Mg. Changes in the distribution of mitochondrial Mg have been shown to affect both mitochondrial integrity and ATP production [56].

Magnesium and inflammatory reactions

Animal studies indicate that insufficient magnesium levels can initiate an inflammatory response. This response activates leukocytes and macrophages, leading to the release of inflammatory cytokines and acute-phase proteins, alongside an increase in free radicals [57]. The inflammatory process involves the activation of phagocytic cells, which triggers a reaction characterized by elevated levels of substance P, a tachykinin neuropeptide that encourages the secretion of pro-inflammatory cytokines and stimulates nuclear factor kappa B (NFkB), a transcription factor responsible for regulating genes associated with inflammation [58]. A lack of magnesium increases the recruitment of phagocytic cells, resulting in heightened production of reactive oxygen species (ROS) [57]. The excessive generation of ROS can lead to tissue damage, thus categorizing inflammatory stress as a contributing factor for various chronic diseases. Additionally, an increased entry of intracellular Ca^{2+} through the N-methyl-D-aspartate receptor has been connected to higher production of substance P, which correlates with elevated pro-inflammatory cytokine levels in animals deficient in magnesium [59]. Recent research suggests that magnesium deficiency is associated with NFkB's role in promoting TNF-alpha and IL-1B expression [60].

Under conditions of magnesium scarcity, intracellular Ca^{2+} concentrations rise due to increased influx through Ca^{2+} transporter channels and greater release from cellular reservoirs like the sarcoplasmic reticulum [61]. These observations suggest that rising cellular Ca^{2+} is a crucial mechanism through which magnesium deficiency induces inflammatory stress.

Magnesium and muscle contraction

Magnesium is vital for both muscle contraction and relaxation. It regulates calcium homeostasis, a fundamental process in muscle physiology [62]. By blocking calcium channels, magnesium aids in muscle relaxation by preventing excessive calcium entry into muscle cells [56]. A magnesium deficiency has been linked to issues such as muscle cramps, impaired muscle function, and spasms [56]. Additionally, magnesium is crucial for energy metabolism because it acts as a

cofactor in the production of ATP within muscle cells [63]. Numerous studies emphasize the significance of magnesium supplementation for enhancing muscle function and performance, underscoring the importance of maintaining adequate magnesium levels [56, 52, 64].

CONCLUSION

Magnesium is a key micronutrient that is involved in hundreds of enzymatic reactions regulating energy metabolism, protein synthesis, and nucleic acid stability. It helps muscle contraction and relaxation, neurotransmitter synthesis, and neuromuscular function. Magnesium is also important for bone health, immune modulation, and ion transport (most notably calcium and potassium balance). Even though magnesium is important, deficiency of magnesium is prevalent due to poor diet, processed food intake, absorption problems, certain diseases, or medications. And the intake of valuable things like leafy greens, whole grains, legumes, nuts, and seeds are too little and often underconsumed. A deficiency is linked to elevated susceptibility to metabolic syndrome, type 2 diabetes, obesity, and certain cardiovascular diseases. It's also connected to chronic inflammation and neuromuscular signs such as cramps and fatigue. It is very important to get adequate amounts through a well-balanced diet, supplemented in high-risk populations if necessary. Healthcare providers are the gatekeepers of deficiency and ensure the right intake and the right supplementation. More awareness and additional research are required to enhance magnesium status and improve health status.

REFERENCES

1. Mathew AA, Panonnummal R. 'Magnesium'-the master cation-as a drug—possibilities and evidences. Biometals. 2021 Oct;34(5):955-86.
2. Romani AM. Cellular magnesium homeostasis. Archives of biochemistry and biophysics. 2011 Aug 1;512(1):1-23.
3. Reddy ST, Soman SS, Yee J. Magnesium balance and measurement. Advances in chronic kidney disease. 2018 May 1;25(3):224-9.
4. Pasternak K, Kocot J, Horecka A. Biochemistry of magnesium. Journal of Elementology. 2010;15(3):601-16.
5. DiNicolantonio JJ, O'Keefe JH, Wilson W. Subclinical magnesium deficiency: a principal driver of cardiovascular disease and a public health crisis. Open heart. 2018 Jan 13;5(1).
6. Fiorentini D, Cappadone C, Farruggia G, Prata C. Magnesium: biochemistry, nutrition, detection, and social impact of diseases linked to its deficiency. Nutrients. 2021 Mar 30;13(4):1136.
7. Rosanoff A. Changing crop magnesium concentrations: impact on human health. Plant and soil. 2013 Jul;368(1):139-53.
8. Al Alawi AM, Majoni SW, Falhammar H. Magnesium and human health: perspectives and research directions. International journal of endocrinology. 2018;2018(1):9041694.
9. Carvil P, Cronin J. Magnesium and implications on muscle function. Strength & Conditioning Journal. 2010 Feb 1;32(1):48-54.
10. Souza AC, Vasconcelos AR, Dias DD, Komoni G. The integral role of magnesium in muscle integrity and aging: a comprehensive review. Nutrients. 2023 Jan;15(24):5127.
11. Shechter M. Magnesium and cardiovascular system. Magnesium research. 2010 Jun 1;23(2):60-72.
12. Zaychenko G, Горчакова Н, Klymenko O, Shumeiko O, Babák V. Physicochemical, biochemical, pharmacological properties of magnesium. Bulletin of Problems Biology and Medicine. 2023;1(2):74.
13. Slutsky I, Abumaria N, Wu LJ, Huang C, Zhang L, Li B, Zhao X, Govindarajan A, Zhao MG, Zhuo M, Tonegawa S. Enhancement of learning and memory by elevating brain magnesium. Neuron. 2010 Jan 28;65(2):165-77.
14. 14 Serefko A, Szopa A, Poleszak E. Magnesium and depression. Magnesium research. 2016 Sep 1;29(3).
15. Gröber U, Schmidt J, Kisters K. Magnesium in prevention and therapy. Nutrients. 2015 Sep;7(9):8199-226.
16. Saris NE, Mervaala E, Karppanen H, Khawaja JA, Lewenstam A. Magnesium: an update on physiological, clinical and analytical aspects. Clinica chimica acta. 2000 Apr 1;294(1-2):1-26.
17. Dechent W, Ketteler M. Magnesium basics. Clinical kidney journal. 2012 Feb 1;5(Suppl_1): i3-14.
18. Alfrey AC, Miller NL, Trow R. Effect of age and magnesium depletion on bone magnesium pools in rats. The Journal of clinical investigation. 1974;54(5): 1074–1081.
19. Stumpff F, Manneck D. Prebiotics as modulators of colonic calcium and magnesium uptake. Acta Physiologica. 2025 Feb;241(2): e14262.
20. Shahsavani Z, Asadi A, Shamshirgardi E, Akbarzadeh M. Vitamin D, magnesium and their interactions: A review. International Journal of Nutrition Sciences. 2021 Sep 1;6(3):113-8.
21. Allgrove J. Physiology of calcium, phosphate, magnesium and vitamin D. Endocr Dev. 2015 Jun 11;28:7-32.
22. EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA). Scientific opinion on dietary reference values for magnesium. EFSA Journal. 2015 Jul;13(7):4186.
23. Mazza E, Maurotti S, Ferro Y, Castagna A, Pujia C, Sciacqua A, Pujia A, Montalcini T. Magnesium: exploring gender differences in its health impact and dietary intake. Nutrients. 2025 Jul 4;17(13):2226.
24. LaValle JB. Hidden disruptions in metabolic syndrome: drug-induced nutrient depletion as a pathway to accelerated pathophysiology of metabolic syndrome. Alternative Therapies in Health & Medicine. 2006 Mar 1;12(2).

25. Razzaque MS. Magnesium: are we consuming enough? Nutrients. 2018 Dec 2;10(12):1863.
26. Cakmak, I.; Yazici, M.A.; Tutus, Y.; Ozturk, L. Glyphosate reduced seed and leaf concentrations of calcium, manganese, magnesium, and iron in non-glyphosate resistant soybean. *Eur. J. Agron.* **2009**, *31*, 114–119.
27. Zheltova AA, Kharitonova MV, Iezhitsa IN, Spasov AA. Magnesium deficiency and oxidative stress: an update. BioMedicine. 2016 Nov 17;6(4):20.
28. Jha BK, Sherpa ML, Imran M, Mohammed Y, Jha LA, Paudel KR, Jha SK. Progress in understanding metabolic syndrome and knowledge of its complex pathophysiology. Diabetology. 2023 Apr 12;4(2):134-59.
29. Jha BK, Sherpa ML, Dahal BK, Singh JK. Prevalence of metabolic syndrome and its components in adults with central obesity at Janakpur Zone, Nepal.
30. Ahmed SK, Mohammed RA. Obesity: Prevalence, causes, consequences, management, preventive strategies and future research directions. Metabolism Open. 2025 Jun 12:100375.
31. Wong JC, O'Neill S, Beck BR, Forwood MR, Khoo SK. Comparison of obesity and metabolic syndrome prevalence using fat mass index, body mass index and percentage body fat. Plos one. 2021 Jan 14;16(1): e0245436.
32. Cooke AA, Connaughton RM, Lyons CL, McMorrow AM, Roche HM. Fatty acids and chronic low-grade inflammation associated with obesity and the metabolic syndrome. European journal of pharmacology. 2016 Aug 15; 785:207-14.
33. Gluvic Z, Zaric B, Resanovic I, Obradovic M, Mitrovic A, Radak D, R Isenovic E. Link between metabolic syndrome and insulin resistance. Current vascular pharmacology. 2017 Jan 1;15(1):30-9.
34. Shamnani G, Rukadikar CA, Gupta V, Singh S, Tiwari S, Bhartiy SS, Sharma P. Serum magnesium in relation with obesity. National Journal of Physiology, Pharmacy and Pharmacology. 2018 Jul 2;8(7):1074-7.
35. Kurstjens S, van Diepen JA, Overmars-Bos C, Alkema W, Bindels RJ, Ashcroft FM, Tack CJ, Hoenderop JG, de Baaij JH. Magnesium deficiency prevents high-fat-diet-induced obesity in mice. Diabetologia. 2018 Sep;61(9):2030-42.
36. Dos Santos LR, Melo SR, Severo JS, Morais JB, da Silva LD, de Paiva Sousa M, de Sousa TG, Henriques GS, do Nascimento Marreiro D. Cardiovascular diseases in obesity: what is the role of magnesium? Biological trace element research. 2021 Nov;199(11):4020-7.
37. Piuri G, Zocchi M, Della Porta M, Ficara V, Manoni M, Zuccotti GV, Pinotti L, Maier JA, Cazzola R. Magnesium in obesity, metabolic syndrome, and type 2 diabetes. Nutrients. 2021 Feb;13(2):320.
38. Ali A, Mac Dionys Rodrigues da Costa E, Magalhães P, Martins AM. Biological importance of vitamins and minerals. Nutraceuticals: A holistic approach to disease prevention. 2024 Apr 22:63.
39. Fujii J. Redox remodeling of central metabolism as a driving force for cellular protection, proliferation, differentiation, and dysfunction. Free radical research. 2024 Oct 2;58(10):606-29.
40. JHA BK, SHERPA ML, DAHAL BK, SINGH JK, GUPTA C. Demographic and Biochemical Parameters of Community Survey Participants with Metabolic Syndrome from Terai Region of Nepal. Journal of Clinical & Diagnostic Research. 2021 Oct 1;15(10).
41. Kim Y, Je Y. Intake or blood levels of magnesium and risk of metabolic syndrome: a meta-analysis of observational studies. Nutrients. 2025 May 14;17(10):1667.
42. Mitchel MW, Moreno-De-Luca D, Myers SM, Levy RV, Turner S, Ledbetter DH, Martin CL. 17q12 recurrent deletion syndrome. GeneReviews®[Internet]. 2025 Aug 14.
43. Jiao Y, Li W, Wang L, Jiang H, Wang S, Jia X, Wang Z, Wang H, Zhang B, Ding G. Relationship between dietary magnesium intake and metabolic syndrome. Nutrients. 2022 May 11;14(10):2013.
44. Rasic-Milutinovic Z, Perunicic-Pekovic G, Jovanovic D, Gluvic Z, Cankovic-Kadijevic M. Association of blood pressure and metabolic syndrome components with magnesium levels in drinking water in some Serbian municipalities. Journal of water and health. 2012 Mar 1;10(1):161-9.
45. Fang X, Wang K, Han D, He X, Wei J, Zhao L, Imam MU, Ping Z, Li Y, Xu Y, Min J. Dietary magnesium intake and the risk of cardiovascular disease, type 2 diabetes, and all-cause mortality: a dose–response meta-analysis of prospective cohort studies. BMC medicine. 2016 Dec 8;14(1):210.
46. Soriano-Pérez L, Aranda-Rivera AK, Cruz-Gregorio A, Pedraza-Chaverri J. Magnesium and type 2 diabetes mellitus: clinical and molecular mechanisms. Health Sciences Review. 2022 Sep 1; 4:100043.
47. Bentil HJ. Dietary Magnesium Intake and Risk of Type 2 Diabetes Mellitus in Ghanaian Women of Reproductive Age. University of Rhode Island; 2022.
48. Barbagallo M, Dominguez LJ. Magnesium and type 2 diabetes. World journal of diabetes. 2015 Aug 25;6(10):1152.
49. Ehrenpreis ED, Jarrouj G, Meader R, Wagner C, Ellis M. A comprehensive review of hypomagnesemia. Disease-a-Month. 2022 Feb 1;68(2):101285.
50. Siddiqui RW, Nishat SM, Alzaabi AA, Alzaabi FM, Al Tarawneh DJ, Al Tarawneh YJ, Khan A, Khan MA, Siddiqui TW, Siddiqui SW. The connection between magnesium and heart health: understanding its impact on cardiovascular wellness. Cureus. 2024 Oct 24;16(10).

51. Wu W, Gong M, Liu P, Yu H, Gao X, Zhao X. Hypomagnesemia: exploring its multifaceted health impacts and associations with blood pressure regulation and metabolic syndrome. Diabetology & Metabolic Syndrome. 2025 Jun 16;17(1):217.
52. Davlouros P, Xanthopoulou I, Mparampoutis N, Giannopoulos G, Deftereos S, Alexopoulos D. Role of calcium in platelet activation: novel insights and pharmacological implications. Medicinal Chemistry. 2016 Mar 1;12(2):131-8.
53. Tangvoraphonkchai K, Davenport A. Magnesium and cardiovascular disease. Advances in chronic kidney disease. 2018 May 1;25(3):251-60.
54. Duffy S, MacVicar BA. In vitro ischemia promotes calcium influx and intracellular calcium release in hippocampal astrocytes. Journal of Neuroscience. 1996 Jan 1;16(1):71-81.
55. Groisman EA, Duprey A, Choi J. How the PhoP/PhoQ system controls virulence and Mg2+ homeostasis: lessons in signal transduction, pathogenesis, physiology, and evolution. Microbiology and Molecular Biology Reviews. 2021 Jun 30;85(3):10-128.
56. Souza AC, Vasconcelos AR, Dias DD, Komoni G. The integral role of magnesium in muscle integrity and aging: a comprehensive review. Nutrients. 2023 Jan;15(24):5127.
57. Nielsen FH. Magnesium deficiency and increased inflammation: current perspectives. Journal of inflammation research. 2018 Jan 18:25-34.
58. Mazur A, Maier JA, Rock E, Gueux E, Nowacki W, Rayssiguier Y. Magnesium and the inflammatory response: potential physiopathological implications. Archives of biochemistry and biophysics. 2007 Feb 1;458(1):48-56.
59. McGee MA, Abdel-Rahman AA. N-methyl-D-aspartate receptor signaling and function in cardiovascular tissues. Journal of cardiovascular pharmacology. 2016 Aug 1;68(2):97-105.
60. Lima FD, Fock RA. A review of the action of magnesium on several processes involved in the modulation of hematopoiesis. International Journal of Molecular Sciences. 2020 Sep 25;21(19):7084.
61. Pietropaolo G, Castiglioni S, Maier JA, Wolf FI, Trapani V. Magnesium Preserves Calcium Homeostasis and Contributes to Protect Myotubes from Inflammation-Induced Damage. International Journal of Molecular Sciences. 2025 Oct 11;26(20):9912.
62. Castiglioni S, Mazur A, Maier JA. The central role of magnesium in skeletal muscle: from myogenesis to performance. Magnesium research. 2024;37(1):1-1.
63. Dao CT. The influence on mitochondrial energy (ATP), lactate-pyruvate-and muscularity-metabolism (CK): Cellular magnesium level and magnesium supplementation in elite sports. Int. J. Hum. Mov. Sports Sci.. 2021;9(5).
64. Fatima G, Dzupina A, Alhmadi HB, Magomedova A, Siddiqui Z, Mehdi A, Hadi N, RAZA AM. Magnesium matters: A comprehensive review of its vital role in health and diseases. Cureus. 2024 Oct 13;16(10).