Magnesium intake is associated with the metabolically healthy obese phenotype

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Accepted 6 October 2021
Published Online First 1 November 2021

ABSTRACT

Although magnesium intake is inversely associated with the risk of metabolic abnormalities, whether magnesium intake plays a role on metabolically healthy obese (MHO) phenotype has not been explored. Therefore, the purpose of this study was to determine whether the magnesium intake is associated with the MHO phenotype. Apparently, healthy women and men aged 20–65 years with obesity were enrolled in a cross-sectional study. Subjects were allocated into MHO (n=124) and metabolically unhealthy obese (MUO) (n=123) groups. MHO phenotype was defined by abdominal obesity (waist circumference ≥90 cm in men and ≥80 cm in women) and none, or not more than one of the following risk factors: triglyceride levels ≥150 mg/dL; high-density lipoprotein cholesterol (HDL-C) levels <40 mg/dL in men and <50 mg/dL in women; fasting glucose ≥100 mg/dL; and systolic blood pressure ≥130 mm Hg and/or diastolic blood pressure ≥85 mm Hg. The MUO individuals were characterized by abdominal obesity and the presence of two or more of the aforementioned criteria. The proportion of individuals with high blood pressure (40.7% vs 5.6%, p<0.001), hyperglycemia (69.1% vs 16.9%, p<0.001), hypertriglyceridemia (84.6% vs 36.3%, p<0.001), and low HDL-C (51.2% vs 12.9%, p<0.001) was significantly higher in the MUO individuals as compared with individuals in the MHO group. The logistic regression analysis adjusted by sex and age showed that dietary magnesium intake is significantly associated with the MHO phenotype (OR=1.17; 95% CI 1.07 to 1.25, p=0.005). Our results show that magnesium intake is significantly associated with the MHO phenotype.

Significance of this study

What is already known about this subject?
- Metabolically healthy obese (MHO) phenotype is a subset of obese subjects without obesity-related metabolic disorders.
- Dietary magnesium intake is inversely associated with the risk of metabolic abnormalities.

What are the new findings?
- Magnesium intake is directly associated with the metabolically healthy obese phenotype.

How might these results change the focus of research or clinical practice?
- Our results strongly suggest that the magnesium intake may play a central role in the development of the MHO phenotype; therefore, the dietary magnesium intake might prevent the development of metabolic disorders in obese individuals.

INTRODUCTION

Undoubtedly, obesity is one of the most important health problems worldwide.[1] It is well known that obesity is strongly associated with the development of chronic diseases, including type 2 diabetes, hypertension, cardiovascular disease, stroke, certain types of cancer, liver disease, and osteoarthritis.[2 3] However, some individuals who have obesity do not have metabolic abnormalities; these patients are considered the metabolically healthy obese (MHO).[4 5]

Some meta-analyses have reported that dietary magnesium intake is inversely associated with the risk of metabolic abnormalities.[6–8] In addition, it has been proposed that serum magnesium levels play a pivotal role in the development of the MHO phenotype.[9] In this regard, we recently reported that oral magnesium supplementation significantly reduced blood pressure and glucose and triglyceride concentrations in obese individuals with metabolic syndrome.[10] This finding supports the hypothesis that serum magnesium may be related to metabolic syndrome.[11] However, the role that dietary magnesium intake plays in avoiding the development of metabolic abnormalities in obese individuals has not been previously explored. Hence, the objective of this study was to determine whether magnesium intake is associated with the MHO phenotype.

MATERIALS AND METHODS

The Ethics and Research Committee of the Mexican Social Security Institute approved the protocol used in this study (registry number R-2017-785-078). After obtaining written

To cite: Guerrero-Romero F, Morales-Gurrola G, Preza-Rodríguez L, *et al*. *J Investig Med* 2022;**70**:800–804.

informed consent from those involved in this study, a cross-sectional study was conducted.

Obese (body mass index (BMI) ≥30 kg/m^2) women and men aged 20–65 years were recruited from the general population of Durango, a city in northern Mexico, from January to December 2018. The sampling strategy consisted of a general invitation to obese individuals to participate in this study; this invitation was sent through social networks.

Prior to enrollment in this study, anthropometric measurements and routine biochemical parameters were acquired from all participants.

Exclusion criteria were as follows: normal weight (BMI <25 kg/m^2), overweight (BMI >25 and ≤29.9 kg/m^2), absence of abdominal obesity, smoking, alcohol consumption, pregnancy, hypertension, diabetes, cardiovascular disease, stroke, liver diseases, renal diseases, any kind of medical treatment, and use of magnesium supplements.

Subjects that fulfilled the inclusion criteria were separated into the MHO or metabolically unhealthy obese (MUO) groups.

Definitions

The MHO phenotype was defined by abdominal obesity (waist circumference (WC)≥90 cm in men and ≥80 cm in women) and no more than one of the following risk factors: triglyceride levels≥150 mg/dL; reduced high-density lipoprotein cholesterol (HDL-C) <40 mg/dL in men and <50 mg/dL in women; fasting glucose ≥100 mg/dL; and systolic blood pressure (SBP) ≥130 mm Hg and/or diastolic blood pressure (DBP) ≥85 mm Hg.[12] The MUO individuals were characterized by abdominal obesity and the presence of two or more of the aforementioned criteria.

Measurements

Weight and height were measured using a fixed scale and attached stadiometer with the participants in light clothing, in a standing position, and without shoes. BMI was calculated as weight (kg) divided by height-squared (m^2). The WC was measured to the nearest centimeter using a flexible measuring tape; the midpoint between the lowest portion of the rib cage and the superior border of the iliac crest were the anatomical landmarks used to position the tape.[9]

Total body fat was estimated using bioelectrical impedance methodology (Tanita TBF-215, Tokyo, Japan).

Blood pressure was measured with a sphygmomanometer (Microlife AG, Heerbrugg Switzerland) and a stethoscope (3M Littman Classic II, Neuss, Germany), and during the measurement, the participants were seated after resting for at least 5 min. The blood pressure measurement technique was in accordance with the criteria set by the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.[13]

Dietary magnesium intake was estimated by 24-hour dietary recall questionnaires using a microcomputer-based interview system. The 24-hour dietary recall is the most frequently used method for collecting data regarding habitual intake.[14] The 24-hour dietary recall test has been validated comparing data collected against concurrent measurement of total energy expenditure[15] and examining common design of 24-hour dietary recall tools.[16] The 24-hour recall test asks the patients to elaborate a list of foods and beverages as well as the portion sizes intake day before. Data collected were analyzed for nutritional composition using the Nutrimind software V.14 (Aguascalientes, Ags., Mexico). The interviewer paid special attention regarding the report of intake of fruits, grain, nuts, dairy products, poultry, fish and seafood, vegetables, and legumes, foods particularly rich in magnesium. Analysis of magnesium intake was performed.

Assays

Whole blood samples were collected from antecubital veins after 8–10 hours of overnight fasting. Serum glucose was measured using the glucose oxidase method; the intra-assay and interassay coefficients of variation were 1.1% and 1.3%, respectively. Triglyceride levels were measured enzymatically; the HDL-C fraction was obtained after precipitation by phosphotungstic reagent. The intra-assay and interassay coefficients of variation were 1.9% and 3.7% for triglycerides and 1.5% and 3.1% for HDL-C, respectively. Measurements were performed with a Data Pro Plus clinical analyzer (Arlington, Texas, USA).[9]

Sample size was estimated taking into account the prevalence of both MHO (27%)[9] and hypomagnesemia (36.3% and 31.0% for females and males).[17] The α and β values were 0.05 and 0.80, respectively. The required sample size was 120 individuals per group.

Statistical analysis

Numerical values are reported as mean±SD, and categorical variables are expressed as proportions.

The differences between the groups were estimated using unpaired Student's t-tests (Mann-Whitney U tests for skewed data) for numerical variables and χ^2 tests for categorical variables. Comparisons between more than two groups were performed using one-way analysis of variance with the Games-Howell post hoc test.

The relationship between WC and magnesium intake was estimated using the Pearson correlation test.

A multiple logistic regression analysis was conducted to evaluate the association between daily magnesium intakes (independent variable) with the MHO phenotype (dependent variable). The logistic regression analysis was adjusted by age and sex.

The Statistical Package for the Social Sciences (SPSS) for Windows, V.15.0 (SPSS) was used for data management and statistical analysis. A 95% CI and p value <0.05 were used to define statistical significance.

RESULTS

A total of 247 individuals, 176 (71.2%) women and 71 (28.7%) men, were enrolled and allocated into the MHO (n=124) and MUO (n=123) groups.

Total body fat, BMI, and WC showed no significant differences between the study groups. The individuals in the MUO group had higher SBP, DBP, fasting glucose, and triglyceride levels and lower HDL-C levels compared with the individuals in the MHO group (table 1). Besides, the subjects in the MHO group exhibited significantly higher daily magnesium intake than those in the MUO group (figure 1).

Table 1 Characteristics of individuals in study, N=247

	MHO	MUO	
N	124	123	P value
Men/women, n	28/96	42/81	0.002
Age, years	40.6±7.5	43.5±7.7	0.03
Body mass index, kg/m^2	34.9±4.7	34.5±4.1	0.47
Waist circumference, cm	107.0±12.7	107.3±13.4	0.87
Total body fat, %	42.8±6.5	42.4±7.2	0.53
Systolic blood pressure, mm Hg	111.5±8.6	118.6±13.1	<0.005
Diastolic blood pressure, mm Hg	72.3±7.8	77.4±8.9	<0.005
High blood pressure, n (%)	7 (5.6)	50 (40.7)	<0.001
Fasting glucose, mg/dL	94.1±7.8	106.7±16.4	<0.005
Hyperglycemia, n (%)	21 (16.9)	85 (69.1)	<0.001
HDL-C, mg/dL	49.7±11.0	42.5±11.6	<0.005
Low HDL-C, n (%)	16 (12.9)	63 (51.2)	<0.001
Triglycerides, mg/dL	147.8±76.9	241.9±122.4	<0.005
Hypertriglyceridemia, n (%)	45 (36.3)	104 (84.6)	<0.001
Magnesium intake, mg/day	334.1±134.8	251.75±93.9	<0.005
Serum magnesium, mg/dL	2.04±0.21	1.70±0.26	<0.005

Values are mean±SD.
HDL-C, high-density lipoprotein cholesterol; MHO, metabolically healthy obese; MUO, metabolically unhealthy obese.

Individuals in the MUO group had significantly higher SBP values and fasting glucose levels, as well as lower HDL-C, and magnesium intake than those in the MHO group (table 2).

The proportion of individuals with high blood pressure, hyperglycemia, hypertriglyceridemia, and low HDL-C was significantly higher in the MUO group than in the MHO group (table 1).

A total of 35 (28.2%) individuals in the MHO group had not cardiovascular risk factors, whereas 89 showed one risk factor, hypertriglyceridemia the most frequent (table 2). All individuals in the study were obese; hence, in the MUO group, all participants exhibited metabolic syndrome, among them, the hypertriglyceridemia and low HDL-c were the most frequent (table 2).

There was a significant decrease trend of magnesium intake as a function of number the cardiovascular risk factors (figure 2). Individuals with MHO (one or none risk factor) showed a significant greater magnesium intake than individuals with MUO (two or more risk factors), p for trend <0.01.

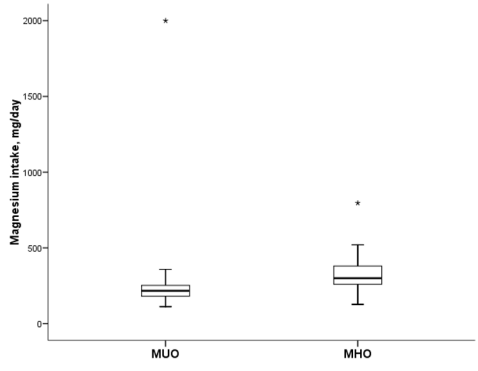

Figure 1 Boxplot of magnesium intake in metabolically unhealthy obese (MUO) and metabolically healthy obese (MHO) groups.

The Pearson correlation test showed no a significant relationship between WC and magnesium intake for the individuals in the MUO (R=0.57, p=0.62 and R=–0.005, p=0.97, for women and men) and MHO groups (R=0.03, p=0.97 and R=–0.026, p=0.90, for women and men).

The adjusted OR between daily magnesium intake and the MHO phenotype was 1.17 (95% CI 1.07 to 1.25, p=0.005). A total of 36 (29%) individuals in the MHO group exhibited no cardiovascular risk factors. An additional logistic regression analysis in this subgroup revealed that daily magnesium intake was also significantly associated with the MHO phenotype (OR 1.17; 95% CI 1.03 to 11.20, p=0.005).

DISCUSSION

Our results show that dietary magnesium intake is associated with the MHO phenotype, a finding suggesting that an appropriate magnesium intake could be related to the absence of cardiovascular risk factors in MHO individuals.

Studies analyzing the role that dietary magnesium intake may play in the cardiometabolic profile of obese individuals are scarce and inconsistent.[18 19] Hankinson *et al*[18] reported that magnesium intake had non-significant differences between adults with the MHO phenotype and MUO. Therefore, they concluded that diet composition does not explain the absence of cardiometabolic abnormalities in obese adults. However, Sukumar *et al*[19] stated that dietary magnesium intake might help to characterize the MHO phenotype, which is in agreement with our results.

Inconsistencies between the study by Hankinson *et al*[18] and the results from Sukumar *et al*[19] and our study could be related to the definition of the MHO phenotype. Hankinson *et al*[18] defined the MHO phenotype as obesity (BMI ≥30 kg/m^2) and no diagnosis or treatment of hypertension, diabetes, dyslipidemia, or cardiovascular disease. In contrast, in our study and the study by Sukumar *et al*,[19] the MHO phenotype was defined by abdominal obesity and no more than one additional risk factor. However, the subanalysis focused on obese individuals without additional risk factors (a population similar to that analyzed by Hankinson *et al*,[18] found that magnesium intake was associated with the MHO phenotype. Undoubtedly, further research is needed in the field.

Although data about the relationship between magnesium intake and the MHO phenotype are scarce,[18 19] the role that magnesium plays in the risk of developing metabolic syndrome has been well explored. Using data from the Third National Health and Nutrition Examination Survey, Ford *et al*[20] examined the association between magnesium dietary intake and the prevalence of metabolic syndrome. The ORs, from the second to the highest quintile of magnesium intake, were 0.84 (95% CI 0.58 to 1.23), 0.76 (95% CI 0.54 to 1.07), 0.62 (95% CI 0.40 to 0.98), and 0.56 (95% CI 0.34 to 0.92) with a p for trend=0.029. These authors concluded that there was an inverse association between dietary magnesium intake and the prevalence of metabolic syndrome.

Song *et al*[21] analyzed data from the Women's Health Study and reported that women in the highest quintile

Table 2 Characteristics of target population stratified by sex, N=247

	MHO			MUO		
	Women	Men		Women	Men	
N	96	28	P value	81	42	P value
Age, years	40.9±7.9	39.7±6.1	0.07	42.9±7.9	44.6±7.4	0.10*
Body mass index, kg/m^2	35.3±4.9	33.9±3.8	0.28	34.3±3.5	35.0±5.1	0.32
Waist circumference, cm	105.2±12.0	112.2±13.1	0.09	109.3±9.6	116.6±14.4	0.13*
Total body fat, %	45.2±4.4	34.5±5.4	0.01	44.6±5.3	37.8±8.2	0.04*§
Systolic blood pressure, mm Hg	110.8±8.9	113.5±8.4	0.12	116.2±14.2	123.2±9.4	0.03*†‡
Diastolic blood pressure, mm Hg	71.8±7.9	76.0±6.9	0.15	75.5±8.8	81.1±8.2	0.04*‡
High blood pressure, n (%)	6 (6.3)	1 (3.6)	0.94	27 (33.3)	23 (54.8)	0.03
Fasting glucose, mg/dL	93.6±8.3	95.7±6.2	0.24	107.9±17.6	104.4±13.7	0.81*†‡§
Hyperglycemia, n (%)	15 (15.6)	6 (21.4)	0.66	63 (77.8)	22 (52.4)	0.007
HDL-C, mg/dL	49.8±10.8	49.4±12.1	0.75	43.9±11.6	39.9±11.3	0.10*†‡
Low HDL-C, n (%)	14 (14.6)	2 (7.1)	0.47	37 (45.7)	26 (61.9)	0.13
Triglycerides, mg/dL	140.8±60.7	171.8±115.0	0.11	222.7±100.9	277.9±149.8	0.04*†‡§
Hypertriglyceridemia, n (%)	31 (32.3)	14 (50.0)	0.14	67 (82.7)	37 (88.1)	0.60
Magnesium intake, mg/day	330.8±137.4	345.4±127.1	0.85	238.9±90.3	276.0±96.8	0.06†‡§
Serum magnesium, mg/dL	2.0±0.21	1.8±0.23	0.09	1.6±0.26	1.8±0.20	0.10*†‡§

Values are mean±SD; p values estimated using one-way analysis of variance test with Games-Howell post hoc test.
*p<0.05 between metabolically healthy obese women and metabolically unhealthy obese men.
†p<0.05 between metabolically healthy obese men and metabolically unhealthy obese men.
‡p<0.05 between metabolically healthy obese women and metabolically unhealthy obese women.
§p<0.05 between metabolically healthy obese men and metabolically unhealthy obese women.
HDL-C, high-density lipoprotein cholesterol; MHO, metabolically healthy obese; MUO, metabolically unhealthy obese.

of magnesium intake had a 27% lower risk of metabolic syndrome. The ORs, from the second to the highest quintile of magnesium intake, were 0.91 (95% CI 0.78 to 1.06), 0.84 (95% CI 0.72 to 0.99), 0.81 (95% CI 0.68 to 0.96), and 0.73 (95% CI 0.60 to 0.88) with a p for trend=0.0008.

Finally, Ju *et al*[7] conducted a meta-analysis that included 30 092 participants enrolled in 10 observational studies, eight cross-sectional studies and two prospective cohort studies. The pooled relative risk of metabolic syndrome per 150 mg/day increment of magnesium intake was 0.88 (95% CI 0.84 to 0.93). They concluded that dietary magnesium intake was inversely associated with the risk of metabolic syndrome.

The results of the abovementioned studies highlight the important role that magnesium intake may play in metabolic parameters. In this regard, hypomagnesemia inhibits insulin pathways that are dependent on kinases and triggers an inflammatory response, both of which are involved in the development of glucose metabolism disorders.[22] Furthermore, by modifying the lipid composition of the membrane bilayer,[23] increasing the synthesis and release of lipids into circulation,[24] and decreasing activity of lecithin cholesterol acyltransferase, the magnesium deficiency is related with development of dyslipidemia. Finally, magnesium, which acts as a calcium channel antagonist by promoting the production of prostacyclins and nitric oxide and altering vascular responses to vasoactive agonists, impacts blood pressure.[25] Hence, it is likely that the appropriate magnesium intake is related to the absence of cardiovascular risk factors in MHO individuals.

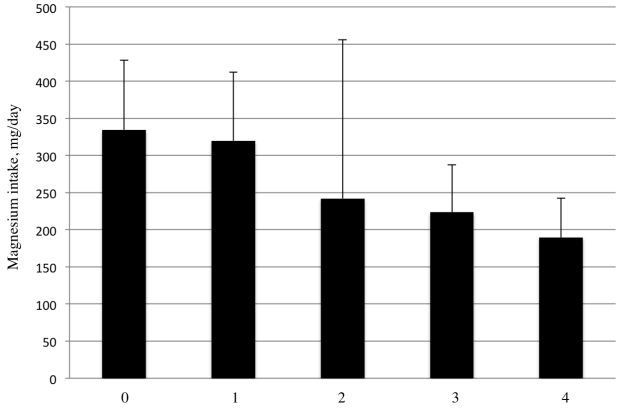

Figure 2 Magnesium intake as a function of metabolic risk factors number (X axis). Magnesium intake shows a decreasing trend as cardiovascular risk factors increase (p for trend=0.01).

Some limitations of our study should be noted. First, due to the nature of the study design, causality between magnesium intake and MHO phenotype cannot be assured with certainty. Second, we did not measure physical activity or gene expression. Taking into account that both MHO phenotype and magnesium status vary according ethnicity and physical activity, this limitation may influence our conclusions. Third, we assessed magnesium intake with a 24-hour recall questionnaire, which could be a potential source of bias. Given that changes in customary diet are unusual, this limitation has a minor effect on our conclusions. Fourth, we have no data regarding HbA1c levels. However, given that fasting glucose in the individuals of both groups were under the cut-off point for diagnosing diabetes, this limitation does not exert significant influence in our conclusions. Finally, to control for potential confounders, the logistic regression model was adjusted by all variables that showed significant differences in the bivariate analysis.

In conclusion, our results show that magnesium intake is associated with the MHO phenotype, suggesting that the benign state of MHO may be magnesium dependent.

Contributors FGR, GMG and LESM equally contributed to the conception and design of the research; LPR contributed to the design of the research; AGB and AIOM contributed to the interpretation of the data; and FGR and LESM drafted the manuscript. LESM is the guarantor. All authors critically revised the manuscript, agree to be fully accountable for ensuring the integrity and accuracy of the work, and read and approved the final manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement All data relevant to the study are included in the article or uploaded as supplementary information. The data used to support the results of our study are shown within the article.

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