The Review Of
DIABETIC
STUDIES OPEN ACCESS

Association Between Serum Magnesium Levels And Glycemic Control (Hba1c) In Obese Patients With Type 2 Diabetes Mellitus: A Narrative Review

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Submission: 15/09/2025 **Accepted: 30/09/2025** **Published: 10/10/2025**

Abstract
Magnesium acts as a cofactor for glucose transport, insulin signaling, and inflammatory pathways. Hypomagnesemia is commonly identified in individuals with type 2 diabetes mellitus (T2DM). We performed a narrative review to evaluate literature published between 2019 and 2025 examining associations between serum magnesium and measures of glycemic control (HbA1c), as well as oral magnesium supplementation interventions in obese adults with T2DM. Observational studies (predominantly cross-sectional and real-world data) demonstrate low serum magnesium to be associated with elevated HbA1c across heterogeneous cohorts, though studies vary in definitions of hypomagnesemia, measures of adiposity used, and covariates adjusted for (Kocyigit et al., 2023; Erinc et al., 2025). Analyses restricted to patients with obesity further demonstrate associations between obesity and abdominal obesity with low serum magnesium in individuals with T2DM; this may identify a susceptibility phenotype in which adiposity-driven insulin resistance contributes to magnesium dysregulation and poor glycemic control (Xu et al., 2024). Determinant analyses have also described both obesity and poor glycemic control as common correlates of hypomagnesemia in those with T2DM (Abdullah et al., 2025). Interventional evidence assessing the impact of oral magnesium supplementation on glycemic indices in T2DM from meta-analyses suggest improvements in glycemic parameters (including HbA1c), however, few studies are stratified by obesity status and there is high heterogeneity between trials (Asbaghi et al., 2022; Xu et al., 2023). In summary, recent literature continues to find an inverse association between serum magnesium and HbA1c in T2DM. There remains a need for obesity-stratified longitudinal studies and pragmatic trials adjusting for renal function, medications, and dietary intake.

Keywords: Serum magnesium; HbA1c; Type 2 diabetes mellitus; Obesity; Hypomagnesemia; Insulin resistance; Glycemic control.

Introduction

Type 2 diabetes mellitus (T2DM) is a major driver of morbidity worldwide. With complex interactions between insulin resistance, progressive β-cell failure, and cardiometabolic comorbidities, diabetes prevalence continues to grow. Surveillance studies continue to find that diabetes affects a large proportion of adults worldwide and that many cases of diabetes remain undiagnosed (IDF, 2025).

Diabetes prevention, early detection, and improved long-term metabolic control are thus major clinical imperatives (IDF, 2025). Clinicians currently assess glycemic control using measures of glycated haemoglobin (HbA1c), which approximates mean glucose exposure over prior weeks to months and is incorporated into modern standards of diagnosis, risk assessment, and treatment target monitoring (American Diabetes Association Professional Practice Committee, 2025). HbA1c levels are also meaningful as persistent hyperglycemia hastens oxidative stress and inflammation, pathophysiological processes driving microvascular and macrovascular complications of T2DM (Caturano et al., 2023; Weinberg Sibony et al., 2024).

Obesity and overweight are inherently linked to the onset and development of T2DM. For adults, obesity is defined as having a BMI ≥30 kg/m2 (World Health Organization, 2025). Additionally, obesity has recently been described as a chronic relapsing disease associated with dysfunctional adipose tissue leading to ectopic fat accumulation, systemic inflammation, and altered endocrine and immune signaling that promotes insulin resistance and metabolic inflexibility. Due to obesity's role in driving dysglycaemia and cardiometabolic dysfunction, major practice guidelines now recognize weight management as a key component to the prevention and management of T2DM alongside pharmacologic and lifestyle interventions (American Diabetes Association Professional Practice Committee, 2025; IDF, 2025). In fact, improvements to glycemic control in obese adults with T2DM have been difficult to sustain without a multidisciplinary approach to care that includes improving diet quality, energy intake, physical activity levels, and pharmacologic therapy. The phenotype of obesity may also impact micronutrient levels due to dietary intake, systemic inflammation, changes in volume of distribution, gastrointestinal absorption, renal excretion, and medication interactions which can impact glycemic control.

Micronutrients have recently been re-emerging as a modifiable factor influencing glucose homeostasis and cardiometabolic health. While micronutrients should not be considered replacements for medications with evidence for glucose lowering, nutrient status is of clinical importance given that deficiencies can worsen insulin resistance, oxidative stress, immune dysfunction, and vascular injury - especially among patient populations who have higher chronic disease burden and polypharmacy. Recent reviews looking at the evidence behind nutrient supplementation on glycemic outcomes report mixed results across micronutrients and study designs, though there seems to be greater benefit where there is a pre-existing deficiency or the intervention is addressing a plausible mechanism (Fong et al., 2022). With respect to this, magnesium stands out as one of the most biologically plausible and clinically practical minerals in T2DM given its involvement in over 300 enzymatic reactions including cellular energetics, and insulin signaling.

Magnesium is the second most prevalent intracellular cation and plays a critical role in a wide variety of physiological processes such as ATP-dependent reactions, neuromuscular transmission, and cardiovascular function (Adomako et al., 2024). With respect to metabolism, magnesium aids in insulin receptor activity and signaling cascades, modulates glucose transport, and plays a role in intracellular calcium homeostasis which directly impacts insulin secretion and sensitivity (Soriano-Pérez et al., 2022; Cazzola et al., 2024). The pathways by which magnesium may influence insulin resistance include downstream effects on insulin sensitivity, oxidative stress, and pro-inflammatory signaling. Notably, these mechanisms are already believed to be upregulated in states of obesity and T2DM due to chronic inflammation and ROS elevation (Caturano et al., 2023; Cazzola et al., 2024). Thus, there is substantial mechanistic rationale to suspect that low magnesium status would be associated with poor glycemic control, clinically measured as high HbA1c.

Clinically, hypomagnesemia is prevalent in T2DM and thought to have bidirectional relationship with diabetes pathophysiology (Adomako et al., 2024). Abnormalities in renal physiology in diabetes and insulin resistance have been shown to increase urinary magnesium wasting and decrease renal magnesium reabsorption, and magnesium deficiency has been shown to increase insulin resistance and impair glucose control forming a vicious cycle (Soriano-Pérez et al., 2022). Drug exposures also impact magnesium status. Diabetes drugs including metformin have been shown to associate with lower magnesium levels using observational cohort research designs, potentially due to gastrointestinal loss and changes to renal transporters (Chen et al., 2024; Xu et al., 2025). Metformin is the primary therapy for most adults diagnosed with T2DM; thus drug-induced changes in magnesium could act as confounders to the association between serum magnesium levels and HbA1c if they are not accounted for. Comorbid kidney disease, diuretic usage, and many other illnesses commonly seen in obese

populations with T2DM also alter magnesium levels and should be considered when interpreting serum magnesium (Adomako et al., 2024).

Observational data published between 2019 and 2025 has bolstered this signal. Associations between low serum magnesium and poor glycemic indices have been reported. Observational studies assessing dietary magnesium intake alongside serum magnesium concentrations have described inverse associations between serum magnesium and HbA1c specifically in adults with T2DM, indicating that low serum magnesium appears to be concurrently present with poorer glycemic control over time (Kocyigit et al., 2023). Notably for purposes of this review, this relationship has recently been shown to be present in the context of obesity as well. A novel real-world study published in patients with T2DM found an inverse relationship between serum magnesium and both obesity and abdominal obesity specifically, indicating that magnesium status may be related to adiposity patterns in a manner that impacts glycemic control and risk for complications (Xu et al., 2024). Other recent clinical studies have echoed these findings, characterizing hypomagnesemia as common in patients with T2DM and describing poor glycemic control as one of several correlates found with low serum magnesium levels (Abdullah et al., 2025). These observations do not establish causality but further support magnesium assessment in high-risk metabolic patients, particularly those with concomitant obesity.

Evidence has also emerged from interventions. Oral magnesium supplementation has been shown to improve glycemic outcomes among those with T2DM, including HbA1c in some systematic reviews/pooled analyses of randomized controlled trials, though results have been inconsistent when accounting for baseline magnesium status, formulation, dose and duration of trial, and study population characteristics (Asbaghi et al., 2022; Xu et al., 2023). It is important to note that conclusions have not always been stratified by obesity status, and heterogeneity between trials evaluating magnesium supplementation has been identified as a limitation for applying signals towards precision recommendations for supplementation in obese adults with T2DM. This is an important consideration given that obese versus non-obese populations with T2DM may systematically differ in factors that affect insulin resistance severity, inflammation, medication intensity, diet quality, renal function etc. that could impact magnesium status and its association with HbA1c.

Based on these gaps and challenges, there is need for a more streamlined review of the literature. While many studies now point to an association between serum magnesium and HbA1c, research is limited by being published across multiple study designs using different cut-offs for hypomagnesemia as well as adjustment for confounders like kidney function, pharmacotherapy, dietary Mg intake, and measures of obesity. Additionally, studies adjust for obesity instead of stratifying by obesity as it is often the clinical presentation in which researchers and clinicians are focused on because obesity can also significantly impact Mg levels and glucose metabolism. Thus, the purpose of this narrative review is to compile and discuss literature from 2019-2025 exploring the relationship between serum magnesium and glycemic control (HbA1c) specifically among obese individuals living with T2DM. This review will contextualize both mechanistic hypotheses and clinical/epidemiological evidence to summarize what is currently known, point out existing limitations, and emphasize directions for obesity-stratified longitudinal studies and pragmatics trials to consider for clinical monitoring and potential future Mg supplementation.

Literature Search Strategy

Databases and sources

A further search was conducted between 1 January 2019 and 31 December 2025 to identify literature investigating associations between serum magnesium levels and glycemic control (HbA1c) in obese individuals with T2DM. MEDLINE/PubMed, Embase, Scopus, Web of Science Core Collection, and Cochrane Library databases were searched. Google Scholar was used to identify any extra records not discovered through database searching (i.e., gray literature such as early online releases). The first few pages of articles were screened (sorted by relevance). Reference lists of included articles and pertinent reviews were manually searched.

Inclusion criteria

Studies were included if they met all the following criteria: Population: Adults (≥18 years) with T2DM which clearly defined participants as obese (i.e. BMI criteria, measures of central obesity, or author's

definition stating that participants were obese). Exposure: Reported serum magnesium (or indirect measure of circulating magnesium that explicitly stated that it was derived from serum/plasma). Outcome: Reported HbA1c and assessed its association with serum magnesium (correlation/association). Publication type: Peer-reviewed original research articles were included; systematic reviews/meta-analyses were used to contextualize findings and identify additional primary studies.

Study design: Observational studies (cross-sectional, case-control, cohort) and clinical trials were included if they allowed evaluation of the association between serum magnesium and HbA1c levels among obese adults with T2DM (data reported either as obesity-stratified or obesity-only analyses).

Publication type: Peer-reviewed original research articles and relevant systematic reviews/meta-analyses were used to contextualize findings and identify additional primary studies.

Exclusion criteria

Studies were excluded if they met any of the following criteria:

- Included populations with type 1 diabetes, gestational diabetes, pediatric/adolescent samples, or were animal/in vitro studies.
- Reported neither serum magnesium and HbA1c data (or data not presenting analyzable association information between these two variables).
- Included mixed BMI populations without separate result reporting for obese T2DM subjects and data that could not be extracted from authors.
- Were case reports, editorials, commentaries, conference abstracts (without full-text), protocols, or non-peer-reviewed sources.
- We're not available in their entirety or did not provide sufficient methodological detail to understand the exposure–outcome relationship.

Physiological Role of Magnesium in Glucose Metabolism

Magnesium is an essential intracellular divalent cation that serves as a cofactor in numerous reactions that have relevance to glucose homeostasis, including ATP-dependent phosphorylation reactions, ion channel activity, and maintenance of cellular redox potential. Dysregulation of magnesium levels is a common clinical feature of T2DM and has been associated with poor glycemic control leading some researchers to investigate magnesium levels as both a potential biomarker and modifiable driver of metabolic dysfunction (Soriano-Pérez et al., 2022; Oost et al., 2023). There are multiple biological mechanisms by which serum magnesium could be associated with HbA1c as magnesium deficiency can impact insulin secretion and sensitivity as well as inflammatory and oxidative stress pathways that contribute to insulin resistance.

Magnesium and insulin secretion

The tightly coupled metabolic and electrophysiological events that give rise to glucose-stimulated insulin secretion include the metabolic gating of ATP-sensitive potassium (KATP) channels. Metabolic signals such as the increase in intracellular ATP ([ATP]) and MgADP concentrations induce closure of KATP channels leading to membrane depolarization and opening of voltage-gated calcium channels (VGCCs). Calcium influx from VGCCs then triggers insulin granule exocytosis (Ashcroft, 2023). Magnesium-dependent processes are deeply involved in this pathway as magnesium complexes with adenine nucleotides. Mg is a physiological gating ligand of the KATP channel. Therefore, Mg^{2+} concentrations are connected to changes in cell metabolism and β-cell electrical activity and insulin secretion (Ashcroft, 2023). Deficient magnesium status may impair glucose sensing and insulin secretion from β-cells by altering nucleotide metabolism and potassium channel activity.

Magnesium also modulates pathways related to β-cell metabolism and insulin secretion capabilities. One recent mechanistic review that compiled experimental and clinical data concluded that Mg^{2+} deficiency is linked to decreased β-cell function and may play a role in β-cell KATP channel dysregulation leading to impaired insulin secretion (Akimbekov et al. 2024). Mg^{2+} serves as a cofactor for many reactions so it also stands to reason that Mg^{2+} would be needed for optimal glucose metabolism within β-cells including steps regulating glucose phosphorylation and flux leading to the

rise in ATP that triggers insulin secretion (Akimbekov et al. 2024). Obese adults with T2DM have chronically overworked β-cells due to higher insulin needs. Insufficient Mg2+ may further these cells' inability to cope and keep blood glucose regulated.

Magnesium and insulin sensitivity

Insulin sensitivity is dependent on adequate insulin receptor activation and downstream signaling events that allow for glucose uptake and inhibit glucose release by the liver. Insulin receptor signaling requires phosphorylation steps, and Mg-ATP is required for kinase activity. Reviews regarding magnesium and T2DM demonstrate that hypomagnesemia affects tyrosine kinase activity of the insulin receptor and downstream events decrease glucose utilization by cells leading to insulin resistance (Akimbekov et al. 2024; Soriano-Pérez et al. 2022). Skeletal muscle and adipose tissue are significant tissues of insulin-mediated glucose disposal and are frequently insulin resistant in the setting of obesity.

Clinical and translational reviews have noted the cyclical relationship between hypomagnesemia and insulin resistance. Epidemiologically, hypomagnesemia has been shown to be significantly more prevalent among those with T2DM and is associated with insulin resistance that promotes poor glycemic control among diabetics (Oost et al., 2023). Low magnesium levels may promote insulin resistance through urinary magnesium wasting. Insulin resistance may lead to low serum magnesium levels by causing urinary magnesium wasting. This reciprocal relationship makes it difficult to parse out using observational data (Soriano-Pérez et al., 2022; Oost et al., 2023). The cycle may be exacerbated by obesity, which is typically associated with higher insulin resistance. Obesity is also associated with dysfunctional adipose tissue that leads to various hormonal and inflammatory abnormalities that affect mineral metabolism.

Magnesium's effects on tissues affected by insulin, including roles in cellular ion transport, membrane stabilization, and energy metabolism may play a role at the tissue level. Summary evidence presented in a review of therapeutic considerations of magnesium on insulin secretion and resistance notes that normalization of magnesium levels promotes insulin signaling and glucose metabolism in insulin target tissues (including liver and muscle tissue) and that magnesium deficiency may lead to post-receptor defects in insulin action (Dastgerdi et al., 2022). Serum magnesium is not a direct reflection of total body magnesium stores but may provide some insight into the noted association between lower magnesium concentrations and higher HbA1c observed in clinical populations.

Magnesium, inflammation, and adipose tissue dysfunction

Low-grade chronic inflammation is characteristic of obesity and a primary contributor to insulin resistance in T2DM. Magnesium status has also been increasingly discussed as a potential modifier of inflammatory signaling pathways. One review on magnesium deficiency provides a comprehensive look at inflammatory physiology, illustrating how low magnesium increases production of pro-inflammatory mediators such as cytokines. It also discusses how inflammatory activation can crosstalk with oxidative pathways, creating feed-forward loops that promote risk for chronic disease (Arancibia-Hernández et al., 2023). Notably, inflammatory signaling through pathways like NF-κB are repeatedly identified as contributors to insulin resistance. And, systematic reviews and evidence syntheses discuss how magnesium may affect inflammatory activation, specifically mentioning its ability to modulate NF-κB-related responses (Wu et al., 2025).

Immune cell infiltration and cytokine release are a part of how adipose tissue becomes enlarged and metabolically dysregulated in obese adults with T2DM. Inflammatory factors released by this tissue can act to interfere with insulin signaling. Within this milieu, low magnesium may exacerbate risk for inflammatory amplification through multiple mechanisms, including both direct immune modulation and indirect alterations to calcium homeostasis and stress signalling that promote cytokine production (Arancibia-Hernández et al., 2023). The previously mentioned relationships are clinically relevant because inflammation has been connected to insulin resistance as well as vascular complications of T2DM, and magnesium deficiency has been hypothesized to affect cardiometabolic risk more broadly in T2DM (Oost et al., 2023). Magnesium's relationship with inflammation may be one mechanism by which low serum magnesium levels are associated with poor glycemic control (as measured by HbA1c).

Magnesium and oxidative stress

An imbalance between the production of reactive oxygen species and antioxidant capacity leading to oxidative stress lies at the heart of insulin resistance and β-cell dysfunction pathophysiology. Increased oxidative stress because of magnesium deficiency has been hypothesized to occur via a multitude of interrelated pathways including mitochondrial dysfunction, aberrant calcium handling, and stimulation of neurohormonal cascades such as the renin-angiotensin-aldosterone system, which lead to increased production of reactive oxygen species (Arancibia-Hernández et al., 2023). Elevated reactive oxygen species can further promote dysfunction by inhibiting insulin signaling, aggravating endothelial dysfunction, and inducing β-cell damage (especially under chronic hyperglycemic conditions). Chronic inflammation and oxidative stress are closely linked; oxidative stress induces inflammatory transcription factors and inflammation leads to increased oxidative stress, thus creating a vicious cycle of metabolic stress (Arancibia-Hernández et al., 2023).

Evidence for use as a therapeutic intervention is somewhat mixed but still yields helpful signals. A meta-analysis published last year evaluating magnesium supplementation and biomarkers of interest found a statistically significant decrease in C-reactive protein (consistent with an anti-inflammatory benefit) but no significant changes in oxidative stress biomarkers, indicating that there may still be some uncertainty around the size and generalizability of magnesium's direct antioxidant effect under clinical conditions (Cepeda et al., 2025). This is relevant to this review's question because improvements to inflammatory tone may positively impact insulin sensitivity and subsequent glycemic control downstream, even if shifts in biomarkers of oxidative stress are inconsistent across the varied trials.

Altogether, magnesium's involvement in β-cell stimulus-secretion coupling, insulin receptor signaling, inflammatory signaling, and oxidative stress supports a physiologically plausible rationale behind the inverse association observed between magnesium status and HbA1c in obese individuals with T2DM (Soriano-Pérez et al., 2022; Akimbekov et al., 2024; Oost et al., 2023). This hypothesis also helps to explain why many confounders of observational associations measure disturbances in magnesium balance such as renal handling, medication usage, and diet should be carefully accounted for in future obesity-stratified clinical trials.

Serum Magnesium Status in Obese Patients with Type 2 Diabetes Mellitus

Serum magnesium disturbances have been noted consistently in adults with T2DM and obesity may represent a particularly susceptible subset due to an interplay of dietary habits, inflammation, degree of insulin resistance and comorbidity profile. In recent literature, low serum magnesium levels have been ubiquitously noted to be prevalent amongst those with T2DM; however, there is significant heterogeneity in the reported prevalence depending on the clinical environment, case-mix, and cut-off value (Waanders et al. 2020; Pitliya et al. 2024). This matters from a clinical perspective as small decreases in serum magnesium have been shown to align with worsening glycemic control and increased cardiometabolic risk, further supporting the need for context when evaluating serum magnesium levels in obese individuals with T2DM (Oost et al. 2023).

Prevalence of hypomagnesemia and heterogeneity across studies

Pooling data from recent observational studies (published 2019–2023) via systematic review and meta-analysis, a recent study estimated a pooled prevalence of hypomagnesemia of 32% in persons with T2DM and concluded that low serum magnesium concentrations are "far from rare" in routine diabetes populations (Pitliya et al., 2024). Prevalence estimates vary widely between cohorts, however. In one recent primary care cohort, hypomagnesemia prevalence was 9.6%, substantially lower than many previously published estimates. Authors attributed this finding to the cohort potentially being representative of an "unselected" primary-care population, as well as differences in patient characteristics and definitions used (Waanders et al., 2020). Conversely, a retrospective cross-sectional study of adults with T2DM in Yemen (resource-limited setting) defined hypomagnesemia as <1.6 mg/dL serum magnesium and found hypomagnesemia prevalence of 37.2% (Abdullah et al., 2025). Variations such as these are likely due to a combination of: (i) different magnesium cut-offs and laboratory methods, (ii) duration of and control on diabetes, (iii) comorbidity profiles that influence magnesium levels (renal disease, hypertension), and (iv) differences in nutrition security and dietary magnesium intake (Waanders et al., 2020; Abdullah et al., 2025).

In studies of diabetic populations, obesity has also been repeatedly linked with low magnesium status as a correlate or predictor. Obesity, defined by the authors as a BMI ≥25 kg/m², was independently

associated with hypomagnesemia in Yemeni patients with diabetes in multivariable analyses that also identified poor glycemic control and long duration of diabetes as independent correlates (Abdullah et al., 2025). Findings from a retrospective, large-scale "real-world" database analysis were also suggestive; serum magnesium levels were inversely associated with BMI and abdominal obesity in T2DM, and trends over BMI categories were suggestive that the relationship may be mediated in part by inflammatory burden (Xu et al., 2024). Though these studies cannot establish causality between obesity and magnesium status, they align with clinical observations that obese adults with T2DM frequently harbor many risk factors for hypomagnesemia.

Contributing factors to low serum magnesium in obese T2DM

Hypomagnesemia in T2DM should be viewed as multifactorial renal-driven process related to metabolic and inflammatory abnormalities, low dietary intake and medication exposures that are often exacerbated by obesity.

1) Renal magnesium wasting and hypomagnesuria

Renal magnesium wasting is an important process driving hypomagnesemia in T2DM. As reviewed and synthesized mechanistically here, hypomagnesuria (increased fractional excretion of magnesium) is widely believed to be the primary driver of hypomagnesemia in T2DM (Oost et al., 2023). Crucially, this is distinct from glucosuria. Very recent literature highlighted in Endocrine Reviews demonstrates that while prior reports had implicated urinary magnesium wasting on glucosuria, this hypothesis has been called into question by SGLT2 inhibitors: they cause glucosuria without necessarily causing magnesium wasting and may even increase serum magnesium levels (Oost et al., 2023).

Alternately, proposed mechanisms involve hyperfiltration/increased tubular flow (leading to diluted luminal magnesium concentrations) as well as impaired control of magnesium transporters due to impaired insulin signaling, which is reduced in insulin-resistant states like obesity-associated T2DM (Oost et al., 2023). Obese patients may therefore be more susceptible to chronic renal magnesium wasting due to more severe insulin resistance and diabetic kidney disease.

2) Poor glycemic control and metabolic severity

In multiple observational cohorts, hypomagnesemia has been found to be strongly associated with poor glycemic control, which may represent either a sequela of disease severity, a driver of insulin resistance, or both (Waanders et al., 2020; Abdullah et al., 2025). Abdullah et al. also found that hypomagnesemia was independently associated with poor glycemic control in their Yemen cohort and noted a synergistic risk relationship between obesity and poor glycemic control when both were present (Abdullah et al., 2025). Mechanistically, this clinical finding makes sense as higher HbA1c leads to increased osmotic diuresis, altered renal hemodynamics, and medication escalations which could all impact magnesium status.

3) Obesity-related inflammation and adipose tissue dysfunction

Obesity involves low-grade inflammation that exacerbates insulin resistance. In the observational real-world T2DM study investigating outcomes associated with obesity, serum magnesium concentrations displayed an inverse correlation with obesity and abdominal obesity. Statistical mediation analyses demonstrated the potential involvement of inflammatory markers, including CRP, as mediators (Xu et al., 2024). This supports larger mechanistic contexts where decreased magnesium status is associated with pro-inflammatory physiology and metabolic risk factors, likely establishing reciprocal feedback loops that perpetuate insulin resistance and dysglycaemia in obese T2DM patients (Oost et al., 2023; Piuri et al., 2021).

4) Dietary patterns and nutritional adequacy

Nutrition intake levels can vary among populations with T2DM. Reviews have indicated some studies show individuals with T2DM falling below recommended levels for magnesium intake while others have shown adequate intake. This is likely due to variation in nutritional intake by population, how intake is assessed, and general challenges with confounding in nutrition science (Oost et al., 2023). In obesity, consumption of a diet higher in ultra-processed foods and lower in magnesium dense foods such as whole grains, legumes, nuts, and green leafy vegetables could promote inadequate intake.

Additionally, micronutrient density can be further decreased in energy restriction if diet quality is not emphasized (Piuri et al., 2021; Oost et al., 2023). For these reasons, nutritional inadequacy could reasonably lead to low serum magnesium levels in obese individuals with T2DM if they live in food deserts or do not meet dietary recommendations.

5) Medication exposures and comorbidities

Polypharmacy occurs frequently with T2DM. Obese patients with T2DM may also have comorbid hypertension, dyslipidemia and cardiovascular disease which can warrant additional medications. Some medications may cause decreased magnesium levels through renal wasting effects or causing problems with intestinal absorption. According to a recent review by Endocrine Reviews, thiazide diuretics and proton pump inhibitors (PPIs) are among the most prescribed medications and have been postulated as possible contributors to hypomagnesemia, though cohort results have been mixed (Oost et al., 2023). Metformin usage has also been associated: mechanistically, metformin appears to affect magnesium transport (i.e. targeting TRPM6 pathways), and in a recent 2024 meta-analysis, usage of diabetic medications (especially metformin) was associated with increased odds of having hypomagnesemia (Oost et al., 2023; Chen et al., 2024). Because obese adults with T2DM may be on medications that affect magnesium levels and are also frequently taking multiple drugs to manage their weight and T2DM, as well as potentially having varying stages of kidney dysfunction, it is important to consider medication usage and comorbidities when assessing serum magnesium levels (Al Harasi et al., 2024; Oost et al., 2023).

Clinical interpretation considerations

Serum magnesium is inexpensive and easy to order clinically but it does not account for total body magnesium stores and may not accurately represent intracellular magnesium depletion, particularly among patients with chronic illnesses. However, given the epidemiologic consistency that hypomagnesemia is prevalent among patients with T2DM and that serum magnesium levels are lower among patients with obesity and abdominal obesity (Pitliya et al., 2024; Xu et al., 2024), serum magnesium levels are relevant to consider when caring for obese patients with T2DM. Contextualizing results with renal function, glucose control, dietary intake, and concomitant medications can help differentiate causes and allow clinicians to pick out who may benefit most from formal assessment and replacement (Oost et al., 2023; Chen et al., 2024).

Association Between Serum Magnesium Levels and HbA1c

Evidence from observational studies (2019–2025)

Serum magnesium concentrations across observational studies published since 2019 are, for the most part, inversely associated with HbA1c in adults with T2DM, suggesting that lower serum magnesium is associated with poorer glycemic control. Earlier studies within our selected timeframe, including a clinic-based study, have found lower serum magnesium in those with T2DM compared to controls and positive correlations between serum magnesium and HbA1c (Saeed et al., 2019), supporting earlier hypotheses that magnesium depletion is linked to dysglycaemia. Larger datasets and routine-care samples have since replicated the same directionality in the association. HbA1c was one variable inversely associated with serum magnesium following multivariable adjustment in a recent primary care cohort, indicating that the magnesium–glycaemia association is not merely due to case-mix differences (Waanders et al., 2020). This year, researchers assessed both serum magnesium concentrations and dietary magnesium intake and concluded serum magnesium was lower in those with T2DM and higher HbA1c levels (Kocyigit et al., 2023).

Several additional studies from 2024–2025 further support the robustness of this relationship, albeit with significant heterogeneity in methodology. One study from 2024 showed a negative association between serum magnesium and HbA1c and found a correlation between serum magnesium levels and odds of glycemic control (Luo et al., 2024). Another cross-sectional study from 2022 conducted specifically within primary-care and investigating the prevalence of hypomagnesaemia and its relationship with glycemic control and diabetic complications found that lower magnesium was associated with worse glycemic parameters, including HbA1c (full text) (Hamarshih et al., 2022). Lastly, a recent 2025 study investigating risk factors for hypomagnesemia in T2DM patients found poor

glycemic control and obesity to be among the most important factors associated with the development of hypomagnesemia, suggesting that low magnesium levels are commonly found alongside both higher BMI and poorer HbA1c values in clinical settings (Abdullah et al., 2025).

Interpretation of observational results should be critical. Although directionally consistent, there are several limitations that constrain inference: Cross-sectional design. In many studies magnesium and HbA1c values were obtained at the same time point. Due to this major limitation, causal inference is limited, and reverse causality is possible. In other words, poorly controlled diabetes increases urinary magnesium wasting and low magnesium exacerbates insulin resistance and poor glycemic control, so both pathways are possible (Oost et al., 2023).

Definition/measurement bias. Numerous studies have different definitions for hypomagnesemia and used only serum magnesium, which is just 1% of total body magnesium stores (Oost et al., 2023).

Confounding by kidney function and medications: Magnesium homeostasis is directly tied to kidney function, medications. Common diabetes medications can also be related to serum magnesium levels. Exposure to metformin and other medications assessed was associated with serum magnesium as well as HbA1c in a primary care cohort study (Waanders et al., 2020). Associations presented in the model are likely biased without strict adjustment for kidney function, diuretics, proton pump inhibitors, and intensity of glycemic medications (Oost et al., 2023). Evidence specific to obesity is limited: Obesity-specific research is increasing; however, many studies adjust for BMI as a covariate rather than looking at obese phenotypes of T2DM. Serum magnesium levels were inversely associated with BMI category and severity of abdominal obesity in patients with T2DM in a recent real-world evidence study, which helps justify obesity as an upstream driver or effect modifier (Xu et al., 2024). There are limited studies from 2019–2025 that directly evaluate if the association between magnesium and HbA1c is stronger, weaker, or perhaps mechanistically different in obese versus non-obese adults.

Evidence from interventional studies and meta-analyses

Evidence from interventions is primarily derived from RCTs examining oral magnesium supplementation or meta-analyses that pool trials with heterogeneous T2DM samples. Overall, meta-analytic results suggest small improvements in glycemic outcomes such as HbA1c with substantial heterogeneity across studies in baseline magnesium status, formulations, doses, and length of intervention. A dose–response meta-analysis of controlled clinical trials concluded that oral magnesium supplementation improved glycemic control in individuals with T2DM, with HbA1c being among the outcomes demonstrating improvements in pooled analyses. This study also concluded that current trials are not adequate to meet criteria necessary for guideline-level recommendations. (Asbaghi et al. 2022). Most recently, a pooled analysis of 24 RCTs reported statistically significant reductions in HbA1c with magnesium supplementation in T2DM patients overall (Xu et al. 2023). Other broad evidence syntheses have reported favorable effects of magnesium supplementation on parameters of glucose metabolism in diabetic or diabetes risk populations but often cite heterogeneity by population and study quality. (Veronese et al. 2021).

Discrepant trial evidence and potential reasons for discrepancy. Trials showing no benefit. One well-controlled randomized crossover trial assessed the impact of oral magnesium supplementation in people with insulin treated T2DM and documented low serum magnesium levels. While magnesium supplementation modestly elevated serum magnesium concentrations, it did not enhance insulin sensitivity, an upstream driver of glycemic control (Drenthen et al. 2024). Several characteristics of the literature can help understand how this null result from a high-quality trial can exist alongside meta-analytic evidence of benefit: 1) Disease severity/population chronology on therapy: Individuals requiring insulin for glycemic management often have more severe T2DM characterized by longstanding insulin resistance and β-cell failure, which may be less amenable to interventions like magnesium repletion (Drenthen et al. 2024). 2) Duration of trials and glycemic endpoints: Compared with the meta-analyses described above, some trials investigating magnesium and glycemic control are short in duration and may be underpowered to detect an effect on HbA1c, a metric that integrates glycemia over several months. Null results for insulin sensitivity or glucose tolerance in the short term could still exist with modest shifts in HbA1c in longer trials, or vice versa. 3) Heterogeneity of baseline magnesium deficiency state: Included trials within meta-analyses vary in the magnesium status of their samples at baseline. Trials that enriched for magnesium deficiency or patients with more inflammation may have driven the beneficial effects of magnesium supplementation on glycemic metrics, while trials

that enrolled people with marginal magnesium status diluted these effects (Asbaghi et al. 2022; Xu et al. 2023). 4) Formulation and heterogeneity of adherence to supplements: Magnesium supplements contain different salts that can alter magnesium bioavailability. Furthermore, differences in tolerability can impact adherence to supplements. Many pooled analyses do not account for these differences (Asbaghi et al. 2022).

Integrating observational and interventional evidence for obese T2DM

Interpretation of the totality of epidemiologic data can be summarized as observational studies demonstrating consistent associations in the inverse direction of serum magnesium–HbA1c levels, while intervention studies demonstrate some evidence for improvement but lack consistency in glycemic markers with magnesium supplementation. Reasons we can make a stronger argument for potential clinical relevance in obese adults with T2DM include: findings that support obesity and abdominal obesity as independent correlates of lower serum magnesium specifically in T2DM, highlighting the presence of an obese T2DM phenotype that may be at higher risk for magnesium abnormalities (Xu et al., 2024), and determinant studies have highlighted obesity and poor glycemic control as overlapping correlates of low serum magnesium levels (hypomagnesemia) (Abdullah et al., 2025). There remains a notable lack of obesity-stratified randomized controlled trial evidence and few studies that either isolate obese-only cohorts or assess if obesity alters the efficacy of treatment. Overall, literature from 2019–2025 has shown low serum magnesium and high HbA1c to be biologically plausible and often present together in T2DM. There remains a need for obesity-specific longitudinal cohorts and well-designed pragmatic trials that account for renal function, medications, and dietary magnesium intake to determine causality and elucidate which populations of obesity would benefit from magnesium supplementation to improve glycemic control. (Oost et al., 2023; Asbaghi et al., 2022; Drenthen et al., 2024).

Clinical Implications for Nutrition Practice

Magnesium status is important in obese adults with T2DM due to the high prevalence of low serum magnesium in diabetes populations, as well as associations with poor glycemic control and cardiometabolic risk among these individuals (Pitliya et al., 2024; Oost et al., 2023). While observational studies showing associations between low magnesium status and poor health outcomes do not confirm causality and serum magnesium is not a reliable indicator of total-body magnesium stores, these findings support practical application for nutrition practitioners consisting of screening for magnesium inadequacy risk, personalized nutrition interventions to increase magnesium intake if needed, and careful integration of magnesium supplementation if warranted (Oost et al., 2023).

Screening and assessment in practice

Who to screen. Magnesium is not consistently included on routine laboratory panels. Due to high rates among those with T2DM and the clustering of poor glycemic control and obesity with hypomagnesemia, there is benefit to screening obese adults with T2DM who have persistent hyperglycemia despite optimized therapies, long duration of diabetes, GI losses, suspected low-quality diet, or are on multiple medications (eg. diuretics, proton pump inhibitors). (Oost et al., 2023; Abdullah et al., 2025). Consultation with clinicians is important for those with chronic kidney disease since renal health has major impacts on magnesium levels and patients can be hypo- or hypermagnesemic dependent on disease progression and treatments. (Oost et al., 2023).

How to assess. Serum magnesium is the most common, affordable test option; however, serum magnesium may be normal with intracellular deficiency. Clinicians should interpret laboratory findings with other factors such as dietary intake, symptoms of GI losses, renal health, and current medications instead of using one marker alone to define magnesium status. (Oost et al., 2023). Assessment of magnesium intake through dietary questionnaires is preferred when available. While overall diet quality tends to be low in those with obesity, there may be potential to improve magnesium intake through food frequency modifications in populations consuming high quantities of ultra-processed foods and low consumption of legumes, whole grains, and vegetables. (Piuri et al., 2021) Magnesium deficiency is operationally defined across studies and laboratories. Awareness of the hypomagnesemia cut-off used is important when comparing prevalence across datasets. (Pitliya et al., 2024; Abdullah et al., 2025).

Dietary intake strategies

Food-first approaches are recommended for nutrition practice, as magnesium-rich foods overlap with general cardiometabolic nutrition guidelines. Adequate magnesium intake may be recommended as part of dietary patterns that are already encouraged for managing T2DM and obesity risk (increased fiber intake, micronutrient density). Primary food sources of magnesium are:

- Legumes (beans, lentils, chickpeas)
- Nuts/seeds (almonds, pumpkin seeds, sesame seeds)
- Whole grains (oats, brown rice, whole wheat)
- Dark leafy greens (spinach, kale)

Other vegetables/fruits; some types of fish (ex. avocado; some fish contain small amounts).

Many of these foods overlap with weight loss or maintenance interventions due to higher satiety, more favorable lipid responses, and blood glucose control compared to refined carbohydrates (Piuri et al., 2021). When discussing options with overweight/obese adults with T2DM, practical swaps should be discussed (ex. whole grains in place of refined grains, legumes in place of or alongside rice, nuts/seeds as portion-controlled snacks) to ensure clients do not consume excess energy.

There are common roadblocks that should be considered when providing nutrition counseling to those with obesity and T2DM. These include appetite control, food availability, and cultural influences. Another consideration is that GI symptoms and dieting tend to lower micronutrient intake as well, so clinicians should emphasize nutrient density in energy-restricted diets.

Supplementation considerations

Optimizing magnesium intake through diet may have benefits for glycemic management. When supplementation may be considered. Magnesium supplementation, delivered orally, has been shown to improve glycemic outcomes in T2DM in pooled analyses of randomized controlled trials (RCTs), including HbA1c in some meta-analyses, however, effects are inconsistent and not generalizable to all patients (Asbaghi et al., 2022; Xu et al., 2023). In practice, clinicians may consider supplementation in obese adults with T2DM and documented low serum magnesium or other strong indicators of inadequacy (i.e. dietary assessment suggests difficulty achieving intake recommendations) when optimizing diet alone is unlikely to be feasible or effective. Clinicians should not assume, however, that supplementation will consistently reduce HbA1c across all patients. High-quality trial data have demonstrated null effects on measures of insulin sensitivity, such as HOMA-IR, in certain subgroups of patients, such as insulin-treated patients with low magnesium levels (Drenthen et al., 2024).

Individualization and safety. There are several important safety considerations that supplement decisions should account for, such as renal function. Impaired kidney function can limit magnesium excretion and lead to hypermagnesemia if supplemented in excess (Oost et al., 2023). Medication review is another crucial factor. Diabetes medication exposures appear to influence magnesium levels, with observational data suggesting metformin use in some contexts is associated with hypomagnesemia and certain common medications such as diuretics and PPIs can affect magnesium status (Oost et al., 2023; Chen et al., 2024). If medication-induced magnesium loss is suspected, it may be appropriate to work with other members of the medical team to identify opportunities to reduce dosing, switch medications, or increase monitoring.

Common Sense Method. The most reasonable approach I can provide based on a narrative-review style answer is:

Validate low/borderline serum magnesium levels, address factors involved (intake, renal function, drugs, GI losses), increase dietary consumption of magnesium as part of the dietary management of diabetes/metabolic syndrome, supplement if needed and monitor based on kidney function/response if levels are still low despite adequate intake.

Magnesium salts can have variable side effects such as diarrhea and abdominal pain which can impact patient compliance, so be sure to educate them on this as well (Asbaghi et al., 2022). Additionally, make sure supplementation doesn't replace improving their diet.

Monitoring and outcomes beyond HbA1c

Despite HbA1c being the most important clinical endpoint, magnesium optimization care should focus on other outcomes such as BP, inflammation, and patient-reported symptoms/outcomes. Magnesium supplementation has been shown to decrease inflammatory markers including C-reactive protein in

meta-analyses which shows there could still be beneficial effects on obesity related low-grade inflammation despite limited or mixed glycemic control results (Cepeda et al., 2025). It is important to note that heterogeneity among trials is high and evidence specifically stratified for those with obesity is limited, so clinicians should discuss magnesium optimization as part of diabetes nutrition care and not a singular solution (Oost et al., 2023; Xu et al., 2024).

In summary, current literature from 2019–2025 supports assessing magnesium status and optimizing magnesium levels through nutrition care interventions in obese adults with T2DM on an individualized basis by considering lab values, dietary intake, kidney health, and medication use.

Research Gaps and Future Directions

Despite a growing body of literature from 2019 to 2025 showing that lower serum magnesium is often associated with higher HbA1c in T2DM, several gaps limit clinical translation for obese adults.

1) BMI-stratified evidence remains scarce. Many observational studies report data across a mix of BMI groups and adjust for BMI rather than restricting cohorts to obese individuals or formally testing for effect modification by obesity phenotype. Emerging real-world evidence indicates serum magnesium concentrations are inversely associated with obesity-related measures, including abdominal obesity, among patients with T2DM (Xu et al., 2024). This suggests adiposity may modify magnesium–glycaemia associations. Future studies should stratify analyses by BMI class and central adiposity measures and report obesity-specific estimates of the magnesium–HbA1c association.

2) Unclear directionality of causality: Temporal ambiguity. Cross-sectional studies dominate our understanding of the magnesium and metabolic syndrome literature. As such, it is unclear whether low magnesium is causal or a consequence of worsening HbA1c because these studies cannot infer directionality (Oost et al., 2023). Does low magnesium contribute to worsening hyperglycemia over time OR do poor glycemic control/status drive renal magnesium waste and subsequent decreases in serum magnesium? Future investigations should be prospective cohorts with repeated measures of serum magnesium, HbA1c, renal function, and medications while adjusting for changes in medications and renal function to better understand temporality of associations.

3) Challenges with measurement and variable definitions used. Serum magnesium is only a marker of total body magnesium and represents a small amount of magnesium stored in the body. Additionally, serum magnesium may not be an adequate representation of intracellular magnesium stores, which can become depleted in the setting of chronic metabolic disease (Oost et al., 2023). Moreover, the definition of hypomagnesemia is not universal between investigations and clinical settings which has led to very heterogeneous prevalence estimates. We continue to see this even within recent cohorts reporting prevalence of hypomagnesemia (Waanders et al., 2020; Pitliya et al., 2024). Future studies should report assay methods, reference ranges, ionized vs. total magnesium (if available), and conduct sensitivity analyses utilizing well-established cut-offs.

4) Adjustment for kidney function and medications is typically incomplete. Renal processing is a keystone of magnesium homeostasis that is perturbed by T2DM, and drug exposure is among the largest reservoirs for potential confounding. Observational associations between magnesium concentrations and HbA1c are negative in primary care cohorts, for instance, and mirror usage of several glucose-lowering medications (Waanders et al., 2020). Systematic reviews discuss urinary magnesium wasting at length, along with pharmacologic sources that can bias serum measurements in diabetes (Oost et al., 2023). Ideally, future observational work will adjust for estimated glomerular filtration rate, albuminuria, diuretic and PPI use, and current glucose-lowering agents, and report if associations hold after these covariates are included in models.

5) Interventional data is inconsistent, and stratified trials based on obesity status are uncommon. Magnesium supplementation is consistently associated with improved glycemic indices across meta-analyses, such as HbA1c, but variability in formulations, doses, baseline deficiency states, and study durations challenge confidence in results (Asbaghi et al., 2022; Xu et al., 2023). Several high-quality trials also demonstrate null associations with insulin sensitivity within certain populations (e.g., insulin treated T2DM with low magnesium levels), indicating that benefits may not apply to all stages of disease (Drenthen et al., 2024). Future intervention trials should: (i) recruit obese adults specifically, (ii) stratify by baseline magnesium status, kidney function, and intensity of diabetes medications, (iii) allow for long enough duration to observe a change in HbA1c, and (iv) standardize adherence and gastrointestinal tolerability reporting.

6) Context of diet and lifestyle are inadequately described. Although a subset of studies assessed dietary magnesium intake and/or overall diet quality, most failed to do so which makes interpretation of low serum magnesium difficult. Was it due to inadequate intake? Altered absorption? Inflammation? Increased renal wasting? Prospective studies should include a validated measure of dietary intake in addition to biomarkers and explore if improving dietary magnesium intake through magnesium rich dietary patterns improves magnesium status and glycemic control (HbA1c) among obese adults with T2DM compared to supplementation alone.
7) Additional outcomes should be included beyond HbA1c and proposed mechanisms. Studies aiming to understand mechanisms should include markers of inflammation and oxidative stress, insulin resistance, and measures of magnesium absorption/utilization (i.e. urinary magnesium excretion) in addition to changes in HbA1c as evidence currently suggests the relationship between magnesium and insulin resistance is bidirectional and linked with CVD risk in T2DM (Oost et al., 2023). Taken together, if these recommendations are considered in future studies, it will allow us to better identify which obese adults with T2DM are at-risk for clinically significant magnesium deficiency and may benefit from dietary modification or magnesium supplementation.

Conclusion

Research published from 2019 to 2025 has shown that there is likely an inverse correlation between serum magnesium and HbA1c in individuals with T2DM as several observational studies noted that low serum magnesium levels were accompanied by poor glycemic control (Waanders et al., 2020; Kocyigit et al., 2023). Research also found that hypomagnesemia prevalence was high in individuals with T2DM worldwide, however; there was significant heterogeneity among studies depending on the setting and definition of hypomagnesemia used (Pitliya et al., 2024). A correlation that appears to be important to this review is that obesity and abdominal obesity have been associated with decreased serum magnesium levels in individuals with T2DM possibly indicating a phenotype where adiposity-driven insulin resistance and inflammation could further exaggerate magnesium abnormalities and poor glycemic control (Xu et al., 2024; Abdullah et al., 2025).
Intervention studies have also been conducted. While meta-analysis data has shown improvements to glycemic parameters such as HbA1c in pooled T2DM cohorts following oral magnesium supplementation, evidence is heterogeneous and not universal across all clinical subsets (Asbaghi et al., 2022; Xu et al., 2023). High-quality studies specifically conducted in those with late-stage T2DM or insulin requiring T2DM have shown supplementation to have no effect on insulin resistance (Drenthen et al., 2024). Overall, while magnesium status appears to be an important consideration during nutrition care provision for obese adults with T2DM, there is a need for stronger longitudinal and pragmatically-obesity focused research to establish causality, screening, and identification of when lifestyle modification or supplementation is most likely to beneficially impact HbA1c and cardiometabolic disease.

Acknowledgments

The authors would like to acknowledge University Putra Malaysia (UPM) for supporting this project.

Conflicts of Interest

The authors declare no conflict of interest.

References

1. Kocyigit, E., Akturk, M., & Koksal, E. (2023). Relationships between serum and dietary magnesium, calcium, and metabolic parameters in women with type 2 diabetes mellitus. Clinical Nutrition ESPEN, 54, 304-310.
2. Erinc, O., & Yesilyurt, S. (2025). Relationship between serum magnesium levels and glycemic control and insulin resistance. Northern Clinics of Istanbul, 12(2), 239.
3. Xu, M. R., Wang, A. P., Wang, Y. J., Lu, J. X., Shen, L., & Li, L. X. (2024). Serum magnesium levels are negatively associated with obesity and abdominal obesity in type 2 diabetes mellitus: a real-world study. Diabetes & Metabolism Journal, 48(6), 1147-1159.

4. Abdullah, M., Abdo, B., AlShoaibi, I., Alzanen, K., Alhakamy, M., Al-Namer, M., ... & Ahmed, F. (2025). Prevalence and determinants of hypomagnesemia in type 2 diabetes mellitus: A retrospective cross-sectional study from a resource-limited setting. Medicine, 104(32), e43851.
5. Asbaghi, O., Moradi, S., Kashkooli, S., Zobeiri, M., Nezamoleslami, S., Kermani, M. A. H., ... & Miraghajani, M. (2022). The effects of oral magnesium supplementation on glycaemic control in patients with type 2 diabetes: a systematic review and dose–response meta-analysis of controlled clinical trials. British Journal of Nutrition, 128(12), 2363-2372..
6. Xu, L., Li, X., Wang, X., & Xu, M. (2023). Effects of magnesium supplementation on improving hyperglycemia, hypercholesterolemia, and hypertension in type 2 diabetes: A pooled analysis of 24 randomized controlled trials. Frontiers in Nutrition, 9, 1020327.
7. International Diabetes Federation (IDF). IDF Diabetes Atlas. 11th ed. Brussels: International Diabetes Federation; 2025.
8. Caturano, A., D'Angelo, M., Mormone, A., Russo, V., Mollica, M. P., Salvatore, T., ... & Sasso, F. C. (2023). Oxidative stress in type 2 diabetes: impacts from pathogenesis to lifestyle modifications. Current Issues in Molecular Biology, 45(8), 6651-6666.
9. Weinberg Sibony, R., Segev, O., Dor, S., & Raz, I. (2024). Overview of oxidative stress and inflammation in diabetes. Journal of diabetes, 16(10), e70014.
10. World Health Organization. Obesity and overweight. WHO Fact Sheet; 2025.
11. Fong, C., Alesi, S., Mousa, A., Moran, L. J., Deed, G., Grant, S., ... & Ee, C. (2022). Efficacy and safety of nutrient supplements for glycaemic control and insulin resistance in type 2 diabetes: An umbrella review and hierarchical evidence synthesis. Nutrients, 14(11), 2295.
12. Adomako, E. A., & Alan, S. L. (2024). Magnesium disorders: core curriculum 2024. American Journal of Kidney Diseases, 83(6), 803-815.
13. Soriano-Pérez, L., Aranda-Rivera, A. K., Cruz-Gregorio, A., & Pedraza-Chaverri, J. (2022). Magnesium and type 2 diabetes mellitus: clinical and molecular mechanisms. Health Sciences Review, 4, 100043.
14. Cazzola, R., Della Porta, M., Piuri, G., & Maier, J. A. (2024). Magnesium: A defense line to mitigate inflammation and oxidative stress in adipose tissue. Antioxidants, 13(8), 893.
15. Chen, F., Mangano, K. M., Garelnabi, M., Cardaleen, K., & Tucker, K. L. (2024). Associations among diabetes medication use, serum magnesium, and insulin resistance in a cohort of older Puerto Rican adults. The American Journal of Clinical Nutrition, 119(6), 1523-1532.
16. Xu, L. H., Ye, L. T., Wang, J. Y., & Qiu, X. (2025). Magnesium-based alloys for bone regeneration and beyond: A review of advances and therapeutic prospects. Regenerative Therapy, 30, 977-983.
17. Oost LJ, Kurstjens S, de Baaij JHF, Tack CJ, Hoenderop JGJ. Hypomagnesemia and cardiovascular risk in type 2 diabetes. Endocrine Reviews. 2023;44(3):357–378. doi:10.1210/endrev/bnac028
18. Ashcroft, F. M., Lloyd, M., & Haythorne, E. A. (2023). Glucokinase activity in diabetes: too much of a good thing?. Trends in Endocrinology & Metabolism, 34(2), 119-130.
19. Akimbekov, N. S., Coban, S. O., Atfi, A., & Razzaque, M. S. (2024). The role of magnesium in pancreatic beta-cell function and homeostasis. Frontiers in Nutrition, 11, 1458700.
20. Dastgerdi, A. H., Rad, M. G., & Soltani, N. (2022). The therapeutic effects of magnesium in insulin secretion and insulin resistance. Advanced Biomedical Research, 11(1), 54.
21. Arancibia-Hernández, Y. L., Hernández-Cruz, E. Y., & Pedraza-Chaverri, J. (2023). Magnesium (Mg2+) deficiency, not well-recognized non-infectious pandemic: origin and consequence of chronic inflammatory and oxidative stress-associated diseases. Cell Physiol Biochem, 57(S1), 1-23.
22. Wu W, Gong M, Liu P, Yu H, Gao X, Zhao X. Hypomagnesemia: exploring its multifaceted health impacts and clinical applications. Diabetology & Metabolic Syndrome. 2025;17:217. doi:10.1186/s13098-025-01772-y
23. Cepeda V, Ródenas-Munar M, García S, Bouzas C, Tur JA. Unlocking the Power of Magnesium: A Systematic Review and Meta-Analysis Regarding Its Role in Oxidative Stress and Inflammation. Antioxidants (Basel). 2025 Jun 16;14(6):740. doi: 10.3390/antiox14060740. PMID: 40563371; PMCID: PMC12189353.
24. Waanders, F., Dullaart, R. P., Vos, M. J., Hendriks, S. H., van Goor, H., Bilo, H. J., & van Dijk, P. R. (2020). Hypomagnesaemia and its determinants in a contemporary primary care cohort of persons with type 2 diabetes. Endocrine, 67(1), 80-86.

25. Pitliya, A., Vasudevan, S. S., Batra, V., Patel, M. B., Desai, A., Nethagani, S., & Pitliya, A. (2024). Global prevalence of hypomagnesemia in type 2 diabetes mellitus–a comprehensive systematic review and meta-analysis of observational studies. Endocrine, 84(3), 842-851.
26. Piuri, G., Zocchi, M., Della Porta, M., Ficara, V., Manoni, M., Zuccotti, G. V., ... & Cazzola, R. (2021). Magnesium in obesity, metabolic syndrome, and type 2 diabetes. Nutrients, 13(2), 320.
27. Al Harasi, S., Al-Maqbali, J. S., Falhammar, H., Al-Mamari, A., Al Futisi, A., Al-Farqani, A., ... & Al Alawi, A. M. (2024). Prevalence of dysmagnesemia among patients with diabetes mellitus and the associated health outcomes: a cross-sectional study. Biomedicines, 12(5), 1068.
28. Saeed, H., Haj, S., & Qasim, B. (2019). Estimation of magnesium level in type 2 diabetes mellitus and its correlation with HbA1c level. Endocrinology, diabetes & metabolism, 2(1), e00048.
29. Luo, B., Pan, B., Zhao, G., Li, J., & Sun, L. (2024). Association between serum magnesium levels and glycemic control in type 2 diabetes. Diabetes, Metabolic Syndrome and Obesity, 2823-2829.
30. Hamarshih, M., Hamshari, S., Nazzal, Z., Snobar, F., Mletat, R., Abu-Mazen, O., & Maraqa, B. (2022). Hypomagnesemia and poor glycemic control among type 2 diabetic patients: a cross-sectional study. Indian Journal of Endocrinology and Metabolism, 26(6), 575-580.
31. Veronese, N., Dominguez, L. J., Pizzol, D., Demurtas, J., Smith, L., & Barbagallo, M. (2021). Oral magnesium supplementation for treating glucose metabolism parameters in people with or at risk of diabetes: a systematic review and meta-analysis of double-blind randomized controlled trials. Nutrients, 13(11), 4074.
32. Drenthen, L. C., Ajie, M., de Baaij, J. H., Tack, C. J., de Galan, B. E., & Stienstra, R. (2024). Magnesium supplementation modulates T-cell function in people with type 2 diabetes and low serum magnesium levels. The Journal of Clinical Endocrinology & Metabolism, 109(12), e2240-e2245.
33. "Introduction and Methodology: Standards of Care in Diabetes—2026." Diabetes care 49, no. Supplement_1 (2026): S1-S5.