Magnesium Deficiency

What Is Our Status?

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Low magnesium intake has been implicated in a broad range of cardiometabolic conditions, including diabetes, hypertension, and cardiovascular disease. Dietary magnesium and total body magnesium status are widely used but imperfect biomarkers in serum magnesium. Despite serum magnesium's limitations, it is nevertheless observed to be lower in those with cardiometabolic disease than in generally healthy people. Although some 50% of Americans do not meet recommended levels of magnesium intake, the extent of prevalent magnesium deficiency is unknown. Given magnesium's role in a multitude of chronic conditions that are increasingly common across the globe, here, we summarize recently published literature reporting prevalent hypomagnesaemia in generally representative populations and in populations with type 2 diabetes mellitus, metabolic syndrome, and obesity. On the basis of these studies, we estimate that up to a third of the general population may be magnesium deficient and that outside of acute clinical encounters or hospitalization, hypomagnesaemia is likely frequently overlooked in general clinical practice. Nutr Today. 2016;51(3):121–128

On Halloween 2014, Joe Queenan lamented in the *Wall Street Journal*'s op-ed pages the unsung heroics and underdog status of our favorite mineral, magnesium.[1] If anyone took notice—and let us hope that they did—magnesium may be poised to become the next nutrient superhero. "You keep hoping that someday your personal idols will have their day in the sun," he writes, and indeed, perhaps the time has come for magnesium to have its shining moment. Why? As Mr Queenan has correctly read, and duly points out to his readers, magnesium is among the world's most common minerals (eighth most abundant[2]), and yet, despite its ubiquity on Earth and in our food supply (it is at the center of the chlorophyll molecule), an estimated 50% of Americans have inadequate intake.[3] This latter point is particularly interesting given that, according to the same report, 36% of Americans manage to consume *over* the adequate intake level of calcium, magnesium's divalent cationic and much more lauded mineral sister.[3]

What does dietary deficiency tell us about the breadth of actual magnesium deficiency? Deficiency estimates are more difficult to come by, partly because magnesium is not routinely measured in many outpatient settings.[4] The most recent population-based report on biomarkers of nutrient status in the United States, *The Second National Report on Biochemical Indicators of Diet and Nutrition in the US Population, 2012*, makes no mention of magnesium in its 495 pages.[5] In fact, to our knowledge, the last national (US) estimates of average serum magnesium were based on the National Health and Nutrition Examination Survey I (1970–1974).[6] Using these data, authors generated a normative range of serum magnesium for healthy US adults (1.50–1.91 mEq/L for adults 18–74 years of age, equivalent to 0.75–0.96 mmol/L or 1.8–2.3 mg/dL).[6] Thus, since the early 1970s, there has been no measure of serum magnesium or estimated prevalent hypomagnesaemia in a nationally representative US population. However, various estimates published in the 1980s indicate that hypomagnesaemia could range from 11% of a random sample of hospitalized patients[7] to 61% of postoperative intensive care patients.[8] In a 1990 survey of 1033 routine electrolyte panel measurements in an urban primary care hospital, the prevalence of hypomagnesaemia was 47%. Notably, however, just 10% of the hypomagnesaemic cases were identified because a physician had specifically requested that magnesium be measured (ie, tested because of a suspected a magnesium imbalance).[9] Although hospital emergency, cardiac, and obstetrics departments are likely most familiar with magnesium (as magnesium-based intravenous therapies in myocardial infarction, stroke, asthma attack, and hypertension of pregnancy), magnesium also deserves greater attention

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in other clinical practices. In this review, we focus on recent literature on magnesium deficiency, iterating its relevance to very common conditions. To achieve this, we summarize studies published in the last 5 years that have reported the prevalence of hypomagnesaemia in their sample populations, both healthy and with cardiometabolic risk factors, such as obesity, type 2 diabetes mellitus (T2DM), and hypertension. We selected studies from recent literature with approximately 100 participants or more, appearing to reflect a typical patient panel of an outpatient clinical practice, or reflecting a general population.

A Word About Serum Magnesium

Total serum magnesium is the most commonly used clinical measure of magnesium status, and the one by which hypomagnesemia and hypermagnesemia are clinically identified. However, serum magnesium is not an ideal biomarker of magnesium status, or even magnesium intake.[10] The serum contains less than 1% of the body's magnesium, where it is very tightly homeostatically controlled, and thus does not reflect true magnesium status of the body. Clinical hypomagnesemia typically arises only after prolonged periods of deficiency (eg, induced by low intake or malabsorption) or high urinary excretion (eg, as induced by kidney disease, and diuretics). Although the gold standard test of magnesium status is the urinary magnesium load test, it is time consuming, expensive, and requires a magnesium infusion. Intracellular and serum ionized magnesium have been proposed as better blood-based indicators of total body magnesium, although these too are imperfect markers for a variety of reasons, not least because such laboratory measures are much less frequently available in clinical settings. Furthermore, intracellular magnesium is not as consistent a marker as is ionized magnesium.[10] However, low levels of both intracellular and serum ionized magnesium have been observed even in the presence of normal levels of total serum magnesium.[11] Therefore, it may be possible to identify depletion or increased risk of hypomagnesaemia earlier in populations known to be more likely to develop the condition (eg, those with prediabetes and T2DM) using measures other than total serum magnesium.[11] In addition, 24-hour urinary magnesium concentrations, which are somewhat more easily determined—although also not routinely clinically measured—also gives clues to healthcare providers as to the underlying cause of deficiency, possibly pointing to increased excretion or reduced absorption, although urinary magnesium is unreliable when kidney function is impaired.[10] Nevertheless, all measures of magnesium, including total serum magnesium, rely on cut points and assays that have not universally been agreed upon, thus perhaps obscuring the uniformity of prevalence estimates of hypomagnesaemia in various populations. As total serum magnesium continues to be the de facto biomarker, a widely used cut point for hypomagnesaemia is less than 0.7 mmol/L (1.7 mg/dL),[2] although a range of cut points from 0.65 to 0.80 mmol/L, as shown in the present article, is also evident across the literature.

Estimating Hypomagnesaemia in the General Population

Several recent studies have examined hypomagnesaemia in large populations derived from either regional or national surveys,[12–17] outpatient hospitals/clinics,[18,19] or large population-based cohort studies[20,21] (Table 1). These populations would seem to most closely resemble typical patient/client panels, representing healthy individuals as well as those with various comorbidities, including T2DM, obesity, and other cardiometabolic risk factors. Across these studies of large, representative populations, overall hypomagnesaemia prevalence ranged from 1.7%[16] up to 36%.[13]

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The most recently published national estimates of hypomagnesaemia emanate from Mexico's 2006 National Health and Nutrition Survey. Researchers studying various age groups estimated that among children 1 to 11 years of age, the prevalence of hypomagnesaemia was 22.6%[15]; among adolescents, it was 37.6%[12]; and among adult women and men, it was 36.3% and 31.0%, respectively.[13] Notably, 35.4% of the adolescents[12] and 70% of the adults were overweight or obese.[13] In stark contrast to Mexico, the overall prevalence of hypomagnesaemia was 1.7% in a regional household survey in rural Victoria, Australia, although there was notably higher prevalence in those with comorbidities (see specific morbidities sections below).[16] One clear difference between the Australian and Mexican populations was prevalent obesity: 27.7% of the Australian versus 70% of the Mexican adults were estimated to be obese.

Markovits et al used data from a large Israeli health maintenance organization database to retrospectively examine serum magnesium concentrations in 95 205 ambulatory patients with a mean age of 48 years and with various comorbidities (eg, 30.4% of the population was hypertensive, 13.3% had T2DM).[18] In this population, the overall prevalence of hypomagnesaemia was 6.0%. From the same world region, among the 1558 participants of the population-based Tehran Lipid and Glucose Study, hypomagnesaemia was prevalent in 4.6% of the population, with prevalence in women (6%) nearly double that of men (3.2%).[21]

In Germany, participants of the population-based Study of Health in Pomerania, serum magnesium concentrations in 24.9% of the participants were considered deficient, and women had a higher prevalence (27.9%) as compared with men (21.9%). The mean age of the population was 50 years and various comorbidities were present (eg, 52.4% of the population was hypertensive).[20]

A smaller study in 115 presumably healthy, mostly normal weight Brazilian university students indicated plasma hypomagnesaemia in 34%, erythrocyte hypomagnesaemia

TABLE 1 Recently Published Population-Based Studies Reporting Prevalence of Hypomagnesaemia

Study, Year, Country (Ref)	Population Description	Hypomagnesaemia Definition, as Given in Text	Hypomagnesaemia Prevalence
De la Cruz-Góngora, 2012, Mexico[12]	1972 adolescents, representing ~17 million adolescents from the National Health and Nutrition Survey (2006) Mean age: 15.1 y (range, 12–19 y) 54.1% girls 35.4% overweight/obese	Serum Mg <0.75 mmol/L (1.82 mg/dL)	Overall: 37.6% Women: 40.0% Men: 35.4%
Guerrero-Romero, 2013, Mexico[14]	427 adults from a population-based study Mean age: 41.5 ± 13.7 y 64.2% women Mean BMI: 29.9 ± 6.2 kg/m^2 MONW: n = 31 NW: n = 45 MHO: n = 98 Obese: n = 253	Serum Mg ≤0.7 mmol/L (1.7 mg/dL)	Overall: 37.7% NW: 17.8% MONW: 70.1% MHO: 10.2% Obese: 47.8%
Hermes Sales, 2014, Brazil[17]	115 nonobese, nonmalnourished, nonsmoking, non-athletic, disease-free university students Mean age: 22.5 ± 2.5 y (range, 19–29) 48% women Mean: BMI 22.5 ± 2.6 kg/m^2	Plasma Mg ≤0.75 mmol/L Erythrocyte Mg ≤1.65 mmol/L	Based on plasma Mg: 34% Based on erythrocyte Mg: 17% Based on both plasma and erythrocyte Mg: 8%
Markovits, 2014, Israel[18]	95 205 ambulatory patients in a large health maintenance organization database (2008–2011) Mean age: 48.1 ± 20.4 y 63.2% women 30.4% with hypertension 13.3% with T2D 23.6% using PPI 9.6% using diuretics	Serum Mg ≤0.7 mmol/L Moderate/severe: ≤0.6 mmol/L Severe: ≤0.55 mmol/L	Overall: 6.0% Overall moderate/severe: 0.9% Overall severe: 0.5% Women: 6.1% Men: 5.8% Diuretics users: 14.6% PPI users: 14.1% With hypertension: 11.7% With T2D: 23.0%
Mejía-Rodríguez, 2013, Mexico[13]	5410 adults representing ~59 million Mexican adults from the National Health and Nutrition Survey (2006) Age: ≥20 y 63.2% women 70% overweight/obese	Serum Mg <0.75 mmol/L (<1.82 mg/dL)	Women: 36.3% Men: 31.0%
Morales-Ruán, 2012, Mexico[15]	5,060 children, representing ~24 million children, from the National Health and Nutrition Survey (2006) Age range: 1–11 y 49.9% girls	Serum Mg <0.75 mmol/L	Overall: 22.6% 1–4–year-olds: 12.0% 5–11–year-olds: 28.4%

(continues)

TABLE 1 Recently Published Population-Based Studies Reporting Prevalence of Hypomagnesaemia, Continued

Study, Year, Country (Ref)	Population Description	Hypomagnesaemia Definition, as Given in Text	Hypomagnesaemia Prevalence
Reffelmann, 2011, Germany (West Pomerania)[20]	3,910 participants of the population-based Study of Health in Pomerania (1997–2001) Mean age ~50 y 50.8% female Mean BMI ~27 kg/m^2 52.4% with hypertension 10.2% calcium-antagonist users 4.8% β-blocker users 8.1% diuretics users 13.1% ACE-inhibitor users	Serum Mg ≤0.73 mmol/L (reference range of assay: 0.74–0.99 mmol/L)	Overall: 24.9% Men: 21.9% Women: 27.9% With hypertension: 24.4% Calcium-antagonist users: 29.1% β-blocker users: 32.4% Diuretics users: 33.3% ACE-inhibitor users: 29.3%
Simmons, 2010, Australia[16]	1453 participants in a regional household survey (2001–2003) Mean age: 53 ± 16 y 56.3% women 27.7% obese 9.5% with T2D 25.2% with metabolic syndrome	Serum Mg <0.70 mmol/L	Overall: 1.7% Control/normal: 0.9% With T2D: 10.5% With new T2D: 6.5% With IGT: 1.0% With IFG: 0% With treated hypertension: 5.2% With metabolic syndrome: 2.8%
Song, 2011, Korea[19]	949 adult patients visiting outpatient family medicine department at a university hospital (2007–2008) Mean age: 50.5 ± 14.8 y (range: 18–70) 46.1% women 14.9% history of diabetes	Serum Mg <0.78 mmol/L (1.9 mg/dL)	8.7%
Syedmoradi, 2011, Iran[21]	1,558 participants of the population-based Tehran Lipid and Glucose Study (2006–2007) Mean age: ~39 y 51.6% women Mean BMI: ~27.3 kg/m^2	Serum Mg <0.75 mmol/L Suboptimal <0.8 mmol/L	Overall: 4.6% Women: 6.0% Men: 3.2% Overall suboptimal: 14.6%
Wang, 2013, China (Shanghai)[25]	1902 participants in a follow-up study of the community-based Shanghai Diabetes Study I and II (2010–2011) Mean age: 57.4 ± 8.8 y 60.1% women NGR: n = 1,170 IGR: n = 389 T2D: n = 343	Serum Mg ≤0.65 mmol/L	With NGR: 0% With IGR: 0% With T2D: 0.6%

Conversion factor: 0.5 mEq/L or 0.411 mg/dL per 1 mmol/L.

Abbreviations: ACE, angiotensin-converting enzyme; BMI, body mass index; IFG, impaired fasting glucose; IGR, impaired glucose regulation; IGT, impaired glucose tolerance; Mg, magnesium; MHO, metabolically healthy obese; MONW, metabolically obese normal weight; NGR, normal glucose regulation; NW, normal weight without metabolic disturbances; PPI, proton pump inhibitor; T2D, type 2 diabetes.

in 17%, and deficiency in both in 8% of the population.[17] Authors speculated that inadequate magnesium intake was the primary cause, as they found deficient intake in more than 70% of the students. Finally, in a Korean outpatient family medicine department, among 949 patients with a mean age of about 50 years, 8.7% of the study population was hypomagnesaemic.[19]

Magnesium Status in T2DM

There is a long history of research in magnesium in T2DM and related conditions, including prediabetes and insulin resistance.[22] Recent contributions to the literature continue to report higher prevalence of hypomagnesaemia in individuals with T2DM. In the studies described above where hypomagnesaemia was reported

by condition, it was universally more prevalent in those with T2DM than in those without the condition. In the Australian household survey, for example, hypomagnesaemia was prevalent in 10.5% of those with T2DM and 6.5% of those with newly diagnosed T2DM, as compared to 0.9% of normoglycemic controls.[16] In Israel, of the 13.3% of participants reported to have T2DM, nearly a quarter were hypomagnesaemic.[19]

Several recent studies have also explicitly examined populations with prevalent T2DM (Table 2). Barbagallo et al measured serum total and serum ionized magnesium in 105 older Italian patients with recently diagnosed T2DM, and compared them with 100 age-matched controls, reporting that by serum magnesium, hypomagnesaemia prevalence was about 8% in those with T2DM and 2% in those without T2DM.[11] However, based on serum ionized magnesium, using the lowest value in the controls as a cut point, 44.8% of those with T2DM were magnesium deficient. As was mentioned earlier, and what is highlighted by this study, risk for clinical deficiency, which is typically assessed by serum magnesium, may be more obvious and detectable earlier when measured by magnesium compartments other than total serum, in this case, serum ionized magnesium.

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Peters et al recently compared hypomagnesaemia prevalence by T2DM treatment type in 940 patients with non–insulin-dependent T2DM enrolled in the Fremantle Diabetes Study.[25] In this study, the prevalence of hypomagnesaemia was 11.1% in those treated with diet alone, as compared with 41.3% in those treated with both Metformin and sulfonylurea. The obvious implication of this study is that the severity and duration of diabetes likely plays a role in magnesium concentrations and that it is possible that the treatment type may also be a factor, although the role of treatment clearly may be confounded by the severity and duration of diabetes in this observational study. Nevertheless, of note to clinicians is that the overall prevalence of hypomagnesaemia in the study population was 19%, or about 1 in 5.

Another recent study in an elderly sample with T2DM, this time, a Taiwanese population, reported that 37.1% of the 210 participants studied were hypomagnesaemic.[24] In contrast, Wang et al, examining 1,902 Chinese adults of the Shanghai Diabetes Studies with normal glucose response, impaired glucose response, or T2DM, observed that just 0.6% of the participants with T2DM had hypomagnesaemia, and none of those with normal or impaired glucose response had hypomagnesaemia.[27] However, the criteria for hypomagnesaemia in this study was 0.65 mmol/L or lower, about 0.05 to 0.1 mmol/L lower than most cut points in the studies presently reviewed, thereby perhaps underestimating prevalent deficiency. Indeed, another study conducted in a smaller sample of 137 Chinese adults diagnosed with T2DM and 50 healthy controls observed that those with T2DM had a prevalence of hypomagnesaemia of 35% (using a cut point of <0.75 mmol/L), as compared with 0% of controls. Furthermore, in those with T2DM accompanied by diabetic nephropathy, the prevalence reached 54%.[26] One obvious point illustrated by the abovementioned studies is that the cut point matters in determining deficiency prevalence. Were either author group to use the more typical 0.7 mmol/L cut point, prevalence estimates would have likely been more similar in the 2 sample populations groups.

Magnesium Status in Obesity and in Those with Metabolic Syndrome

In 2013, Guerrero-Romero and colleagues[14] measured serum magnesium in a population-based study of 427 adults in Mexico (Table 2). The overall prevalence of hypomagnesaemia was 37.7%. The authors categorized the participants according to their obesity status and their metabolic health, based on presence of 1 or more metabolic abnormalities (ie, fasting hyperglycemia, insulin resistance, hypertriglyceridemia, and/or hypertension). In the metabolically *un*healthy, 70.1% of those with normal weight were magnesium deficient, compared with 47.8% of those who were obese. In the metabolically healthy, 17.8% of those with normal weight were deficient, compared with 10.2% of those who were obese. Obviously, in this study, metabolic health, rather than obesity, played a key role in prevalent hypomagnesaemia.

Lima et al examined serum magnesium and intramononuclear magnesium in 72 patients with metabolic syndrome (72.2% obese) and 57 healthy nonobese individuals in Brazil. Using serum magnesium, 23.2% of those with metabolic syndrome were identified as hypomagnesaemic, compared with 3.3% of healthy controls. In addition, 36.1% of those with metabolic syndrome showed intramononuclear depletion, compared with 9.8% of controls. Notably, in those with metabolic syndrome, 13.4% had only low serum, 26.8% had only low intramononuclear, 10.2% had both low serum and low intramononuclear, and 49.2% had both concentrations at normal levels.[24] As with the comparisons between total serum and serum ionized magnesium performed by Barbagallo and colleagues among those with T2DM,[11] another compartment—intramononuclear

TABLE 2 Recently Published Large (N ≥ 80) Studies of Obese or Diabetic Populations Reporting Prevalence of Hypomagnesaemia

Study, Year, Country (Ref)	Population Description	Hypomagnesaemia Definition, as Given in Text	Hypomagnesaemia Prevalence
Barbagallo, 2014, Italy[11]	105 untreated, recently diagnosed, older T2D patients, 100 age-matched non-T2D controls With T2D Mean age: 71.1 ± 0.8 y Without T2D Mean age: 72.2 ± 0.8 y 58% women	Serum Mg <0.70 mmol/L Serum ionized Mg <0.47 mmol/L (lowest value in those without T2D)	Based on serum Mg With T2D: ~8% Without T2D: ~2% Based on ionized Mg With T2D: 44.8% Without T2D: 0%
Huang, 2012, Taiwan[23]	210 elderly T2D patients Mean age: 72.3 ± 5.4 y 53% women 7% obese (BMI >27 kg/m^2)	Serum Mg <0.75 mmol/L	37.1%
Lima, 2009, Brazil[24]	72 patients with metabolic syndrome, 57 healthy controls Mean age: 45.7 ± 11.8 y 91.7% women Mean BMI: 35.3 ± 7.3 kg/m^2 72.2% obese 77.8% with hypertension 45.1% with IFG 36.1% with hypertriglyceridemia 62.5% with low HDL level	Serum Mg <0.7 mmol/L (1.7 mg/dL) Intramononuclear Mg ≤0.76 μg/mg protein	With metabolic syndrome Serum Mg: 23.2% Intramononuclear Mg: 36.1% Control Serum Mg: 3.3% Intramononuclear Mg: 9.8%
Peters, 2013, Australia[25]	940 non-insulin-treated T2D patients Mean age 63.4 ± 11.6 y 51% female	Serum Mg <0.70 mmol/L	Overall: 19% Treated by diet only: 11.1% Treated by metformin only: 17.6% Treated by sufonylurea only: 14.7% Treated by metformin and sulfonylurea: 41.3%
Xu, 2013, China[26]	137 adults diagnosed with T2D Median age: 56 y (range 42–62) 38% women DN: n = 24 DPN: n = 50 Control: n = 50	Serum Mg <0.74 mmol/L (18 mg/L)	Control: 0% With T2D: 35% With T2D and DN: 54% With T2D and DPN: 36%

Abbreviations: BMI, body mass index; DN, diabetic nephropathy; DPN, diabetic peripheral neuropathy; HDL, high-density lipoprotein cholesterol; IFG, impaired fasting glucose; Mg, magnesium; T2D, type 2 diabetes.

magnesium—also revealed itself to be a perhaps better indicator of magnesium deficiency.

Although metabolic dysfunction may appear to be a more prominent factor than obesity in hypomagnesaemia, several studies looked at magnesium deficiency in the growing population of severely obese individuals, notably candidates for bariatric surgery, in which the mean body mass index of patients ranged from 45.8 to 55.7 kg/m^2 (Table 2). Preoperative magnesium deficiency ranged from 0.8% in Swiss patients[28] to 30.8% of a Spanish sample.[29] In a US study, 17.9% of patients were hypomagnesaemic,[30] whereas in a separate Spanish study, prevalence was 48% in patients who also had T2DM but 15% in those without T2DM.[31] Notably, in the latter study, the authors reported that serum magnesium concentrations only returned to normal in patients postoperatively when T2DM was also resolved, whereas when T2DM remained unresolved even after surgery, serum magnesium remained low. This supports the findings of Guerrero-Romero and colleagues,[14] noted above, who observed that hypomagnesaemia was more prevalent in the metabolically unhealthy, rather than in the obese, per se. Two of the bariatric studies[29,30] also reported 5-year postoperative hypomagnesaemia, a period through which both patient populations were prescribed

magnesium as a part of a multivitamin/mineral supplement: In the US study, 5-year postoperative prevalence was 0% among 30 of 82 baseline individuals with 5-year follow-up data,[30] whereas in the other study in a Spanish population, prevalence ranged from 5.8% to 12.5% in 159 of 355 baseline individuals with 5-year follow-up data.[29]

Hypomagnesaemia in Other Common Conditions and Treatments

Hypomagnesaemia has also been noted in a host of other common disorders, such as hypertension, as well as with treatment for these conditions.[4] Markovits et al, using data from a large health maintenance organization database in Israel, reported a hypertension prevalence of 30.4% and that 11.7% of those who were hypertensive were magnesium deficient (as compared with 6.0% of the overall population).[18] The authors also noted that the hypomagnesaemia was more prevalent in the 9.6% of participants who were users of diuretics (14.6% were hypomagnesaemic) and the 23.6% of participants who were users of proton pump inhibitors (PPIs) (14.1% were hypomagnesaemic). In addition, in a meta-analysis of PPI use and hypomagnesaemia, Park and colleagues reported on 9 studies in diverse populations, totaling 115 455 patients.[32] Hypomagnesaemia was more prevalent in PPI users (median prevalence 27.1%) than in nonusers (median prevalence 18.4%), although the authors found significant heterogeneity between studies.

Meanwhile, in the Study of Health in Pomerania, Germany, 52.4% of the study population was hypertensive, and of those, 24.4% were magnesium deficient.[20] Hypomagnesaemia was also highly prevalent in users of calcium antagonists (29.1%), β-blockers (32.4%), diuretics (33.3%), and angiotensin-converting enzyme inhibitors (29.3%), as compared with the overall population (24.9%).

Concluding Comments

Hypomagnesaemia is a common condition that may be frequently overlooked in nonacute clinical situations, as in T2DM, obesity, and related conditions. The few recent studies reviewed here indicate that the overall prevalence in a general population can range from about 1 to 36 in 100 and is more prevalent in populations with cardiometabolic disorders. Where clinicians begin to test for magnesium status, particularly in those with T2DM, hypertension, or metabolic syndrome, they are likely to find a high prevalence of hypomagnesaemia, perhaps 10% or higher depending on the patient population. This is particularly disconcerting as the prevalence of these diseases continues to rise worldwide. Clinicians working with individuals with suspected or demonstrated hypomagnesaemia may further consider other mineral/electrolyte disorders: hypomagnesaemia is known to exacerbate or co-occur with other deficiencies and in certain cases such deficiencies can be resolved by correcting magnesium deficiency.[9,10,33]

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Magnesium is found in a wide variety of foods and is among the less expensive supplements patient populations can obtain. Hypomagnesaemia in many circumstances can be relatively easily corrected with dietary and/or supplement counseling or prescription. Magnesium trials have shown that magnesium supplements are well tolerated and generally improve multiple indicators of disease status, even without clear changes in magnesium status (when assessed by serum magnesium).[10,33,34] Moreover, the long-term observational literature supports associations between higher magnesium intake and lower risk of diabetes, stroke, heart disease, as well as mortality.[33] Taken together, the recent body of evidence regarding magnesium appears to indicate prevalent deficiency of 20% to 30% in numerous common conditions and indicates a considerably protective role of this essential mineral in cardiometabolic disease.

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