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A review of TRPM6 rs2274924 polymorphism and magnesium deficiency: Genetic–nutritional interaction in type 2 diabetes mellitus

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Abstract
The Transient Receptor Potential Melastatin 6 (TRPM6) gene plays a central role in intestinal magnesium absorption and renal reabsorption. Genetic polymorphisms in TRPM6, particularly rs2274924, have been linked to altered magnesium transport, hypomagnesemia, and increased susceptibility to type 2 diabetes mellitus (T2DM). This narrative review synthesizes current findings on the interaction between TRPM6 genetic variants and dietary magnesium in relation to glucose regulation and insulin resistance. A structured literature search was conducted in PubMed, Scopus, and Web of Science to identify observational and interventional studies exploring TRPM6 polymorphisms, magnesium status, and metabolic outcomes. Evidence consistently shows that magnesium is essential for enzymatic processes in glycolysis, ATP production, and insulin secretion, and that deficiency impairs β-cell function, glucose uptake, and insulin signaling. Several studies indicate that individuals—particularly women—harboring the rs2274924 variant may exhibit greater metabolic vulnerability when habitual magnesium intake is insufficient, while adequate intake appears to reduce this genetic risk. By summarizing evidence across molecular, nutritional, and clinical perspectives, this review highlights the importance of TRPM6–magnesium interactions and their potential implications for personalized nutrition and diabetes prevention strategies.

Keywords: TRPM6 gene, rs2274924 olymorphism, Magnesium intake, Insulin resistance, Type 2 diabetes mellitus

Abstrak
Gen Transient Receptor Potential Melastatin 6 (TRPM6) berperan penting dalam penyerapan magnesium di usus dan reabsorpsi di ginjal. Polimorfisme genetik pada TRPM6, khususnya rs2274924, telah dikaitkan dengan gangguan transport magnesium, hipomagnesemia, dan peningkatan risiko diabetes melitus tipe 2 (T2DM). *Narrative review* ini merangkum bukti terkini mengenai interaksi antara variasi genetik TRPM6 dan asupan magnesium dalam kaitannya dengan regulasi glukosa dan resistensi insulin. Pencarian literatur terstruktur dilakukan melalui PubMed, Scopus, dan Web of Science untuk mengidentifikasi studi observasional dan intervensi yang mengevaluasi polimorfisme TRPM6, status magnesium, dan luaran metabolik. Bukti menunjukkan bahwa magnesium berperan dalam proses enzimatik glikolisis, produksi ATP, serta sekresi insulin, dan bahwa defisiensinya dapat mengganggu fungsi sel β, pengambilan glukosa, dan sinyal insulin. Sejumlah studi melaporkan bahwa individu terutama perempuan dengan varian rs2274924 memiliki kerentanan metabolik lebih tinggi ketika asupan magnesium tidak mencukupi, sementara asupan yang memadai tampak mengurangi risiko tersebut. Dengan mengintegrasikan temuan dari aspek molekuler, nutrisi, dan klinis, review ini menegaskan pentingnya interaksi TRPM6–magnesium serta implikasinya terhadap nutrisi personal dan strategi pencegahan T2DM.

Kata kunci: Gen TRPM6, polimorfisme rs2274924, asupan magnesium, resistensi insulin, diabetes mellitus tipe 2

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1. Introduction

Magnesium (Mg^{2+}) is an essential mineral involved in more than 600 enzymatic reactions, including energy metabolism, neuromuscular function, and glucose homeostasis. Maintaining optimal Mg^{2+} balance requires tightly regulated intestinal absorption and renal reabsorption. Among the molecular regulators of magnesium homeostasis, the Transient Receptor Potential Melastatin 6 (TRPM6) channel plays a central and irreplaceable role. TRPM6 is highly expressed in the distal convoluted tubule (DCT) of the kidney and the intestine, functioning as the primary epithelial Mg^{2+} entry pathway (van der Wijst et al., 2014).

The TRPM6 gene plays a crucial role in magnesium absorption from the intestine and magnesium reabsorption from body fluids in the distal convoluted tubules of the kidneys. When the body requires additional magnesium, the TRPM6 channels facilitate magnesium uptake in the intestines and reabsorption in the kidneys. Conversely, when magnesium levels are sufficient or excessive, these channels promote magnesium excretion through the urine (Alina, 2021). Impaired magnesium reabsorption observed in individuals with TRPM6 mutations underscores the essential role of the TRPM6 channel in regulating epithelial magnesium movement and homeostasis (Chubanov et al., 2004).

Mutations in the TRPM6 gene can lead to hypomagnesemia, a condition characterized by low levels of magnesium and calcium in the body. This condition may cause neurological symptoms such as muscle spasms (Alina, 2021). Hypomagnesemia (serum Mg^{2+} concentration <0.7 mmol/L) is common in individuals with type 2 diabetes mellitus (T2DM). Evidence shows that hypomagnesemia occurs up to ten times more frequently in T2DM patients compared with the general population and is associated with insulin resistance, hyperglycemia, and accelerated disease progression. In contrast, hypomagnesemia is rarely observed in individuals with type 1 diabetes (T1D), highlighting insulin resistance as a key factor underlying magnesium homeostasis disturbances (Oost et al., 2023).

The prevalence of hypomagnesemia in the healthy population is approximately 2% (Simmons et al., 2010). Low serum Mg^{2+} levels are even more common in hospitalized patients, reaching up to 60% among those in intensive care units. In T2DM, cohort studies report a highly variable prevalence of hypomagnesemia, ranging from 9.1% to 47.7% (Oost et al., 2023). In the context of T2DM, TRPM6 mutations have been linked to an increased risk of the disease, particularly among women with low magnesium intake (Alina, 2021).

Considering these observations, TRPM6 emerges not only as a regulator of mineral balance but also as a potential determinant of metabolic health. Therefore, investigating the TRPM6 rs2274924 polymorphism is essential for elucidating its role in the pathogenesis of T2DM. This review aims to explore the relationship between the TRPM6 rs2274924 gene variant and type 2 diabetes mellitus through a synthesis of existing literature and to provide dietary recommendations for individuals with hypomagnesemia as a risk factor for T2DM.

2. Research Method

This narrative literature review was conducted to examine the relationship between the TRPM6 rs2274924 gene polymorphism, magnesium intake, and the risk of type 2 diabetes mellitus. Relevant scientific articles were collected from databases including PubMed, ScienceDirect, Scopus, SpringerLink, and Google Scholar using the keywords *"TRPM6 gene," "rs2274924 polymorphism," "magnesium," "type 2 diabetes,"* and *"insulin resistance".* Publications from 2000 to 2024 were screened.

Studies were selected through a two-stage screening process (title/abstract screening followed by full-text review). Inclusion criteria involved original research articles (observational studies, cohort studies, case–control studies, clinical trials) and review articles that discussed TRPM6 polymorphisms, magnesium metabolism, or their association with glucose regulation and T2DM. Studies were included if they involved human populations or relevant experimental models that contributed to understanding gene nutrient interactions. Exclusion criteria included articles that were irrelevant to TRPM6 or magnesium deficiency, studies with incomplete data, non-scientific reports, editorials, and publications not available in English. Data were analyzed descriptively to identify consistent patterns linking TRPM6 gene variations, magnesium deficiency, and glucose metabolism disorders. The findings were synthesized narratively to provide an overview of the genetic–nutrient interaction and its implications for personalized nutrition and diabetes prevention.

3. Results and Discussion

Magnesium and Its Role in the Development of Type 2 Diabetes Mellitus

Magnesium is the fourth most abundant element in the human body (Ca^{2+} > K^{+} > Na^{+} > Mg^{2+}) and the second most prevalent intracellular cation after potassium. The total amount of Mg^{2+} in the body ranges from 20 to 28 g (Fiorentini et al., 2021). Magnesium is an essential electrolyte for living organisms, and a regular intake is required to prevent deficiency. The recommended daily intake for adults is approximately 350 mg/day (Kemenkes RI, 2019). Magnesium plays a vital role in regulating glucose metabolism (Shanshan Huang, Yanyan Ge, Yanli, Ning Ning Cui, Le Tan, Shu Guo, Shanshan Wang, Liping Hao, Geng Lei, 2023). It is primarily found within cells, where it acts as a counter-ion for ATP and energy-rich nucleic acids (Gröber et al., 2015).

The importance of magnesium in glycolytic pathways and mitochondrial ATP synthesis makes many glycolytic enzymes magnesium-dependent. Key enzymes such as hexokinase, phosphofructokinase, phosphoglycerate kinase, and pyruvate kinase require Mg^{2+} for optimal function, while aldolase and enolase depend on it for stability and activity. The liver is a major organ where Mg^{2+} regulates gluconeogenic enzymes, including glucose-6-phosphatase and phosphoenolpyruvate carboxykinase (Feng et al., 2020). Studies have shown that magnesium deficiency significantly reduces hepatic levels of glucose-6-phosphate, citrate, fumarate, and malate, indicating the essential role of magnesium balance in carbohydrate metabolism (Shigematsu et al., 2016).

Hypomagnesemia is typically defined as a serum magnesium concentration below 0.75 mmol/L (Elin, 2010). Magnesium deficiency has been associated with T2DM, metabolic syndrome, and insulin resistance. Research indicates that magnesium enhances glucose uptake and tolerance through two main mechanisms: stimulating GLUT4 gene expression and translocation, and suppressing glucagon effects and gluconeogenic pathways in the liver and muscles (Sohrabipour et al., 2018).

Normal intracellular Mg^{2+} concentration is crucial for optimal insulin secretion. In pancreatic β-cells, glucose metabolism begins with the conversion of glucose to glucose-6-phosphate by glucokinase, generating ATP. Magnesium deficiency reduces intracellular ATP and MgATP levels, impairing β-cell electrical signaling and disrupting normal insulin release (Fiorentini et al., 2021). Several factors can negatively affect magnesium balance, leading to hypomagnesemia, β-cell dysfunction, impaired insulin signaling, chronic inflammation, and altered activity of Mg^{2+}-dependent kinases and metabolic enzymes. Collectively, these disturbances contribute to insulin resistance and the pathogenesis of T2DM (Kostov, 2019).

Magnesium is primarily absorbed in the small intestine and to a lesser extent in the large intestine. Two transport systems are known: passive paracellular transport and active transcellular transport via specific magnesium channels and transporters, including solute carrier family 41 member 1 (SLC41A1), magnesium transporter 1 (MagT1), and transient receptor potential melastatin type 6 and 7 (TRPM6 and TRPM7) (Fiorentini et al., 2021).

TRPM6 Gene

The TRPM6 gene (Transient Receptor Potential Cation Channel Subfamily Melastatin member 6) encodes a protein containing both an ion channel domain and a protein kinase domain. This gene is expressed in the kidney and the large intestine. The TRPM6 protein consists of 2,022 amino acids with a calculated molecular mass of approximately 234 kDa. The TRPM6 gene comprises 39 exons spanning a region of 166 kb. Its cytogenetic location is 9q21.13, situated on the long arm (q) of chromosome 9, at position 21, band 13. Mutations in this gene are associated with hypomagnesemia and hypocalcemia (Alina, 2021).

The TRPM6 gene plays a crucial role in epithelial magnesium transport and active magnesium absorption in the intestine and kidney. The normal function of *TRPM6* provides instructions for producing a protein that acts as a channel, allowing magnesium ions (Mg^{2+}) to flow into cells; this channel also permits the passage of a small amount of calcium ions (Ca^{2+}). The TRPM6 rs2274924 variant has been linked to metabolic syndrome in patients with type 2 diabetes mellitus. Advanced nutrigenomic testing has shown that TRPM6 rs2274924 (T/C) influences intestinal magnesium absorption and renal reabsorption. Women carrying the T/C haplotype have a higher risk of developing T2DM when their magnesium intake is below 250 mg/day. Other studies also indicate that two SNPs in TRPM6, rs3750425 and rs2274924, may increase susceptibility to T2DM among women with low magnesium intake (Alina, 2021).

TRPM6 Polymorphism

The *TRPM6* polymorphism (SNP: pLys1584Glu) is associated with an increased risk of diabetes mellitus. Haplotype analysis revealed a significant association between type 2 diabetes risk and carriers of the rare nonsynonymous SNP allele in *TRPM6* (Lys1584Glu in exon 27 [rs2274924]) when magnesium intake was below 250 mg per day. Compared with non-carriers, women carrying the 1393Ile–1584Glu haplotype showed an elevated risk of developing T2DM when their magnesium intake was low (<250 mg/day). Two distinct coding region variants of TRPM6, Ile1393Val and Lys1584Glu, may contribute to T2DM susceptibility in women with inadequate magnesium intake (Alina, 2021).

Hypomagnesemia affects insulin resistance and serves as a risk factor for type 2 diabetes mellitus. A study investigating the influence of TRPM6 on women with T2DM reported that those who were homozygous for both rare alleles of two nonsynonymous SNPs in TRPM6 and had low magnesium intake exhibited a substantially higher risk of T2DM. Approximately 33% of subjects were heterozygous for the rare G allele of rs2274924 and 18% were heterozygous for the A allele of rs3750425. Both SNPs are commonly found across populations with diverse genetic backgrounds. Individuals carrying both rare alleles, whose TRPM6 channels show reduced magnesium absorption, may be more prone to magnesium deficiency when dietary intake is low, thereby increasing diabetes risk. High magnesium concentrations in the intestinal lumen may compensate for the genetic defect in magnesium absorption by enhancing passive paracellular transport. Conversely, when dietary magnesium intake is insufficient, TRPM6 function in active intestinal magnesium absorption and renal reabsorption becomes critically important (Song et al., 2009).

Magnesium Intake Recommendations

Magnesium-rich foods are essential to maintain normal magnesium levels in the body and to prevent deficiency. Drinking water, especially hard water rich in minerals, provides an important additional source of Mg^{2+} salts and can serve as an alternative oral source of magnesium (Galan et al., 2002). Water contributes approximately 10% of total daily magnesium intake, while chlorophyll (and green vegetables such as spinach) is the main dietary source of magnesium. Nuts, seeds, and unrefined cereals are also rich in magnesium. Moderate concentrations of magnesium are found in legumes, fruits, fish, and meat. Certain food processing methods, such as grain refining that removes nutrient-rich germ and bran, significantly reduce magnesium content. Low magnesium concentrations are typically found in dairy products, except in fresh milk (Gröber et al., 2015).

Several dietary patterns can be applied to address hypomagnesemia. The Mediterranean diet emphasizes the consumption of whole grains, fruits, and vegetables in large portions, while fish, poultry, eggs, dairy products, meat, and sweets are consumed in smaller quantities. This diet is rich in Mg^{2+}, dietary fiber, antioxidants, and polyphenolic compounds (Park et al., 2017). However, in Indonesia, the Mediterranean diet remains difficult to implement due to the inclusion of wine as a common component.

The DASH (Dietary Approach to Stop Hypertension) diet is another dietary pattern suitable for individuals with diabetes. In addition to improving blood pressure control, the DASH diet has been shown to enhance insulin sensitivity, reduce hyperlipidemia, and help manage overweight and obesity. It provides higher levels of Mg^{2+}, K^{+}, Ca^{2+}, dietary fiber, and protein, with lower amounts of total saturated fat and cholesterol compared with conventional diets (Campbell, 2017).

Many nutrition experts suggest that ideal magnesium intake should be based on body weight (approximately 4–6 mg/kg/day). Magnesium supplements are available in various forms, including magnesium oxide, magnesium chloride, magnesium citrate, magnesium taurinate, magnesium orotate, and other amino acid chelates (Kisters, 2013).

The following studies summarize the effects of oral Mg^{2+} supplementation on patients with type 2 diabetes mellitus and overweight non-diabetic subjects:

Table 1. Magnesium Intake Recommendations

Magnesium Intake (mg/day)	Result	References
365 mg/day for 6 months	Mg^{2+} supplementation significantly improved fasting plasma glucose and insulin sensitivity in overweight, non-diabetic normomagnesemic subjects.	(Mooren et al., 2011)
300 mg/day for 3 months	Oral Mg^{2+} supplementation had beneficial effects on blood glucose, lipid profile, and blood pressure in patients with type 2 diabetes mellitus.	(Solati et al., 2014)
250 mg/day for 3 months	Oral Mg^{2+} supplementation reduced insulin resistance and improved glycemic control in patients with type 2 diabetes mellitus.	(ELDerawi et al., 2018)

5. Conclusion

The TRPM6 gene plays a vital role in maintaining magnesium homeostasis by regulating active absorption in the intestine and reabsorption in the kidney. Magnesium is an essential cofactor in glucose metabolism, insulin secretion, and cellular energy production. Deficiency in magnesium disrupts these processes, leading to impaired insulin signaling, systemic inflammation, and an increased risk of type 2 diabetes mellitus. Genetic variations in TRPM6, particularly rs2274924, have been associated with higher susceptibility to T2DM.

Adequate dietary magnesium, derived from green vegetables, nuts, seeds, whole grains, and mineral-rich water, can mitigate the adverse effects of these genetic polymorphisms and improve metabolic outcomes. Diets such as the Mediterranean and DASH patterns offer practical approaches to achieving optimal magnesium intake. Personalized nutrition strategies that consider TRPM6 polymorphisms and magnesium status may provide an effective means to prevent or delay the onset of T2DM and improve overall metabolic health. Future studies should focus on cohort research in Southeast Asian populations, genotype-based magnesium interventions, and molecular validation of the rs2274924 variant to clarify its functional impact on TRPM6. Additionally, multi-omics approaches and gender analyses are needed to strengthen the evidence base for nutrigenetic screening and personalized diabetes prevention.

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