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Review

Magnesium status and the metabolic syndrome: A systematic review and meta-analysis

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ABSTRACT

Objectives: To systematically review the published evidence regarding the association between Mg intake and serum concentrations with MetS and, if possible, to summarize the results using a meta-analysis.
Methods: PubMed, ISI Web of Science, Scopus, and Google Scholar were searched to identify related articles. Fully adjusted odds ratios (ORs) of MetS in participants with the highest intake of Mg compared with those who had the least consumption, and the mean difference in serum Mg levels between patients with MetS and their controls were extracted for the meta-analysis.
Results: In total, 9 articles with 31 876 participants were included in the meta-analysis regarding the association between dietary Mg intake and MetS, and 8 studies that assessed the mean level of serum Mg in 3487 individuals with and without MetS were eligible. Our analysis found that higher consumption of Mg is associated with lower risk of MetS (OR = 0.73; 95% confidence interval: 0.62, 0.86; $P < 0.001$); we also could find a significant but heterogeneous association between serum Mg and MetS (mean difference: −0.19; 95% confidence interval: −0.36, 0.03; $P = 0.023$).
Conclusions: The present systematic review and meta-analysis found an inverse association between Mg intake and MetS. However, the inverse association for serum Mg levels was highly heterogeneous and sensitive. The link between Mg status and MetS should be confirmed by prospective cohort studies controlling the association for other nutrients related to MetS risk.

Introduction

Controversy exists about serum magnesium (Mg) concentrations and low intakes of Mg in association with the metabolic syndrome (MetS) and related diseases such as insulin resistance, diabetes mellitus, hypertension, dyslipidemia, and cardiovascular diseases. MetS, a highly widespread disease among developed and developing populations [1], is characterized as a cluster of risk factors that threat public health and increase disability, mortality, and health-care costs [2]. On the basis of the Adult Treatment Panel III (ATP III) guidelines, MetS is defined as the presence of three or more of the following characteristics: high blood pressure, hyperglycemia, hypertriglyceridemia, low concentrations of high-density lipoprotein (HDL) cholesterol, and abdominal obesity [3]. Regardless of its definition, MetS increases the risk of diabetes and cardiovascular diseases [4].

Although the role of dietary ingredients is less understood, dietary components are important in many chronic diseases and it is believed that nutrients play important roles in the

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development of MetS [5]. Mg is an essential nutrient that particularly is found in green leafy vegetables, whole grains, legumes, and nuts [6]. As the second intracellular cation in abundance [7], Mg is a critical cofactor for hundreds of enzymatic reactions, including energy metabolism, protein and nucleic acid synthesis, and insulin secretion and action [8,9]. Previous studies reported that Mg deficiency is frequently (25% to 47%) observed in diabetic patients [10]. Rapidly growing evidence suggests that dietary Mg has valuable effects, including regulation of systemic inflammation [11] and hypertension [12], modification of lipids [13,14], glucose and insulin metabolism [15], improvement of insulin sensitivity [16,17], and decreasing the risk of diabetes [6].

Reduced serum Mg concentrations and low intakes of Mg are proposed to be associated with MetS, its components, and also other chronic conditions such as insulin resistance [18], diabetes mellitus [19], hypertension [8], dyslipidemia [20], and cardiovascular diseases [21]. Data about the association between Mg status and MetS are conflicting. Some studies have shown a negative association between Mg intake and MetS [22], whereas others could not reach a significant result [23]. We are aware of two systematic review and meta-analyses that found an inverse association between dietary Mg intake and MetS [24,25]. However, data about the association between serum Mg concentrations and MetS are more conflicting; for instance, some studies concluded that serum Mg levels are lower in patients with MetS compared with their controls [26,27], whereas other studies reported a non-significant association [28] or even proposed that there may be a positive association between serum Mg concentration and MetS [27].

Although some studies have reviewed the association between dietary Mg intake and MetS, to the best of our knowledge, no study has tried to summarize the published evidence regarding the association between serum levels of Mg and MetS. Therefore, the aim of the present study was to perform a systematic review and if possible meta-analyses on the observational studies regarding the association between dietary or serum Mg level and the likelihood of MetS.

Materials and methods

Search strategy

We focused on publications considering the association between dietary or serum content of Mg and MetS. PubMed, ISI Web of Science, Scopus, and Google Scholar were searched to identify related articles published up to February 2014. The following keywords, which were selected from the medical subject headings and other related terms, were used for the database search: “magnesium” OR “Mg” OR “magnesium” in combination with “Metabolic Syndrome X” OR “metabolic syndrome” OR “syndrome X.” No language or any other limitation was used in our literature search. Reference lists of related articles were considered for other potentially related papers. Titles and abstracts were separately reviewed by two authors (H.K.B., A.S.A.) to find eligible articles for the present study. Any inconsistency between search results was resolved by discussion with the other authors (N.S., M.L.).

Inclusion criteria

Observational studies published in peer-reviewed journals and assessed the association between Mg intake and MetS, or studies that compared serum levels of Mg in patients with and without MetS were included in our systematic review.

Exclusion criteria

Studies that examined the association of Mg intake or serum levels of Mg with MetS in participants with polycystic ovary syndrome [29], cardiac syndrome [30], diabetes mellitus [31–33], or morbidly obese individuals [34] were excluded from our study, because the risk estimates for becoming MetS for these participants are different from the normal population; furthermore, participants with these conditions are more likely to change their diet to manage their disease and this might confound the nutrient-disease associations. One study that did not compare the Mg content of the whole diet was also excluded [35].

Data extraction

We extracted all relevant information from eligible articles, including first authors’ family name, publication year, MetS definition, study design, participants’ sex, age range, geographic location, total sample size, sample size in case and control studies, Mg intake, and relative risks or odds ratios (ORs) of MetS for comparison between the highest and lowest category of Mg intake in models that had the highest number of adjusted variables in the statistical model. Furthermore, we extracted the mean ± SD of serum Mg levels from studies that compared serum Mg levels between participants with MetS and their controls. Data extraction and the quality assessment were completed by two authors (H.K.B. and A.S.A.) to guarantee equality, and one of the authors entered all of the data (H.K.B.).

Most of the studies presented the serum Mg in mg/dL. For studies in which Mg concentration was not shown in mg/dL, we changed other units, including mg/L [27], mmol/L [28], and meq/L [36], to mg/dL.

Data analysis

The ORs and their 95% confidence intervals (95% CIs) for comparing the prevalence or the incidence of MetS between groups with the highest and lowest Mg intake were used to calculate the log OR and its standard error (SE) as the effect size to be included in meta-analysis [37]. Relative risks (RRs) were considered as ORs when used in meta-analysis because only one study reported RRs [38]. We calculated ORs and their 95% CIs if a study reported the number of participants based on their Mg consumption strata. Eight studies also compared mean ± SD of serum Mg level in participants with MetS and their controls [20,26–28,36,39–41]; therefore, we used them to calculate unstandardized mean difference in serum Mg levels as effect size for another meta-analysis. Overall effect was derived by using random effects model, which takes between-study variations into account [37]. Statistical heterogeneity was assessed using Cochran’s Q test and I^2 [42]. Subgroup analysis and meta-regression were incorporated to search about the possible sources of heterogeneity if needed. Sensitivity analysis was used to explore the extent to which inferences might depend on a particular study or a number of publications. Publication bias was evaluated by looking over Begg’s funnel plots [43]. Formal statistical assessment of funnel plot asymmetry was conducted using Egger’s regression asymmetry test and Begg’s adjusted rank correlation test. Statistical analyses were conducted using STATA version 11.2 (STATA Corp., College Station, TX, USA). P values less than 0.05 were considered statistically significant.

Results

Study characteristics

Our search retrieved 141 articles, of which 19 related articles fulfilled our inclusion criteria [5,10,16,21,33,35,36,38,43–53]. One study that compared mean Mg intake between patients with MetS and controls (but not reported OR) was included in our systematic review but not in the meta-analysis [54]. Another study done by Mirmiran et al. [55] did not represent data on OR for MetS based on Mg intake groups; therefore, we asked authors about data by three e-mails separated at least by 1 wk; however, the authors could not provide the needed data; therefore, we included this article in the systematic review but not in the meta-analysis too. The flow diagram of the study selection process is illustrated in Figure 1.

Among the reviewed articles included in meta-analysis, five were conducted in the United States [43,45–48]; two in Greece [36,39]; two in Mexico [20,26]; one in Australia [28], China [27], Taiwan [22], Italy [23], Brazil [41], South Korea [56], and Saudi Arabia [57]; and three in Iran [40,51,55] (Tables 1 and 2). The participants’ age ranged from 18 to 90 y and the study sample size ranged from 117 to 11 686 participants, and totally 35 363 individuals participated in all studies. Among the 17 studies, 1 study was conducted entirely in women [38], 1 study in men [27], and other studies reported the association for both women and men. Characteristics of included studies that examined the association for dietary and serum Mg levels are presented in Tables 1 and 2, respectively.

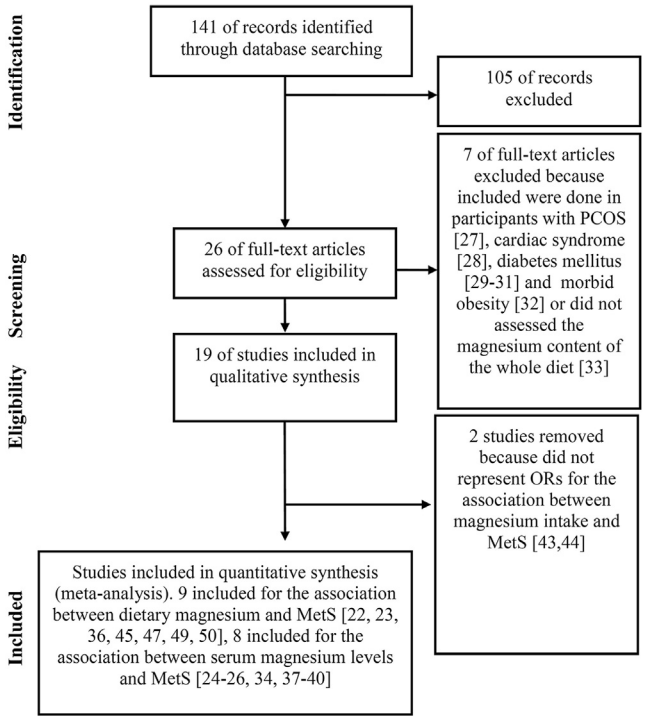

Fig. 1. The study selection process. MetS, metabolic syndrome; PCOS, polycystic ovary syndrome.

In total, 9 articles with 31 876 participants were included in the meta-analysis regarding the association between dietary Mg intake and MetS [22,23,38,45,46,50,51,56,57]. The results of our analysis showed that higher consumption of Mg is associated with lower risk of MetS (OR = 0.73; 95% CI: 0.62, 0.86; $P < 0.001$) (Fig. 2). However, there was a slight level of heterogeneity between studies (Cochrane Q test, $P = 0.149$, $I^2 = 32.4\%$). When the methodology of included studies was carefully reviewed, we noticed that a study done by McKeown et al. [50] used body mass index (BMI) higher than 31 kg/m^2 for men and 27 kg/m^2 for women as an index for abdominal obesity, whereas other studies defined MetS based on ATP III; therefore, we removed this study from our analysis and the heterogeneity significantly decreased (Cochrane Q test, $P = 0.429$, $I^2 = 0.5\%$), whereas the association remained significant (OR = 0.78; 95% CI: 0.69, 0.88; $P < 0.001$) (Fig. 3).

Our preliminary analysis on 8 studies [20,26–28,36,39–41], which assessed the mean serum Mg in 3487 individuals with and without MetS (Fig. 4), indicated that serum Mg level in MetS participants is about 0.19 mg/dL lower compared with the normal population (95% CI: −0.36, −0.03; $P = 0.023$), but there was a significant heterogeneity between studies (Cochrane Q test, $P < 0.001$, $I^2 = 98.5\%$). Among included studies, two had used International Diabetes Federation's criteria [39,41] and one had used Chinese Diabetes Society's criteria [27] to define MetS, and a study done by Guerrero-Romero and Rodriguez-Moran [26] had excluded individuals with hypertension and diabetes from the study population. Therefore, these four studies were

Table 1
Characteristics of studies investigating the association between dietary magnesium intake and metabolic syndrome

Author, year (country)	Metabolic syndrome definition	Study design	Sample size	Age	Sex	Adjusted OR or RR	Adjusted variables
Bo et al., 2006 (Italy) [23]	ATP III	Cross-sectional	1658	45–64	Both	1.03 (0.62, 1.70)	Age, sex, BMI, smoking, alcohol, physical activity, calories, fat, fiber
Ford et al., 2007 (United States) [45]	ATP III	Cross-sectional	7669	>20	Both	0.56 (0.34, 0.92)	Age, sex, race, education, smoking, C-reactive protein, alcohol, physical activity, family history of coronary disease, vitamin and supplement intake, diabetes history, fat, carbohydrates, fiber, total energy
He et al., 2006 (United States) [46]	ATP III	Cohort	4637	18–30	Both	0.63 (0.47, 0.84)	Age, sex, race, education, smoking, physical activity, family history of diabetes, alcohol, BMI, fiber, PUFAs, SFAs, CHO, total energy intake
Song et al., 2005 (United States) [38]	ATP III	Cross-sectional	11686	>45	Female	0.65 (0.52, 0.83)	Age, calorie, smoking, physical activity, alcohol, family history of myocardial infarction, fat, cholesterol, folate, glycemic load
Noori et al., 2010 (Iran) [51]	ATP III	Cohort	160	>18	Both	1.73 (0.38, 7.85)	Age, sex, smoking, physical activity, dialysis mode, dose of steroid, FH diabetes and stroke, calorie, BMI, fiber
McKeown et al., 2008 (United States) [50]	ATP III	Cross-sectional	535	>60	Both	0.36 (0.19, 0.69)	Age, sex, race, education, marital status, smoking, alcohol, physical activity, BMI, calorie, SFA, lipid medication, BP medication except for model with BP
Huang et al., 2012 (Taiwan) [22]	ATP III	Cross-sectional	210	<65	Both	0.49 (0.17, 1.43)	—
Morrell et al., 2012 (United States) [54]	AHA	Cross-sectional	2722	18–24	Both	—	—
Mirmiran et al., 2012 (Iran) [55]	ATP III	Cross-sectional	2504	18–74	Both	—	Age, sex, smoking, physical activity, fiber, SFAs, K, Ca, Na, fish and food groups
Choi and Bae, 2013 (South Korea) [56]	ATP III	Cross-sectional	2084 3052	>19 >19	Male Female	0.81 (0.53, 1.23) 0.90 (0.62, 1.30)	Age
Al-Daghri et al., 2013 (Saudi Arabia) [57]	IDF	Cross-sectional	185	19–60	Both	0.50 (0.25, 1.00)	—

AHA, American Heart Association; ATP III, Adult Treatment Panel III; BMI, body mass index; Ca, calcium; CHO, carbohydrate; IDF, International Diabetes Federation; K, potassium; Na, sodium; OR, odds ratio; PUFAs, polyunsaturated fatty acids; RR, relative risk; SFAs, saturated fatty acids

Table 2
Characteristics of studies investigating the association between serum magnesium level and metabolic syndrome (MetS)

Author, year (country)	Metabolic syndrome definition	Study design	Sample size	Age (cases)	Age (controls)	Sex	No MetS serum mg, mean ± SD	MetS serum mg, mean ± SD	Adjusted variable
Evangelopoulos et al., 2008 (Greece) [39]	IDF	Cross-sectional	117	67.0	65.0	Both	1.99 ± 0.24 (n = 35)	1.83 ± 0.23 (n = 82)	—
Ghasemi et al., 2010 (Iran) [40]	ATP III	Cross-sectional	137	60–90	60–90	Both	2.16 ± 0.03 (n = 77)	2.08 ± 0.03 (n = 60)	Sex, creatinine, high TG, high WC, hypertension, low HDL-C, hyperglycemia, diabetes
Guerrero-Romero and Rodriguez-Moran, 2002 (Mexico) [26]	ATP III	Case-control	576	42.3	41.5	Both	2.20 ± 0.20 (n = 384)	1.80 ± 0.30 (n = 192)	—
Guerrero-Romero and Rodriguez-Moran, 2006 (Mexico) [20]	ATP III	Case-control	441	44	43.0	Both	2.1 ± 0.3 (n = 294)	1.8 ± 0.4 (n = 147)	—
Kalaitzidis et al., 2005 (Greece) [36]	ATP III	Cross-sectional	255	48.8	48.7	Both	2.04 ± 0.36 (n = 191)	1.92 ± 0.36 (n = 64)	—
Simmons et al., 2010 (Australia) [28]	ATP III	Cross-sectional	1453	53	53		2.02 ± 0.14 (n = 1168)	2.02 ± 0.17 (n = 363)	—
Yu et al., 2012 (China) [27]	CDS	Cross-sectional	379	24–57	24–57	Male	1.93 ± 0.28 (n = 149)	2.09 ± 0.28 (n = 52)	—
Lima et al., 2009 (Brazil) [41]	IDF	Cross-sectional	129	45.7 ± 11.8	45.7 ± 11.8	Both	2.43 ± 0.43 (n = 57)	1.80 ± 0.18 (n = 72)	—

ATP III, Adult Treatment Panel III; CDS, Chinese Diabetes Society; HDL-C, high-density lipoprotein cholesterol; IDF, International Diabetes Federation; Mg, magnesium; TG, triacylglycerol; WC, waist circumference

removed from our analysis, but the heterogeneity remained significant (Cochrane Q test, $P = 0.000$, $I^2 = 96.2\%$) (Fig. 5). Moreover, removing these articles turned the association between serum levels of Mg and MetS into a non-significant one (mean difference [MD] = −0.12; 95% CI: −0.28, 0.03; $P = 0.113$). Furthermore, we tried to find the source of heterogeneity using a subgroup analysis based on the participants' age and sex, criteria used to define MetS, study design, and country of origin. We also incorporated meta-regression to examine if publication year and participants' mean age can describe the high level of heterogeneity between the included studies; however, none of these methods were successful to find the source of heterogeneity.

Sensitivity analysis found that none of individual studies trying to examine the association between dietary or serum levels of Mg and MetS could significantly change the overall effects. Although Begg's funnel plot for studies that reported the ORs of Mg intake showed that the distribution of calculated effect sizes was slightly asymmetrical (Fig. 6A), the asymmetry tests did not provide any evidence of publication bias (Begg's test, $P = 0.245$; Egger's test, $P = 0.140$). Begg's funnel plot for studies that reported the serum Mg between patients with and without MetS was also the same (Begg's test, $P = 0.621$; Egger's test, $P = 0.210$) (Fig. 6B).

Discussion

The present systematic review and meta-analysis found that increased Mg consumption is associated with lower risk of MetS.

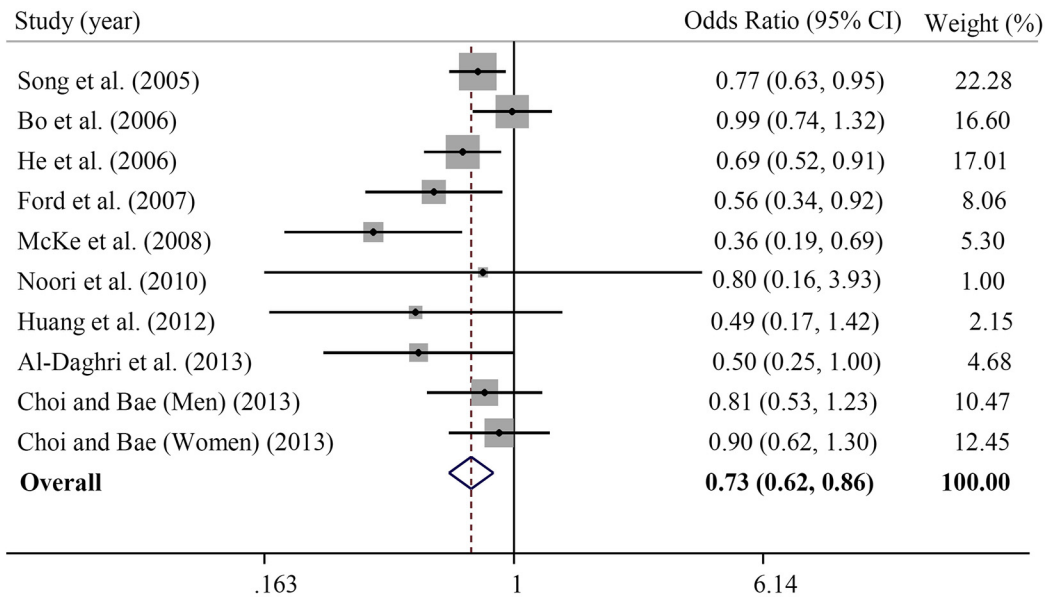

Fig. 2. Meta-analyses of studies examining dietary magnesium intake and metabolic syndrome. CI, confidence interval.

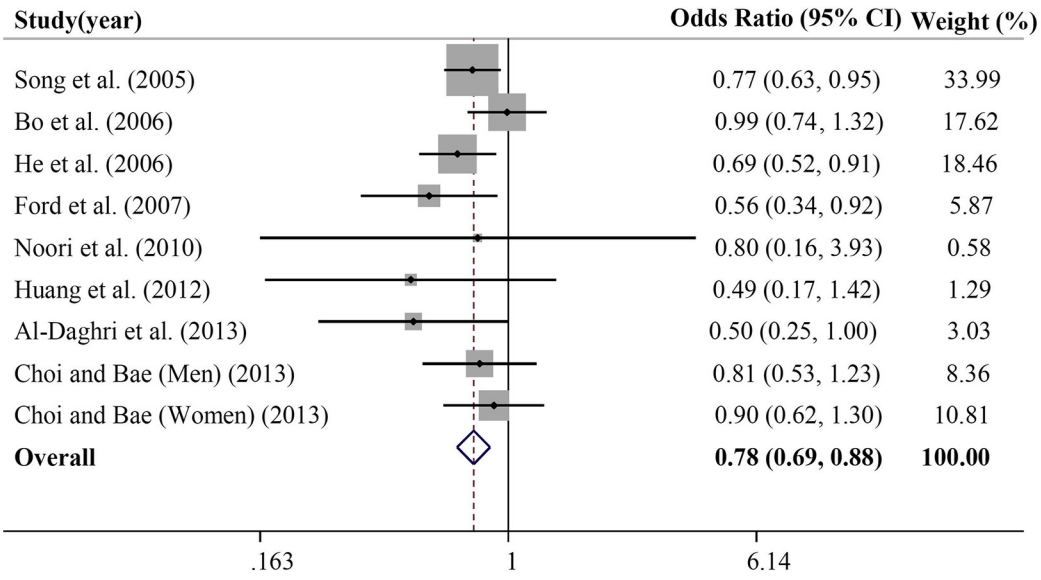

Fig. 3. Meta-analyses of studies examining dietary magnesium intake and metabolic syndrome after removing study done by McKeown et al. [50], which used body mass index higher than 31 kg/m^2 for men and 27 kg/m^2 for women as an index for abdominal obesity. CI, confidence interval.

Moreover, although the heterogeneity was high, our analyses revealed that patients with MetS have lower levels of blood Mg concentrations than healthy controls; however, this association was not replicated when we excluded studies that used different definitions other than ATP III for MetS. To the best of our knowledge, there are two review articles published in 2014 regarding the association between Mg intake and MetS [24,25]. Ju et al. [24] showed an inverse dose-response association among nine observational studies, whereas Dibaba et al. [25] found the same relationship in six observational studies in their analysis. In the present study we could find 11 studies regarding the association between dietary Mg intake and MetS and included 9 studies in the meta-analysis. Furthermore, we reviewed studies that examined serum Mg levels in association with MetS and summarized their effect using a meta-analysis.

We could not include two studies [54,55] regarding the association between dietary Mg intake and MetS because they did not provide the essential data. However, all these studies have mentioned an inverse association between dietary Mg intake and MetS in their results. Morrell et al. [54] compared individuals with two or more criteria for MetS and participants without any MetS criteria and found a significant difference in their Mg intake (251 versus 269 mg). Mirmiran et al. [55] also observed a negative association between dietary Mg and MetS *Z*-score in obese individuals and high-fiber consumers, but this association was not significant in normal-weight participants or in people with low fiber consumption. After adjustment for other covariates, no significant association was found between Mg intake and MetS. This study indicated that the association between Mg intake and MetS cannot be independent of fiber, calcium, and potassium and other nutrients intake.

Although we found a strong inverse association between Mg intake and the likelihood of MetS, we could not find the same association for serum Mg levels when we pooled studies that had

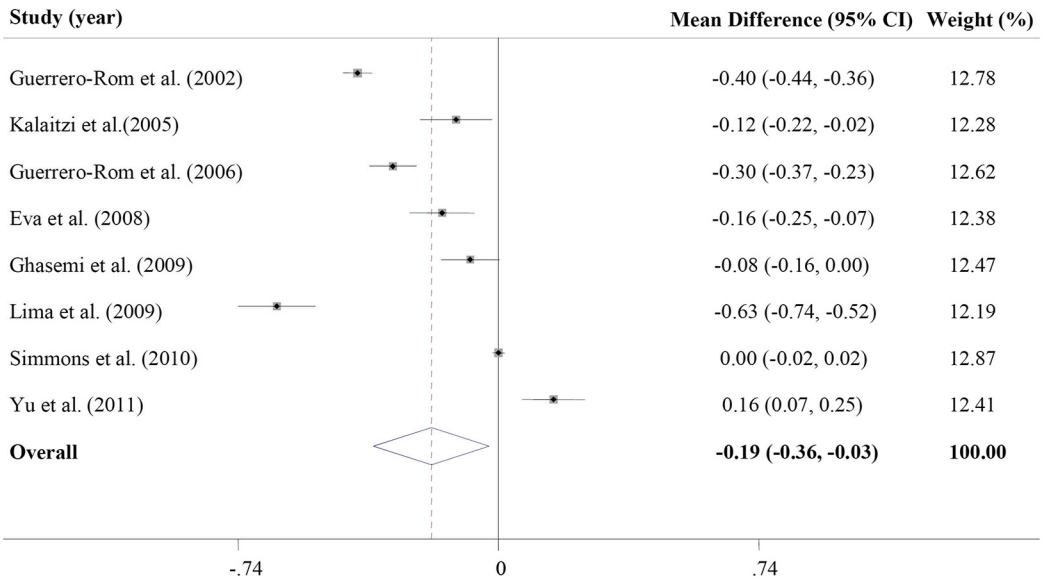

Fig. 4. Meta-analyses of studies examining serum magnesium level and metabolic syndrome. CI, confidence interval.

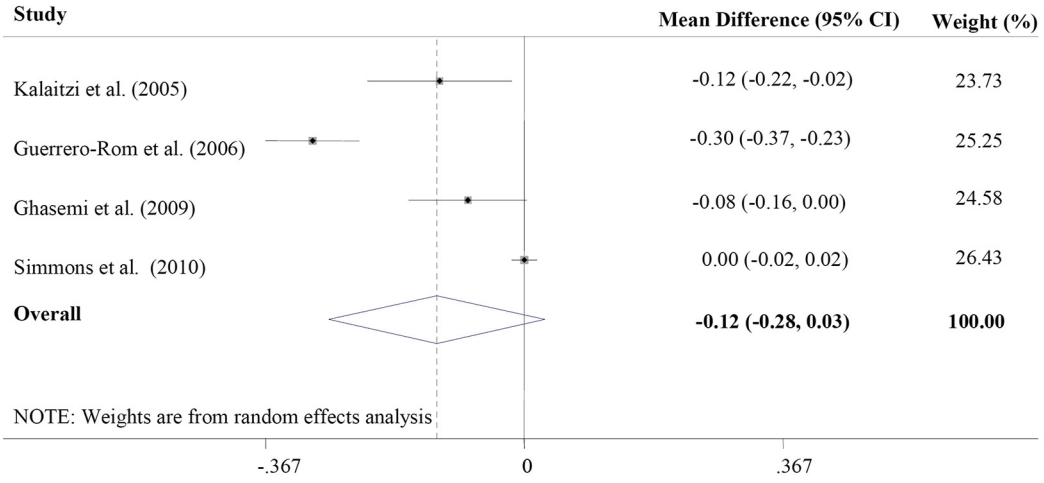

Fig. 5. Meta-analyses of studies examining serum magnesium level and MetS after removing two studies that had used International Diabetes Federation's criteria [37,40] and one study that had used Chinese Diabetes Society's criteria [24] to define MetS and a study done by Guerrero-Romero [26], which had excluded individuals with hypertension and diabetes from the study population. CI, confidence interval; MetS, metabolic syndrome.

used the same definitions for MetS. A previous meta-analysis reported that Mg supplementation significantly increased serum or plasma Mg levels [58]. Serum Mg is the most common indicator for assessing Mg status and it is valuable for clinical purpose, such as rapid assessment of critical changes in Mg status [59]. However, Mg is an intracellular cation and only 1% of Mg is in the extracellular fluid; therefore, only about 0.3% of total body Mg is found in the serum. As a result, some researchers have considered serum mg levels as a poor indicator of body Mg status [60]. Moreover, in some people with chronic Mg deficiency, their serum levels might be within the normal range [59]. It could be a possible reason for not finding a significant association between serum Mg and MetS in the present study. Although in some situations such as being vegetarians (in comparison with omnivorous), after endurance exercises, and during the third trimester of pregnancy the serum Mg levels are higher [61], it is still used as a standard method for assessing the Mg status in patients, because serum Mg measurement is feasible and inexpensive [62]. Additionally, the difference in association between dietary intake and serum levels of Mg in association with MetS might be because of not taking some confounders such as other nutrients intake, particularly dietary fiber, calcium, and potassium intake into account. Prospective cohort studies trying to examine Mg status–MetS relationship, while further adjusting the association by dietary fiber, calcium, and potassium intake and other nutrients that might possibly affect MetS risk, are highly recommended.

Although studies are not entirely consistent about the possible mechanisms, the beneficial effect of Mg on MetS might be related to its potential effect on MetS components. The primary aim of the present study was to assess the association between Mg status and the odds of MetS; as the association for the components of MetS was not assessed in the majority of included studies, we were not able to explore this association in the present work. The beneficial effects of Mg supplementation on some components of MetS such as hypertension [49], fasting blood glucose levels, and HDL cholesterol levels [58] have been revealed in previous systematic reviews and meta-analyses. Previous studies also reported that low dietary intakes of calcium, Mg, and fiber may be associated with insulin resistance and that supplementation with Mg and calcium improved insulin sensitivity [52,63]. Another study conducted by Paolisso et al. [52] found that Mg supplementation in elderly patients improves insulin action. Furthermore, it has been also shown that Mg intake is associated with all components of Met [47], particularly waist circumference, fasting blood glucose, and HDL cholesterol level [46]. Mg intake was also inversely linked to the plasma C-reactive protein concentration and systemic inflammation [38].

The nutrition guidelines of the American Diabetes Association recommended Mg supplementation for diabetic individuals with low serum Mg levels [44]. Although this meta-analysis could not find a strong association between serum Mg and MetS, it has been indicated that low serum Mg is a significant predictor of dyslipidemia and hypertension [3]. Also, serum Mg was inversely associated with waist circumference and triacylglycerol (TG) level [32]. It has been revealed that Mg may regulate serum lipid level [64], glucose metabolism, and insulin activity [65], and also has antioxidant [8], anti-inflammatory [38], and antihypertensive [66] effects. Several mechanisms have been suggested to explain the favorable effects of Mg on features of MetS or its components.

It has been previously suggested that higher Mg intake and intracellular Mg might have a role in insulin action and secretion [16,48] by preserving pancreatic β-cell function via its effect on calcium homeostasis and oxidative stress [8]. Moreover, Mg is necessary for the stimulation and transcription of some enzymes and nuclear proteins that are participating in the glycolytic pathway, Krebs cycle, and release of insulin. Besides, the insulin function is completely dependent on Mg, as it is responsible for the activation of the β-subunit of the insulin receptor, and the motivation of substrates and proteins in the insulin signaling pathway [67]. It also regulates cellular glucose metabolism via its role as a cofactor [68].

Similarly, Mg acts as a cofactor for several critical enzymes involving lipid metabolism. Mg has been reported to raise HDL and reduce LDL cholesterol and TG via restraining the action of lecithin cholesterol acyltransferase [69] and HMG-CoA reductase and by increasing lipoprotein lipase activity [53]. It has been assumed that Mg, in the intestine, by forming an unabsorbable soap with fatty acids and cholesterol, can decrease their absorption, reduce energy intake from the diet, and may have advantages for weight maintenance because of this capability [59].

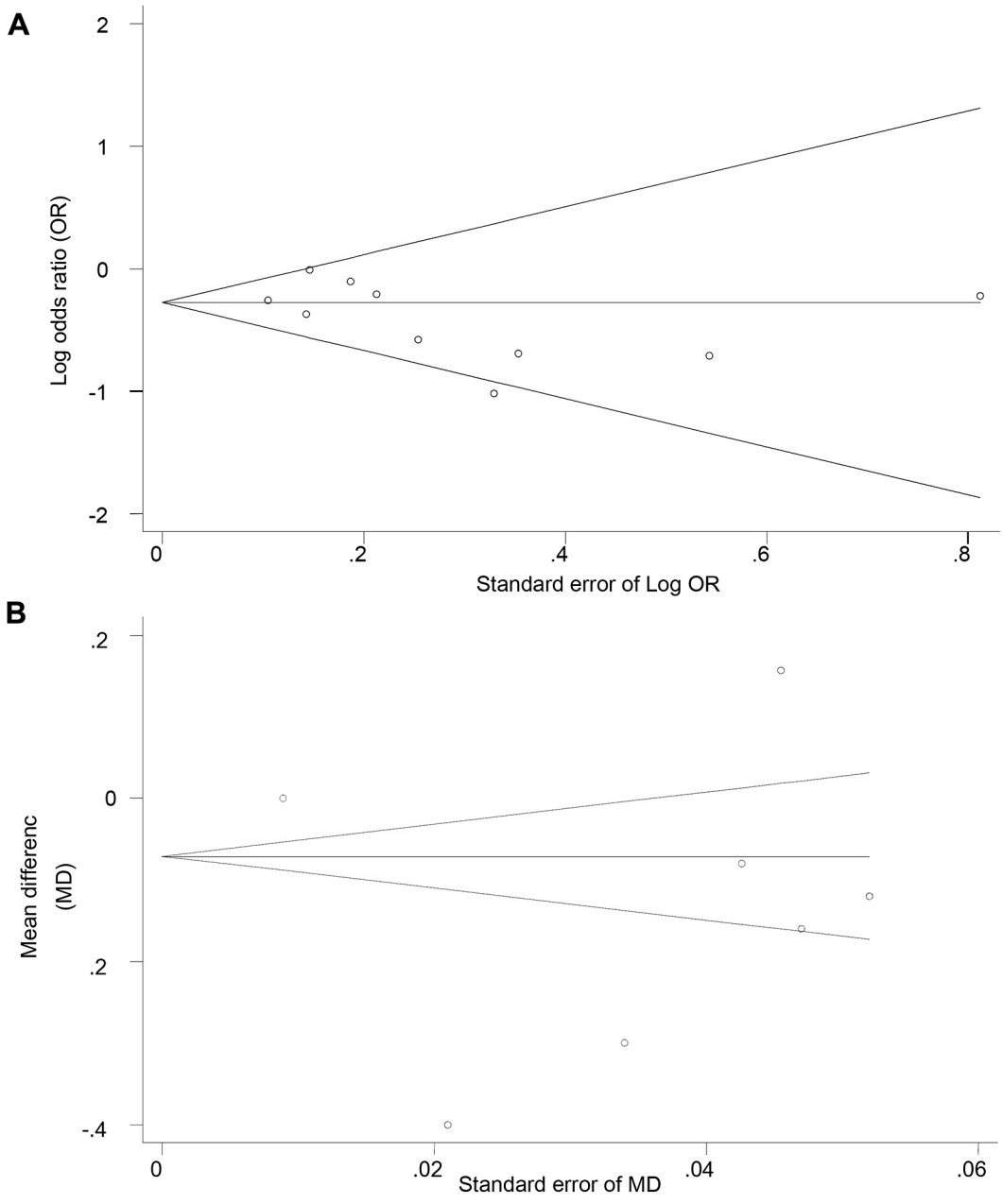

Fig. 6. (A) Begg's funnel plot (with pseudo 95% CI) in log OR versus standard error of log OR for studies that reported the OR of Mg intake. (B) Begg's funnel plot (with pseudo 95% CI) in MD versus standard error of MD for studies that reported the serum Mg. CI, confidence interval; MD, mean difference; Mg, magnesium; OR, odds ratio.

The present study has several limitations that should be considered. We could find only one prospective cohort study, and other included studies that tried to find the possible association between Mg intake and MetS were cross-sectional in design. Furthermore, all included studies, which tried to compare serum levels of Mg between patients with MetS and healthy controls, were case-control studies. Retrospective studies including cross-sectional and case-control studies are prone to recall bias, and also the causal relationship cannot be inferred from these studies; therefore, prospective studies trying to find these associations are strongly recommended. The possible mechanisms for the effect of Mg on MetS and its components can be tested through further clinical trials with large sample sizes and follow-up periods. Moreover, although we used the highest adjusted model when analyzing the association between Mg intake and MetS, most of the studies could not control the dietary factors that are associated with MetS, and it is difficult to entirely separate the effect of Mg from other dietary nutrients such as calcium, potassium, and fiber. Finally, our analysis on the mean serum level of Mg in individuals with and without MetS demonstrated a significant heterogeneity among studies, and we could not find the possible source of heterogeneity in several subgroup analyses and meta-regression.

Conclusions

The present systematic review and meta-analysis found a strong inverse association between Mg intake and MetS.

Although we found the same association about the relationship between serum Mg, as a measure of body Mg, and MetS, the association was highly heterogeneous. Therefore, our results should be discussed with more caution. Assessing the body Mg with better indicators of body storage such as Mg retention test, which shows the stored body Mg, might reveal more accurate results. Prospective cohort studies trying to examine adjusting the Mg status–MetS association for other nutrients that might possibly affect MetS risk are highly recommended.

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