

# Relationship between Serum Magnesium Level and Insulin Resistance in Obese Non-diabetic and Diabetic Patients

## Obez ve Obez Olmayan Diabetik Hastalarda İnsulin Direnci ile Serum Magnezyum Düzeyi İlişkisi

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### ABSTRACT

**Objective:** Type 2 diabetes mellitus (T2DM) and obesity are multifactorial diseases that include interactions between hereditary and environmental factors. Our study aimed to evaluate the relationship between serum magnesium (Mg) level and insulin resistance (HOMA-IR) in obese non-diabetic subjects and obese patients with T2DM who were compared with healthy controls.

**Methods:** The present study included 120 subjects of both genders (age, 20-70 years). The subjects were divided into four groups: Group I included 30 healthy subjects as control (8 males and 22 females); group II included 30 obese non-diabetic subjects (6 males and 24 females) with the body mass index (BMI)  $\geq 25$  kg/m<sup>2</sup>; group III included 30 (14 males and 16 females) obese patients with T2DM and disease history <1 year; and group IV included 30 (17 males and 13 females) obese patients with T2DM and disease history >5 years. Endocrinology and metabolism specialists diagnosed T2DM patients. Serum Mg, fasting glucose, fasting insulin, and fasting lipids were measured including the patients' weight and height. In addition, BMI and HOMA-IR were calculated.

**Results:** Serum Mg level significantly decreased in group IV (1.72 $\pm$ 0.1 mg/dL) compared with group I (2.07 $\pm$ 0.1 mg/dL;  $p < 0.05$ ). HOMA-IR significantly increased in group IV (7.9 $\pm$ 7.0) compared with group I (1.03 $\pm$ 0.3;  $p < 0.05$ ). In addition, serum fasting glucose, serum insulin, and fasting lipids were significantly higher in case groups than in the control group. The serum Mg level was inversely associated with age in all case groups. In group IV, a negative significant correlation was found between serum Mg level and age and HOMA-IR ( $p < 0.01$ ).

**Conclusion:** A low serum Mg level was found in obese patients with T2DM and obese non-diabetic subjects, whereas a high HOMA-IR level was found in obese patients with T2DM and obese non-diabetic subjects. Obese patients with T2DM show a negative correlation between the serum Mg level with HOMA-IR and age. We recommend measuring the serum Mg level regularly in obese patients with T2DM, especially in elderly patients, and patients who require supplementation.

**Keywords:** Type 2 diabetes mellitus, obesity, insulin resistance, magnesium

### ÖZ

**Amaç:** Tip 2 diabetes mellitus (T2DM) ve obezite kalıtsal ve çevresel faktörler arasındaki etkileşimler de dahil olmak üzere multifaktöriyel hastalıklardır. Çalışmamızın amacı diyabetik olmayan kişilerde ve obez T2DM hastalarda serum Magnezyum (Mg) düzeyleri ile insülin direnci (HOMA-IR) arasındaki ilişkiyi değerlendirmektir.

**Yöntemler:** Çalışmamız her iki cinsiyetten (20-70) yaş aralığında olan 120 denekten oluşmaktadır. Denekler dört gruba ayrıldı: Grup I kontrol olarak 30 sağlıklı bireyi (8 erkek 22 kadın) içeriyordu. Grup II, BMI  $\geq 25$  kg/m<sup>2</sup> olan 30 obez diyabetik olmayan (6 erkek 24 kadın) denekten oluşmaktaydı. Grup III, hastalık öyküsü bir yıldan az olan 30 (14 erkek 16 kadın) obez T2DM hastasını ve beşinci yıldan fazla hastalığa sahip 30 (17 erkek ve 13 kadın) obez T2DM'yi grup IV'te gruplandırdı. T2DM hastalarının tanı endokrinoloji ve metabolizma uzmanları tarafından yapıldı. Serum Mg, açlık glikozu, açlık insülini ve açlık lipidleri ölçüldü, ağırlık, boy ölçüldü. Ek olarak vücut kitle indeksi (VKI) ve HOMA-IR hesaplandı.

**Bulgular:** Serum Mg düzeyi grup I'de (1,72 $\pm$ 0,1mg/dL) grup I'e (2,07 $\pm$ 0,1mg/dL) göre anlamlı şekilde düşüktü ( $p < 0,05$ ). HOMA-IR, grup I'de (1,03 $\pm$ 0,3), grup IV'te (7,9 $\pm$ 7,0) anlamlı olarak fazla idi. ( $p < 0,05$ ) Ayrıca serum açlık glikozu, serum insülin ve açlık lipidleri hasta gruplarında kontrol grubuna göre anlamlı olarak daha yüksekti. Serum Mg düzeyi, tüm olgu gruplarında yaşla ters orantılı idi. Grup IV'de serum Mg düzeyleri ile yaş ve HOMA-IR arasında negatif bir korelasyon vardı ( $p < 0,01$ ).

**Sonuç:** Obez T2DM'li hastalarda ve diyabetik olmayan obezlerde serum Mg düzeylerinde düşük bulunurken, obez T2DM'li hastalarda ve diyabetik olmayan obezlerde yüksek düzeyde HOMA-IR tespit edildi. Obez T2DM hastaları serum Mg düzeyleri ile HOMA-IR ve yaş arasında negatif korelasyon göstermektedir. Özellikle yaşlı obez T2DM hastaları olmak üzere obez T2DM li hastalarda serum Mg düzeyi ölçümü yapılmasını eksiklik saptanan hastalarda replasmanın yapılmasının akılda tutulmasını önermekteyiz.

**Anahtar kelimeler:** Tip 2 Diabetes mellitus, obezite, insülin direnci, magnezyum

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## INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a cause of an increasing morbidity and mortality worldwide. T2DM is characterized by hyperglycemia caused by disabled insulin activity in target tissues, such as liver, muscle, and fat tissue (insulin resistance [IR]), and impaired insulin secretion through progressive beta cell dysfunction (1). An increased incidence of obesity has taken a spotlight in the world, and recent epidemiological studies have recorded a rapid increase in its prevalence among all ages, genders, and racial/ethnic groups (2). IR is the main pathogenic factor of several metabolic disorders, including T2DM and obesity, and is a significant cause of cardiovascular disease and early death. IR is an important link between obesity and T2DM (3). Magnesium ( $Mg^{2+}$ ) is a primary cofactor required for many biochemical reactions and plays an essential role in glucose metabolism. It is essential for insulin action since it is a cofactor of tyrosine kinase activity. Several studies have shown that T2DM is associated with Mg depletion. A decreased intracellular Mg concentration can lead to increased IR in diabetic patients. An increased incidence of Mg depletion was identified in T2DM patients, particularly in patients with a long history of diabetes and uncontrolled glycaemic profiles. Hypomagnesaemia can be both an outcome and a reason of diabetic complications (4-6). Several studies have shown that decreased intracellular Mg leads to increased IR in diabetic patients (7). The aim of this study was to evaluate the relationship between the serum Mg level and IR in obese non-diabetic subjects and obese patients with T2DM, who were compared with healthy controls.

## METHODS

### Ethical Aspects

The clinical research ethics committee of Gaziantep University School of Medicine, accepted the study protocol on 08/15/2016, approval no. 2016/237. In addition, all subjects who participated in this study provided written informed consent.

### Subjects And Study Design

The present study included 120 subjects of both genders and aged between 20 and 70 years. The subjects were divided into four groups. Group I included 30 (8 males and 22 females) healthy subjects as control. Group II included 30 (6 males and 24 females) obese non-diabetic subjects with a body mass index (BMI)  $\geq 25$  kg/m<sup>2</sup>. Group III included 30 (14 males and 16 females) obese patients with T2DM and disease history <1 year, and group IV included 30 (17 males and 13 females) obese patients with T2DM and disease history >5 years. Endocrinology and metabolism specialists diagnosed the T2DM patients.

A written consent was obtained from all the participants in this study. Baseline data included basic demographics: age, gender, weight, and height were measured; BMI was calculated as kg/m<sup>2</sup>; and hypertension, cigarette smoking, and medical and family history were recorded. In addition, all subjects in this study were clinically examined by expert doctors to determine the presence of exclusion criteria that included pregnancy, patients taking magnesium supplementation, alcohol consumption, cardiovascular disease, chronic disorders, and malignancy.

### Clinical And Laboratory Evaluation

Fasting, venous blood samples were collected from all the participants. Collected samples were centrifuged, and serum was separated and then stored at -80°C.

All samples were collected for estimation and analytical measurement of biochemical parameters for serum magnesium, serum fasting glucose, total cholesterol, LDL-cholesterol, and triglycerides by the photometric enzymatic procedure using clinical chemistry laboratory instrument (Beckman Coulter, Model Au5800, Tokyo, Japan), and the lab test kits were used for each parameter. The assay of serum insulin was based on the chemiluminescent immunoassay method using the clinical laboratory instrument (Beckman Coulter, Dxl 800 Tokyo, Japan) and lab test kits for Access Ultrasensitive Insulin. The degree of insulin resistance (IR) was determined for all study subjects by using homeostasis model of assessment (HOMA) method. The index (HOMA-IR) was calculated by the following formula: fasting serum glucose (mg/dl)  $\times$  fasting serum insulin ( $\mu$ U/mL)/405.

### Statistical Analysis

Descriptive statistical parameters were presented as mean  $\pm$  standard derivation (mean  $\pm$  SD). Demographic and clinical biochemical data among the groups were compared with one-way analysis of variance using the SPSS (Statistical Package for Social Sciences) Version 16.0 (SPSS Inc.; Chicago, IL, USA). Duncan's multiple range tests were used to distinguish the examined groups. The p value of <0.05 was considered as statistically significant.

## RESULTS

The demographic and clinical biochemical laboratory data of the studied groups are given in Table 1.

With regard to the age of groups, a significant difference was found between the obese patients with diabetes history >5 years and healthy group ( $p < 0.05$ ). However, there was no significant difference between the obese patients with diabetes history >5 years and obese patients with diabetes history <1 year. In addition, this study found a negative statically significant association between the serum Mg level and age ( $r = -0.88$ ,  $p < 0.01$ ) in obese patients with diabetes history >5 years, as shown in Table 2.

HOMA-IR had a negative correlation with serum Mg levels in obese patients with disease history >5 years, but the association was not significant (Table 3).

## DISCUSSION

$Mg^{2+}$  is a primary cofactor required for many biochemical reactions.  $Mg^{2+}$  is an important factor in glucose metabolism, and it is necessary for glucose transportation between the membranes, glucose oxidation, reactions involving phosphorylation, and energy exchange. It is essential for insulin action since it is a cofactor of tyrosine kinase activity (8).

The present study revealed that the serum Mg level in obese non-diabetic subjects was lower (1.98 mg/dL) than in the healthy control group (2.02 mg/dL). These results are in agreement with

**Table 1.** Demographic and clinical biochemical data of studied groups. Group I is control, group II is obese non-diabetic, group III is obese T2DM with disease history <1 year, and group IV is obese T2DM with disease history >5 years. N indicates the number of subjects

		Healthy	Obese Non-DM	Obese DM <1y	Obese DM >5y
N		30	30	30	30
Male/Female		8/22	6/24	14/16	17/13
Parameters	Unit	Mean±SD	Mean±SD	Mean±SD	Mean±SD
Age	year	36.0±10.6 <sup>a</sup>	44.4±9.7 <sup>b</sup>	52.7±9.9 <sup>c</sup>	55.7±8.3 <sup>c</sup>
Height	cm	164.5±5.4 <sup>a</sup>	161.7±6.0 <sup>a</sup>	163.5±6.2 <sup>a</sup>	169.4±3.7 <sup>b</sup>
Weight	kg	63.56±5.4 <sup>a</sup>	87.03±7.8 <sup>b</sup>	88.66±9.5 <sup>b</sup>	97.03±8.8 <sup>c</sup>
BMI	kg/m <sup>2</sup>	19.06±1.6 <sup>a</sup>	27.28±3.1 <sup>b</sup>	26.95±3.2 <sup>b</sup>	28.7±2.3 <sup>c</sup>
Mg	mg/dL	2.07±0.1 <sup>c</sup>	1.98±0.2 <sup>b</sup>	1.98±0.2 <sup>b</sup>	1.72±0.1 <sup>a</sup>
FG	mg/dL	91±4.4 <sup>a</sup>	101.8±8.2 <sup>a</sup>	140.86±44.5 <sup>b</sup>	202.8±42.4 <sup>c</sup>
Insulin	μU/mL	7.9±3.1 <sup>a</sup>	11.39±9.0 <sup>a,b</sup>	14.81±9.2 <sup>b,c</sup>	16.7±7.0 <sup>c</sup>
HOMA-IR		1.03±0.3 <sup>a</sup>	2.82±2.3 <sup>b</sup>	4.99±4.1 <sup>c</sup>	7.9±3.7 <sup>d</sup>
TC	mg/dL	169.0±34.2 <sup>a</sup>	204.8±48.5 <sup>b</sup>	198.8±33.3 <sup>b</sup>	195.7±27.0 <sup>b</sup>
TG	mg/dL	144.1±43.8 <sup>a</sup>	179.1±69.5 <sup>b</sup>	170.7±63.4 <sup>a,b</sup>	201.6±74.9 <sup>b</sup>
LDL-C	mg/dL	90.2±21.7 <sup>a</sup>	111.4±44.4 <sup>b</sup>	105.6±24.2 <sup>a,b</sup>	102.1±23.7 <sup>a,b</sup>

\*BMI: Body mass index; Mg: Magnesium; FG: Fasting glucose; TC: Total cholesterol; TG: Triglyceride; LDL-C: Low-density lipoprotein-cholesterol; HOMA-IR: Homeostasis model assessment of insulin resistance

\*\*Different lowercase letters indicate statistical difference at α=0.05 level among the groups. Values with the same letters in the same parameters indicate that the values did not differ by the Duncan test at 0.95 confidence interval

**Table 2.** Correlation between the serum Mg level with anthropometric measurement and biochemistry parameters in all groups

Parameters		Healthy		Obese Non-DM		Obese DM 1–5 years		Obese DM >5 years	
		r	p	r	p	r	p	r	p
Age	year	-0.057	0.764	-0.033	0.862	-0.673	0.000	-0.886	0.000
Height	cm	-0.150	0.430	0.002	0.991	0.118	0.534	0.120	0.528
Weight	kg	-0.16	0.382	0.039	0.836	0.003	0.987	-0.084	0.657
BMI	kg/m <sup>2</sup>	-0.071	0.713	0.055	0.772	-0.169	0.369	-0.057	0.765
Glucose	mg/dL	-0.097	0.612	-0.190	0.315	-0.055	0.770	-0.317	0.088
Insulin	μU/mL	-0.190	0.314	0.128	0.500	0.104	0.581	0.128	0.499
HOMA-IR	mg/dL	-0.192	0.310	0.121	0.524	0.149	0.429	-0.099	0.604
Cholesterol	mg/dL	0.128	0.499	0.107	0.575	-0.112	0.552	-0.169	0.373
Triglyceride	mg/dL	0.016	0.943	-0.157	0.406	-0.027	0.884	-0.200	0.290
LDL	mg/dL	-0.290	0.120	0.080	0.673	-0.031	0.870	-0.235	0.210

\*r: Pearson correlation coefficient; p<0.05 is significant

\*\*BMI: Body mass index; Mg: Magnesium; FG: fasting glucose; TC: Total cholesterol; TG: Triglyceride; LDL-C: Low-density lipoprotein-cholesterol; HOMA-IR: Homeostasis model assessment of insulin resistance

previous studies (9, 10). Additionally, another study found a low serum Mg level in overweight or obese patients (11). Moreover, as the present study revealed, the serum Mg level was decreased in obese patients with T2DM, especially in obese patients having T2DM >5 years. Previous studies also reported that serum

Mg decreased in T2DM (12, 13). This indicates the association of hypomagnesaemia with T2DM. In addition, the present study indicates that the serum Mg level in obese patients (1.72 mg/dL) with diabetes history >5 years was significantly lower than in obese patients (1.98 mg/dL) with diabetes history <1 year. This

**Table 3.** Correlation between calculated HOMA-IR with anthropometric measurement and biochemistry parameters in all groups

Parameters		Healthy		Obese Non-DM		Obese DM 1–5 years		Obese DM >5 years	
		r	p	r	p	r	p	r	p
Age	year	-0.126	0.504	-0.147	0.437	-0.133	0.48	0.188	0.319
Height	cm	0.191	0.310	-0.19	0.302	-0.097	0.608	-0.250	0.182
Weight	kg	0.351	0.056	-0.124	0.513	-0.035	0.853	-0.086	0.650
BMI	kg/m <sup>2</sup>	0.151	0.424	0.138	0.464	-0.015	0.936	-0.128	0.499
Glucose	mg/dL	0.364	0.040	0.195	0.301	0.355	0.053	0.412	0.020
Insulin	μU/mL	0.990	0.000	0.991	0.000	0.881	0.000	0.818	0.000
Mg	mg/dL	-0.191	0.309	0.121	0.523	0.149	0.429	-0.098	0.604
Cholesterol	mg/dL	0.082	0.665	0.432	0.017	0.024	0.897	0.194	0.303
Triglyceride	mg/dL	0.449	0.012	0.539	0.002	0.007	0.966	-0.097	0.609
LDL-C	mg/dL	0.047	0.804	0.561	0.001	-0.235	0.209	-0.085	0.653

\*r: pearson correlation coefficient, p<0.05 is significant

\*\*BMI: Body mass index; Mg: Magnesium; FG: Fasting glucose; TC: Total cholesterol; TG: Triglyceride; LDL-C: Low-density lipoprotein-cholesterol; HOMA-IR: Homeostasis model assessment of insulin resistance

was in compliance with several other studies that reported an elevated Mg deficiency in T2DM patients with longer duration of diabetes and uncontrolled glycemic profiles (14-15).

Hypomagnesaemia can be both a consequence and a cause of diabetic complications. The causes of hypomagnesaemia in patients with T2DM are not clear, but they may consist of poorer dietary consumption of Mg, decreased intestinal Mg absorption, increased urinary loss of Mg, or decreased Mg uptake into cells compared with that in healthy individuals.

In addition, with respect to age, a significant difference was found between obese patients with diabetes history >5 years and the healthy group (p<0.05). However, there was no significant difference between obese patients with diabetes history >5 years and those with diabetes history <1 year. As this study found, in obese patients having T2DM for >5 years, there was a negative statically significant association between the serum Mg level and age (r=-0.88, p<0.01). The elderly may be susceptible to having a low serum Mg level because aging is related with decreased intracellular Mg levels. In addition, the elderly are unable to profit from Mg-rich foods because of their hard texture and unsuitable physical properties.

A negative correlation between serum Mg levels and serum fasting lipids was found in patients with T2DM for >5 years, but the association was not significant. This finding was in agreement with a study by Elementol et al. (16), which reported no statistically significant effect of Mg concentration on the content of lipids analyzed in blood serum. Noticeably, obesity may increase the cardiovascular risk and the mortality rate related to low Mg levels.

Moreover, in the present study, HOMA-IR had a negative correlation with serum Mg levels in patients with T2DM for >5 years, but the association was not significant. This finding was in agree-

ment with Lima et al. (17), who found a negative association between HOMA-IR and serum Mg level in patients with T2DM, although it was not statistically significant.

The present study reported low serum Mg levels in obese patients with T2DM and obese non-diabetic subjects, while a high level of HOMA-IR was found in obese patients with T2DM and obese non-diabetic subjects. Obese patients with T2DM showed a negative correlation between the serum Mg level with HOMA-IR and age. The association between serum Mg level and IR may be another risk factor for uncontrolled diabetes and diabetic complications. Preservation of the normal levels of serum Mg may prove to be useful in the prevention of diabetic complications.

**CONCLUSION**

Consequently, we recommend weight loss in obese patients and regular measurement of serum Mg levels in obese patients with T2DM, especially in elderly patients and patients who require supplementation should be considered.

**Ethics Committee Approval:** Ethics committee approval was received for this study from the ethics committee of Gaziantep University School of Medicine (Decision date: 08.15.2016/Decision no: 2016/237).

**Informed Consent:** Written informed consent was obtained from patients who participated in this study.

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## REFERENCES

1. Kahn SE. The relative contributions of insulin resistance and beta cell dysfunction to the pathophysiology of Type 2 diabetes. *Diabet Review* 2003; 46: 3-19.
2. Sarkar A, Dash S, Barik BK, Muttigi MS, Kedage V, Shetty JK, et al. Copper and Ceruloplasmin levels in relation to total thiols and GST in type2 diabetes mellitus patients. *Ind j Clin Biochem* 2010; 25: 74-6.
3. Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE. Global estimates of diabetes prevalence for 2013 and projections for 2035. *Diabet research and clinical practice*. 2014; 103: 137-49.
4. Baskin ML, Ard J, Franklin F, Allison DB. Prevalence of obesity in the United States. *Obesity Reviews* 2005; 6: 1-88.
5. DeFronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* 1991; 14: 173-94.
6. Tracey L, Mclaughlin MD, Gerald M, Reaven MD. Beyond type2 Diabetes: The need for a clinically useful way to identify insulin resistance. *The American Journal of Medicine* 2003; 114: 501-2.
7. Pham PC, Pham PM, Pham SV, Miller JM, Pham PT. Hypomagnese-mia in patients with type 2 diabetes. *Clin J Am Soc Nephrol* 2007; 2: 366-73.
8. Barbagallo M, Dominguez LJ. Magnesium metabolism in type 2 diabetes mellitus, metabolic syndrome and insulin resistance. *Arch Biochem Biophys* 2007; 458: 40-7.
9. Cahill F, Shahidi M, Shea J, Wadden D, GulliverW, Randell E, et al. High dietary magnesium intake is associated with low insulin resistance in the Newfoundland population. *Plos ONE* 2013; 8: 1-8.
10. Hedberg J, Haenni A. Increased plasma magnesium concentrations 3 years after biliopancreatic diversion with duodenal switch. *Obes Surg* 2012; 22: 1708-13.
11. Guerrero-Romero F, Rodríguez-Morán M. Serum magnesium in the metabolically-obese normal-weight and healthy-obese subjects. *Eur J Intern Med* 2013; 24: 639-43.
12. Naheed T, Khan A, Masood G, Yunus BB, Chaudhry MA. Dyslipid-emias in type II diabetes mellitus patients in a teaching hospital of Lahore Pakistan. *Pak J Med Sci* 2003; 19: 283-6.
13. Rasic-Milutinovic Z, Perunicic-Pekovic G, Pljexa S, Dangic A, Dangic A. Magnesium deficiency in type 2 diabetes. *Hippokratia* 2004; 8: 179-81.
14. Augusta CN, Chinyere A, Opara U, Maisie HE, Isonguyo NU. Influence of Age, Gender and Duration of Diabetes on Serum and Urine Levels of Zinc, Magnesium, Selenium and Chromium in Type 2 Diabetics in Calabar, Nigeria. *Turkish Journal of Biochemistry-Turk J Biochem* 2006; 31: 107-14.
15. Del Gobbo LC, Song Y, Poirier P, Dewailly E, Elin RJ, Egeland GM. Low serum magnesium concentrations are associated with a high prevalence of premature ventricular complexes in obese adults with type2 diabetes. *Cardiovasc Diabetol* 2012; 11: 23.
16. Elementol J, et al. Evaluation of the correlations between magnesium concentration and selected serum lipid components in women and men of different age with chronic kidney failure. 2010; 15: 321-9.
17. Lima ML, Judith PJ, Barbosa C, Bras TC. Magnesium deficiency and insulin resistance in patients with type2 diabetes mellitus. *Endocrinol Metab* 2005; 49: 959-63.

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