

Nutritional Status and Magnesium Levels in Serum and Urine of Primary Hypertensives and Normotensives†

KADRIYE KAYAKIRILMAZ^{1,*}, EMEL BAYOL¹, HATICE BAL YILMAZ² and SERVET ARIOGUL³

¹Department of Chemistry, Faculty of Sciences and Arts, Nigde University, 51200 Nigde, Turkey ²School of Nursing, Ege University, Izmir, Turkey ³Faculty of Medicine, Hacettepe University, Ankara, Turkey

*Corresponding author: Fax: +90 388 2250180; Tel: +90 388 2252213; E-mail: kkayakirilmaz@nigde.edu.tr

Received:	23 August 20	10;

Accepted: 28 January 2011)

AJC-9519

This research has been designed for the determination of the nutritional status, serum and urinary magnesium (UMg) levels and correlation between urinary magnesium and blood pressure (BP). Thirty two volunteers' patients with essential hypertension (HT) and 35 normotensive (NT) control subjects, aged 21-79 years, have participated in this study. Fifty five subjects' 24-h urinary samples were collected (28NT and 27HT). Besides, for determination of the correlation between 24 hUMgV and overnight excretion rate urinary magnesium (12 hUMgV), 43 urine samples were collected. Nutritional status was evaluated according to their food consumption and physical examination. After analyzing the collected blood and urinary samples, the mineral status was also evaluated. Among 13 hypertensive subjects three women had serum magnesium values lower than 1.7 mg Mg/dL (1.38-1.68 %). After 6 months treatment with magnesium, serum magnesium concentrations have increased to normal levels. In both groups blood pressure was positively correlated with UMgV and in hypertension group blood pressure was negatively correlated with serum magnesium levels (p < 0.05). Among the 43 subjects, 12 h UMgV was correlated with the 24 h UMgV and also in normotensive group UMgV was correlated positively with UCaV (p < 0.05). Overnight urinary magnesium excretion rate was correlated with the 24 h UMgV, so 12 h UMgV should be usually employable.

Key Words: Serum magnesium, Urinary magnesium excretion, Atomic absorption spectrometry, Food consumption.

INTRODUCTION

Hypertension is an important disease of modern civilized life. The overall prevalence of hypertension has been reported to range from 21.9 to 42.3 % in 65-114 years old persons living in Ankara (Turkey)¹, from 6 to 32 % in Australian population². Much epidemiologic data confirm the relevance of nutritional factors in determining blood pressure (BP). Factors epidemiologically related to BP such as nutritional status^{3,4}, weight, calorific intake and the minerals sodium, potassium, calcium and magnesium also have been the focus of therapeutic intervention trials. These trials have shown that lowering dietary calorie and/or Na (salt) content and providing increased amounts of Ca, K or Mg may lower BP in at least some sensitive subjects⁵.

Essential hypertension (EH) or primary hypertension (PH or HT) refers to lasting increase in BP with heterogeneous genetic and environmental causes. Its prevalence rises with age, irrespective of the type of BP measurement and the operational thresholds used for diagnosis. The specific causes of essential hypertension remain incompletely understood. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) thresholds used in clinical medicine are boundaries in normotension: optimum < 120/< 80, normal 120-129/80-84, high-normal 130-139/85-89, in hypertension: grade I (mild) 140-159/90-99, subgroup borderline 140-149/90-94, grade II (moderate)160-179/100-109, grade III (severe) >180/>110, isolated systolic hypertension >140/<90, subgroup borderline 140-149/<90 mm Hg⁶.

The adult human body contains *ca.* 24 g (1 mol or 2 Eq, range 20 to 28 g) Mg, about 0.3 % of the total body Mg is present in serum, yet the majority of analytical data obtained is from this body fluid^{7.8}. Over 300 enzyme reactions are dependent on Mg. Magnesium plays a major role in control in cardiac excitability, neuromuscular transmission, vasomotor tone, hypertension, BP control, metabolic syndrome and diabetes

*Partially presented as an oral presentation at the 3rd International Congress on Food and Nutrition, April 22-25, 2009 in Antalya and at the XX. National Chemical Congress September 04-08, 2006 Kayseri, Turkey.

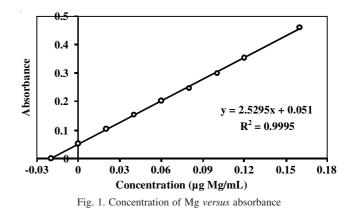
mellitus. Changes in Mg status have been linked to cardiac arrhythmias, coronary heart disease, hypertension and premenstrual syndrome⁹. Magnesium intake varies greatly because of the widely variable content of different foods. The recommended daily allowance (RDA) for magnesium is 300 and 350 mg (25 to 29 mEq) for adult women and men, respectively¹⁰. A number of physiological factors influence normal adsorption³. Magnesium is a dietary source found in fruits, vegetables, leguminouses, dairy products, cereals and certain grains. In a normal adequate diet *ca*. 30 % of the total Mg intake may come from green vegetables that contain the magnesium porphyrin, because Mg is an essential component of chlorophyll. Cereals and vegetables normally contribute more than twothirds of the daily Mg intake¹¹.

Assessing the Mg status of an individual is difficult and simple excretion tests have become increasingly popular¹². In the present investigation, the relationships among the blood pressure and nutritional status, serum and urinary Mg levels were studied in essential hypertensive patients and normotensive subjects. Besides the purpose of this article was also investigated the correlation between overnight urinary Mg excretion rate and 24 h urinary Mg excretion rate.

EXPERIMENTAL

Thirty two volunteers' primary hypertensive patients (HT) (22 females and 10 males, mean resting BP \geq 140/90 mmHg and with medically diagnosed hypertension and 35 normotensive (NT) (20 females and 15 males, blood pressure < 140/ 90 mmHg) from Hacettepe University internal diseases polyclinic, asylum for the aged of Keçiören (Ankara) and Nigde University, aged 21-79 years of old participated in the first step of research. During the first step of research at same time daily energy, Mg and other nutrient intakes were determined and 24 h urine were collected. On the following day, overnight urines samples were collected by 43 subjects from these groups. Physicians from Hacettepe University, asylum for the aged and Nigde State Hospital examined all the subjects' health that joined at the first and second steps of research. Detailed informations about anthropometrics and blood pressure measurements, dietary survey and blood and urine sample collection were published earlier¹³. Urine sample (50 μ L) was diluted 200 fold with 0.2 % La (prepared with La₂O₃ + HCl), mixed well and then centrifuged at 3500 rpm for 10 min. Bread, tea and water samples were prepared (digested with 1 V HClO₄ $+ 3 V H_2O_2$) for analysis using the method of wet digestion. Magnesium concentrations of bread, tea, water, serum and urine, were determined by flame atomic absorption spectrophotometer according to the method of Perkin-Elmer using

the method of standard additions, as described elsewhere^{14,15}. The added standards of different Mg concentrations (*i.e.*, 0.02, 0.04, 0.06, 0.08, 0.10, 0.12 and 0.16 μ g Mg/mL) were prepared from stock standard. The standards and samples were read against the blank solution (0.2 % La). The concentration of Mg in the samples was calculated by reading from the standard curve¹⁵ (Fig. 1).



Data analysis: Daily dietary intakes of Mg were compared with recommended daily allowances (RDA) for Turkish people¹⁰ using following equation:

$$\frac{X_1 - A}{\frac{SD_1}{\sqrt{n}}}$$

where A = RDA, $X_1 = mean$, $SD_1 = standard deviation$.

Data differences were compared by unpaired Student's t-tests or Mann-Whitney U test for hypertensive *versus* normotensive subjects¹⁶. The relations of blood pressure to other variables were analyzed by linear regression and Person correlation coefficients. Data are expressed as the mean ± SD.

RESULTS AND DISCUSSION

Sixty seven persons aged 21-79 years were participated in this study. We tested the hypothesis that the magnesiuria of hypertensives is due to dietary factors and body mass index (MBI). Biochemical data for the 67 subjects (35 NT and 32 HT) and UMg excretion rate for 55 subjects are displayed in Tables 1 and 2. Great majority of groups fall within 30-60 years¹⁷.

As it can be seen in Table-1, diastolic blood pressure, systolic blood pressure, 24 h UMg and daily dietary Mg intake values in hypertensive females were statistically found to be different from those of normotensive females (p < 0.01). Similar results about BP was obtained for males (p < 0.01, Table-2).

BMI, BLOOD PRESSURE	E AND DIET	ARY AND U	TABLE-1 JRINARY Mg	VALUES OF	THE FEMA	LE VOLUNTE	EERS $(n = 42)$	
	Nor	motensive (r	n:20)	Ну	pertensive (n	Statistical analysis		
variables –	X^1	SD^1	Range	X^1	SD^1	Range	Z	р
BMI (kg/m ²)	23	3	19-28	28	4	23-38	-3.820	< 0.01
Diasystolic blood pressure (mmHg)	76	6	67-85	95	13	85-140	-4.200	< 0.01
Systolic blood pressure (mmHg)	120	13	100-135	162	16	140-210	-4.999	< 0.01
Dietary Mg (mg/day)	376 ^a	90	280-545	312	133	157-695	-2.088	< 0.05
Urinary Mg rate (mg/day) (24 h)	67 ^b	27	21-110	97°	33	56-178	-2.494	< 0.05

 X^{1} = Mean, SD¹ = Standard deviation; ^aFor dietary Mg n = 15, ^bfor UMgV n = 17, ^cfor UMgV n = 18.

BMI, BLOOD PRESSUR	RE AND DIE	TARY AND	TABLE-2 URINARY Mg	g VALUES O	F THE MAL	E VOLUNTEE	ERS (n = 25)	
Variables -	Noi	rmotensive (1	n:15)	Ну	pertensive (r	Statistical analysis		
variables -	\mathbf{X}^{1}	SD^1	Range	\mathbf{X}^{1}	SD^1	Range	Z	р
BMI (kg/m ²)	23	4	17-32	24	5	20-35	-0.250	ns
Diasystolic blood pressure (mmHg)	77	6	67-85	89	7	80-100	-3.397	< 0.01
Systolic blood pressure (mmHg)	124	10	106-135	156	19	120-180	-3.424	< 0.01
Dietary Mg (mg/day)	373 ^a	112	220-560	328 ^b	124	191-478	-0.723	ns
Urinary Mg rate (mg/day) (24 h)	79°	43	34-169	97 ^d	43	52-178	-1.179	ns
X^{1} = Mean, SD^{1} = Standard deviation; ^a for dietary Mg n = 11, ^b for dietary Mg n = 9, ^c for UMgV n = 11, ^d for UMgV n = 9.								

higher levels of UMgV than normotensive persons. The urinary Mg excretion is 3.0-5.0 mmol/day (72-120 mg); values for males are slightly higher than those for females and are usually expressed as a 24 h excretion rate⁸. The excretion depends on many variables including dietary intake of Mg and many diseases (renal disorders, diabetic ketoacidosis, hypertension). Excluding gender, the mean 24 h UMgV levels in hypertensive and normotensive subjects were $97 \pm 36 \text{ mg/d}$ (or 4.04 ± 1.5 mmol/d) and 71 \pm 35 mg/d (or 2.96 \pm 1.46 mmol/d), respectively $(n_{NT} = 28, n_{HT} = 27, t = 2.72, p < 0.01, data not shown in$ tables). Including gender, the highest values of urinary Mg concentration were recorded in hypertensive subjects for female group $97 \pm 33 \text{ mg/d}$ (or $4.04 \pm 1.38 \text{ mmol/d}$), for male group $97 \pm 43 \text{ mg/d}$ (or $4.04 \pm 1.79 \text{ mmol/d}$) and in normotensive subjects for female group 67 \pm 27 mg/d (or 2.79 \pm 1.12 mmol/d), for male group 79 ± 43 mg/d (or 3.29 ± 1.79 mmol/d). The amounts of UMgV were varied greatly from person to person (range: 21-178 mg/d); this make interpretation of results of these assays difficult (Tables 1 and 2). According to present results urinary Mg excretion is more dependants to hypertension than to age in female. Delva et al.¹⁸ found that urinary Mg for normotensive control subjects 2.8 ± 0.7 mmol/ 24 h and for patients with primary aldosteronism 2.7 ± 1.3 mmol/24 and our results coincide with those reported their results. Sacks et al.¹⁹ found mean UMg 4.1 ± 1.2 mmol/24 h in a normotensive women group (mean age 39 years), which is not similar to present study. Šimecková et al.²⁰ found that excluding diabetic patients, the first morning urinary Mg concentration was age-dependent, the highest value of urinary Mg concentration was recorded in 4-year-old children 6.64 mmol/L and declines to 4 mmol/L in the group of 18-65 years and 2.35 mmol/L in the group of seniors aged 86-93 years. Although hypertensive subjects were more aged than normotensive subjects present results not well coincide with those reported by Šimecková et al.20.

Patients with hypertension have been reported to have

For daily urinary Mg excretion similar findings were described by Restnic *et al.*⁵. Present UMgV levels of HT groups agree with results obtained with CCNW diet. Most foods contain useful amounts, particularly those of vegetable origin. Cereals and vegetables between them normally contribute more than two-thirds of the daily Mg intake¹¹. Fortunately cereals, vegetables and grain is consumed largely in Turkey, probably Turkish diet supply adequate Mg and conform to CCNW diet. Zozaya *et al.*²¹ have also reported¹³ an increase in Mg excretion in the 24 h urine collections of the hypertensive group. Same authors have also reported an increase in 24 h urinary Ca excretion in the hypertensive group. In a recent paper, we reported on male subjects, the NT group showed higher 24 h

UCaV (153 \pm 83 mg/d) than HT group (105 \pm 25 mg/d, p < 0.05).

However in males, the difference between 24 h UMgV in the NT (79 \pm 43 mg/d) and HT subjects (97 \pm 43 mg/d) was not statistically significant (p > 0.01, Table-1). While, in female subjects the HT group showed higher 24 h UMgV (97 \pm 33 mg/d) than NT group (67 ± 27 mg/d, p < 0.01, Table-2). Results of Tillman and Semple²² differ from present studies. Because they have reported that urinary excretion of Mg was significantly lower in hypertensive $(3.7 \pm 1.3 \text{ mmol}/24 \text{ h})$ than control $(4.5 \pm 1.6 \text{ mmol}/24 \text{ h})$ subjects and there was an inverse correlation between Mg excretion and blood pressure (r =0.3-0.35, p < 0.01). Nicoll et al.¹² reported Mg in urine specimens collected three ways all subjects were apparently healthy, ages 20 to 45 years and maintained their usual life-styles throughout. Magnesium concentrations were 3.71 mmol/L (n = 16), 3.34 mmol/L (n = 12) and 2.68 mmol/L (n = 12) of first morning urine, randomly collected urine and 24 h collections of urine, respectively and 24 h urine Mg output was 3.65 mmol/d (n = 12). Motoyama *et al.*²³ demonstrated that when patients (mild moderate essential hypertension and normal renal function, ages 30-56 years) were given during the 4 weeks of Mg supplementation with 1 g MgO/day (600 mg Mg daily); mean blood pressure decreased from 111 ± 6 mm Hg to $102 \pm$ 6 mm Hg and UMgV excretion was significantly increased from 7.411 meq/day (3.71 mmol/d) to 10.65 meq/day (5.33 mmol/day) (p<0.001) and serum Mg concentration increased from 2.01 meq/L (1.005 mmol/L) to 2.09 meq/L (1.045 mmol/ L) (p < 0.01). Whereas Cappuccio *et al.*²⁴ used Mg aspartate in their study. Although the plasma Mg concentration and urinary Mg excretion significantly increased during Mg supplementation, mean BP did not change throughout the study. The results of Cappuccio et al.24 provide no evidence for a role of dietary Mg in the regulation of high BP and are contrary to recent speculations.

The mean serum Mg levels in hypertensive and normotensive subjects were 1.94 ± 0.26 mg/dL (0.81 ± 0.11 mmol/L range 1.38-2.24 mg/dL) and 1.95 ± 0.13 mg/dL (0.81 ± 0.05 mmol/L range 1.68-2.14 mg/dL), respectively (data not shown in tables). The normal concentration of Mg in serum is between 0.65 and 1.05 mmol/L²⁵ reference range for Mg in serum with AAS method is 0.6-1.1 mmol Mg/L²⁶. According to Perkin-Elmer, normal levels in serum are 1.7-2.8 mg % Mg (1.42-2.33 meq/L)¹⁵. Our mean serum Mg concentration was similar to the literature^{15,25}. The mean serum Mg level in HT was not significantly different when compared with the controls (p >0.05). Among 13 HT subjects three women had serum Mg values lower than 1.7 mg Mg/dL (1.38-1.68%). After 6 months treatment with Mg (Mg supplementation), serum Mg concentrations have increased to normal levels. Serum Ca levels of these three women were also lower than standard values (6.9-7.9 %)¹³. Therefore the present study shows no difference in serum Mg levels between essential hypertensive patients and control group and present results are in close agreement with that of other studies²². In earlier publication²⁷, we couldn't demonstrate any difference between the hypertensive and normotensive pregnant women related to the serum Mg levels. Previous studies reported contrasting data on serum Mg concentrations in hypertensive as compared with normal subjects: lower values in both gender, in men but not women, or similar to control subjects. In an intervention study hypertensive patients on diuretic therapy were given Mg aspartate-HCl, which resulted in a subsequent decrease in blood pressure over 6 months without changes in plasma Mg or other electrolytes. There are a limited number of reports on Mg intake in relation to hypertension and results are conflicting²⁸.

After analyzed drinking water, tea and bread we obtained mean Mg concentration 1.5 mg (n = 59), 2.2 mg (n = 60) and 30 mg (n = 25) per 100 g, respectively. Mean drinking water consumption was 800-1500 g and then drinking water provided 12-23 mg Mg/d. These values are ca. 4-7 % of RDA [(12 mg Mg/320 mg Mg RDA) \times 100 = 4 %]. Daily tea consumption was ca. 200-400 g and then mean Mg from tea was 5-9 mg/d. Daily mean bread consumption in four group (NT + HT) was 250 g. Mean Mg consumption from bread was 75 mg/d and approximately one fourth of RDA [(75 mg Mg/320 mg Mg RDA) \times 100 = 23.4 %]. Magnesium intakes from water, tea and bread are not negligible compared to RDA. Therefore, Mg intakes showed in Table-3, include Mg content of water, tea and bread. In general, bread consumption in Turkey is large. It was determined that pregnant mother consumed daily ca. 330 g (during 2nd trimester), 370 g (during 3th third trimester) and 420 g (during lactation) bread in previous study²⁹. Also during an other study we determined hypertension in males 21.9 and 42.3 % in females (mean age ± standard deviation, males: 71.1 \pm 6.4 years, range 65-114 years, females: 70.2 \pm 7.9 years, range 65-104 years). In these groups daily consumption of bread were 358 and 256 g, respectively¹.

Magnesium consumption in NT subjects both female and male groups was more than HT subjects, but differences are not statistically significant (for female t = 1.73, n = 37 and for male t = 0.85, n = 20, p > 0.05, data not shown in table). Excluding gender daily dietary Mg intake in NT subjects were higher (375 ± 98 mg/d) than those in HT subjects (317 ± 129 mg/d), but differences are not statistically significant (t = 1.93 n = 26 for NT, n = 31 for HT, 0.10 > p >0.05, data not shown in table). Blood pressure is affected by dietary Mg, so dietary Mg may have a modest effect on the development of hypertension in women. Magnesium intake was inversely associated with the risk for developing hypertension. Women consumed more Mg than RDA (mean 376 mg/day) had a decreased risk for hypertension compared with those consumed as RDA (mean 312 mg/day). Song *et al.*³⁰ also reported same results. Some trials have shown that providing increased amounts of Mg may lower BP in at least some sensitive subjects^{9,25,26}. Daily dietary Mg intake in NT female was not different than those in NT male, same results were obtained about HT groups (p > 0.05). The recommended daily dietary intake of Mg is 320 mg/d (13.3 mmol/d) for female and 420 mg/d (17,5 mmol/d) for male in Turkey¹⁰. Daily dietary intake of Mg was compared with RDA for Turkish people and results are given in Table-3.

Normotensive female consumed more Mg than RDA. Hypertensive male consumed lower Mg than RDA (p < 0.05), consumption of Mg in HT female and in NT male are not statistically different from the RDA standards (p > 0.05). The high content of Mg in hard water has been cited as possible reason for the lower incidence of sudden death from heart disease in areas of hard water as compared to soft water areas⁸. Majority of our subjects consumed more hard water (tap water) than soft water. We reported the daily Mg intake obtained by composite analysis of lactating women 347 ± 109 mg/d (range 145-580 mg/d)¹⁴ and strictly breast feeding infant from breast feed (1 and 4 months) $20.9 \pm 4.9 \text{ mg/d}$ and $26.1 \pm 5.8 \text{ mg/d}$ (range 10.0-38.4 mg/d)¹⁴, children aged 2 to 6 years 175 ± 35 mg/d (range 101-249 mg/d)³¹. Bread, especially whole-wheat bread, fruits, cereals, vegetables and grains is consumed largely in Turkey, therefore Turkish diet supply adequate Mg in NT subjects.

Sacks *et al.*¹⁹ found that at baseline mean (\pm SD) 24 h ambulatory blood pressures were 116 \pm 8 and 73 \pm 6 mmHg systolic and diastolic and mean diatery Mg 239 \pm 79 mg/d in a NT women group. In present study, daily dietary Mg intake in all groups was higher than those reported by these authors. In the large-scale Dietary Approaches to Stop Hypertension (DASH) trial, a diet rich in fruits and vegetables that raised lower-than-average K and Mg intakes significantly decreased 24 h ambulatory blood pressures in persons with high-normal diastolic blood pressure or Stage 1 hypertension (-3.1/-2.1 mm Hg) and significantly decreased clinic BP in the hypertensive group.

Blood pressure is affected by nutritional status. In normotensive subjects correlations between BMI and BP were significant ($r_{BMI-DBP} = 0.494$, n = 35, t = 3.26, p < 0.01 and $r_{BMI-SBP} =$ 0.340, n = 35, t = 2.07, p < 0.05, data not shown in table). In cross-sectional and longitudinal population studies, systolic blood pressure increases with age until the eight decade of life. By contrast, diastolic blood pressure rises only until 50 years of age, after which it either becomes constant or even decreases slightly. Below age 50 years, DBP was the strongest

	EV	VALUATION	I OF DAILY		ABLE-3 Mg INTAKE	ACCORDIN	IG TO RDA*	⁻ (mg/d)		
	Female Male Statistical analysis									
Groups n X ¹		\mathbf{v}^{l}	SD^1		X ¹	SD^1	Female (RDA:320)		Male (RDA:420)	
	3D	n	Λ	3D	t	р	t	р		
Normotensive	15	376	90	11	373	112	2.78	< 0.05	-1.62	ns
Hypertensive	22	312	133	9	328	124	-0.28	ns	-2.34	< 0.05
*Include Mg conte	nt of water te	ea and bread								

*Include Mg content of water, tea and bread

predictor and obviously when a person lost weight preliminary DBP decrease⁶. In this study, $r_{BMI-DBP}$ is greater than $r_{BMI-SBP}$. In hypertensive subjects correlations between BMI and BP are not significant ($r_{BMI-DBP} = 0.0150$ and $r_{BMI-SBP} = 0.266$, p > 0.05). Systolic blood pressure correlated positively with DBP ($r_{NT} = 0.588$, n = 35, t = 4.18 and $r_{HT} = 0.741$, n = 32, t = 6.04, p < 0.01, data not shown in table).

The results obtained from regression analysis for NT and HT subjects are given in Table-4. In NT subjects there were statistically significant positive correlations between dietary Mg, Ca, K and Na (Table-4, p < 0.05). In a study reported by Kesteloot and Joosens³², their findings point to a strong positive association between dietary intake and urinary excretion of Na, K, Ca and Mg and no significant relationship was found between these urinary cations and blood pressure. In NT subjects correlations between dietary Mg and BP are not significant (p > 0.05). Urinary Mg excretion was negatively correlated with dietary Na and positively correlated with DBP, SBP and serum Ca.

In case of hypertension, there were statistically significant correlations between Mg intake and energy, protein, fat, Ca, K and Na. In the same group BP was positively correlated with UMgV ($r_{DBP-UMgV} = 0.390$, $r_{SBP-UMgV} = 0.385$) and negatively correlated with serum Mg levels ($r_{DBP-Serum Mg} = -0.593$, $r_{SBP-Serum Mg} = -0.555$, p < 0.05, Table-4).

It had been determined that among hypertensive women (nurses) dietary Mg was not significantly associated with risk of hypertension after adjusting for age, BMI, alcohol and energy intake, among nonhypertensive women, there was a significant inverse association with self-reported SBP and DBP⁹. A prospective study of nutritional factors and hypertension among US men (health professionals) results support that an increased intake of fiber and Mg may contribute to the prevention of hypertension³³. The intake of Mg (along with other nutrients) was related to blood pressure in elderly participants of the Honolulu Heart study. Magnesium had the strongest association with blood pressure, which supports recent interest in its relation to blood pressure and Mg intake from foods and supplements was strongly and inversely associated with both SBP and DBP³⁴. Charlton *et al.*³⁵ found a mean dietary intake of Mg (228-285 mg/d) lower than present results and authors denoted that dietary intake of Mg was not associated with SBP or DBP. Analysis of the first National Health and Nutrition Examination Survey (NHANES) in 1984 revealed that a dietary pattern low in mineral intake, specifically Ca, K and Mg, was associated with hypertension in American adults. Inadequate mineral intake, which in particular is characterized by a low intake of dairy, fruits and vegetables, is the best dietary pattern predictive of elevated blood pressure in persons at increased risk for cardiovascular disease, specifically those with isolated systolic hypertension³⁶.

In both groups, UMgV was not correlated with dietary Mg (Table-4). However dietary Mg was correlated negatively with UCaV in NT group (r = -0.594, p < 0.05). In NT group UMgV was correlated positively with UCaV ($r_{UCaV-UMgV} = 0.837$, n = 28) and graphic of correlation is plotted out in Fig. 2. But in the HT subjects UMgV was not correlated with UCaV. Zozaya *et al.*²¹ reported a high correlation between urinary excretion of Ca and Mg in the hypertensive subjects.

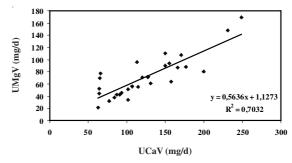


Fig. 2. Correlation between urinary Mg and Ca excretion rates in NT group (n = 28, r = 0.839)

In both groups of blood pressure were directly related to urinary Mg excretion (p < 0.05) particularly among NT group subjects ($r_{SBP-UMgV} = 0.600$, $r_{DBP-UMgV} = 0.466$, Table-4). Resnict *et al.*⁵ also reported a negative correlation between diet-induced changes in urinary Mg excretion and diet-induced changes in SBP and DBP.

Variables Diet M	1	Normotensive $(n = 35)$	5)	Hypertensive $(n = 32)$			
	Diet Mg	UMgV	Serum Mg	Diet Mg	UMgV	Serum Mg	
Energy	0.198	0.174	0.170	0.616*	0.132	-0.055	
Protein	0.011	0.03	0.068	0.606*	0.017	0.100	
Fat	-0.198	0.141	0.305	0.377*	-0.007	0.230	
Diet Ca	0.390*	0.144	-0.383	0.667*	-0.047	0.265	
Diet K	0.679*	-0.254	-0.167	0.703*	-0.064	0.233	
Diet Na	0.469*	-0.405**	0.183	0.359*	0.126	0.285	
DBP	0.181	0.466**	0.240	0.016	0.390**	-0.593***	
SBP	0.029	0.600**	0.092	0.089	0.385**	-0.555***	
BMI	-0.007	-0.212	0.187	0.071	0.057	-0.204	
UMgV	-0.116	1	0.040	-0.276	1	0.014	
Serum Ca	-0.258	0.439***	0.555***	0.055	0.102	0.910****	
Serum Mg	0.305	0.040	1	-0.144	0.004	1	

TABLE-4 SIMPLE CORRELATION COEFFICIENTS AMONG ENERGY, NUTRIENTS, BODY MASS INDEX AND DIET, SERUM AND URINARY MAGNESIUM AND BLOOD PRESSURE (r)

*Correlation is significant at the 0.05 level (2-tailed) ($n_{NT} = 26$ and $n_{HT} = 31$ for dietary research).

**Correlation is significant at the 0.05 level (2-tailed) ($n_{NT} = 28$ and $n_{HT} = 27$ urinary research).

***Correlation is significant at the 0.05 level (2-tailed) ($n_{NT} = 13$ for serum research).

****Correlation is significant at the 0.01 level (2-tailed) ($n_{HT} = 13$ for serum research).

There were highly significant correlations between the total serum Ca level and the serum Mg level (p < 0.05) in the two groups (Table-4). Zozaya *et al.*²¹ reported that a highly significant correlation between the total serum Ca level and the serum Mg level (p < 0.001) in the normal subjects. This correlation was not found in the hypertensive group.

Correlation among urinary Mg excretion rates: Since 24 h urine collections are difficult to obtain and evaluate in free-living population groups^{13,37}, overnight and 24 h urine specimens were collected by 43 subjects (11 HT, 32 NT). Mean urine collection time and collected urinary volume were during overnight 843 min (range 530-1050 min) and 772 mL (range 240-1850 mL) in other words 0.93 ± 0.48 mL/min, during 24 h 1408 min (range 1255-1630 min) and 1543 mL [(range 465-3650 mL) 1.10 ± 0.49 mL/min], respectively. The mean Mg excretion rates were during the overnight and 24 h period $82 \pm 45 \text{ mg/d}$ [(range 21-223 mg/d) $0.285 \pm 0.156 \text{ meq/h}$] and $93 \pm 41 \text{ mg/d}$ [(range 21-178 mg/d) $0.323 \pm 0.142 \text{ meq/h}$], respectively. Overnight Mg excretion rate was statistically correlated with the 24 h excretion rate (n = 43, t = 5.23, p <0.01). This is very important relationship shown in Fig. 3. as a scatter diagram.

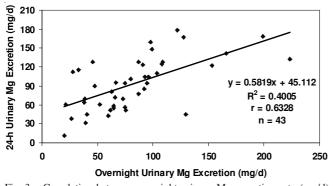


Fig. 3. Correlation between overnight urinary Mg excretion rate (mg/d) and 24 h urinary Mg excretion rate (mg/d)

Conclusion

(1) The results suggest that higher intake of dietary Mg may have a modest effect on the development of hypertension in women. (2) Overnight urinary Mg excretion rate was correlated with the 24 h UMgV, so 12-h UMgV should be usually employable. (3) Correlation between 24 h UMg and dietary Mg was not statistically significant, so the urinary Mg excretion does not accurately reflect dietary Mg intake and therefore should not be routinely evaluated.

ACKNOWLEDGEMENTS

The authors are thankful to the dieticians, Tülay Ozbek and Cemil Saka for their valuable nutritional survey. The authors are also grateful to Dr. Ibrahim Narin for providing technical aid during AAS analysis, Dr. Tülay Bagci Bosi and Dervis Topuz for statistical analysis and all subjects for collection of urine.

REFERENCES

- G. Pekcan, S. Yücecan, M. Tayfur, K. Kayakirilmaz, N. Rakicioglu, S. Mercanligil and G. Eroglu, Euronut Seneca First Europeen Congress on Nutrition and Health in the Elderly, Noordwijkerhout, The Netherlands, pp. 87-95 (1991).
- K. Sudhakar, M. Sujatha, B.S. Ramesh, P. Padmavathi and P.P. Reddy, Indian J. Clin. Biochem., 19, 21 (2004).
- M.L. Brown, Hypertension in Present Knowledge in Nutrition, International Life Sciences Institute Nutrition Foundation, Washington DC, USA, edn. 6, pp. 355-370 (1990).
- A. Leiba, A. Vald, E. Peleg, A. Shamiss and E. Grossman, *Nutrition*, 21, 462 (2005).
- L.M. Resnick, S. Oparil, A. Chait, R.B. Haynes, P. Kris-Etherton, J.S. Stern, S. Clark, S. Holcomb, D.C. Hatton, J.A. Metz, M. McMahon, F.X. Pi-Sunyer and D.A. McCarron, *Am. J. Hemtol.*, 13, 956 (2000).
- J.A. Staessen, J. Wang, G. Bianchi and W.H. Birkenhager, *Lancet*, 361, 1629 (2003).
- R.J. Elin, Assessment of Magnesium Status, Clinical Chemistry, Vol. 33, 1965-1970, Copyright© 1987 by American Association for Clinical Chemistry.
- R.L. Pike and L.M. Brown, Minerals and Water in Integrated Approach, John Wiley and Sons, Inc., edn. 2, pp. 180-210 (1975).
- 9. M.C. Catherine, Nutr. Clin. Prac., 23, 142 (2008).
- 10. Ministry of Health of the Republic of Turkey, Handbook of Turkish Nutrition, Ankara, Turkey (2004).
- S.S. Davidson, R. Passmore, J.F. Brock and A.S. Truswell, Magnesium in: Human Nutrition and Dietetics, Churchill Livingstone Edinburgh London and New York, edn. 7, pp. 98-99 (1979).
- G.W. Nicoll, A.D. Struthers and C.G. Fraser, *Clin. Chem.*, **37**, 1794 (1991).
 K. Kayakirilmaz, E. Bayol, H.B. Yilmaz, H.K. Özgün, T. Özbek, D.
- Topuz and R. Battaloglu, *Asian J. Chem.*, **20**, 3598 (2008). 14. K. Kayakirilmaz and O. Köksal. *Doga Tip ve Ecz. D.*, **11**, 253 (1987).
- K. Kayakirilmaz and O. Köksal, *Doga Tip ve Ecz. D.*. **11**, 253 (1987).
 Perkin-Elmer, Analytical Methods for Atomic Absorption Spectroscopy, The Perkin-Elmer Corporation, USA (1996).
- E.J. Oudewicz, Modern Mathematical Statistics, John Wiley & Sons Inc., New York (1988).
- World Health Organization, Protein and Energy Requirement Report of a Joint FAO/WHO/UN Ad Hoc Expert Committee FAO Nutrition Report Technique Series No. 724, Geneva, Switzerland (1986).
- P. Delva, C. Pastori, M. Degan, G. Montesi, P. Brazzarola and A. Lechi, *Hypertension*, 35, 113 (2000).
- F.M. Sacks, W.C. Willett, A. Smith, L.E. Brown, B. Rosner and T.J. Moore, *Hypertension*, **31**, 131 (1998).
- 20. A. Šimecková, V. Zamrazil and J. Cerovská, Physiol. Res., 47, 35 (1998).
- 21. G.J.L. Zozaya, P.M. Viloria and A. Castro, South Med. J., 81, 350 (1988).
- 22. D.M. Tillman and P.F. Semple, Clin. Sci., 75, 395 (1988).
- 23. T. Motoyama, H. Sano and H. Fukuzaki, Hypertension, 13, 227 (1989).
- F.P. Cappuccio, N.D. Markandu, G.W. Beynon, A.C. Shore, B. Sampson and G.A. MacGregor, *Br. Med. J. (Clin. Res. Ed.)*, 291, 235 (1985).
- 25. R. Iva, K. Petar and B. Vanja, Acta Clin. Croat., 42, 59 (2003).
- N.W. Tietz, Reference Ranges in Fundamentals of Clinical Chemistry, W.B. Saunders Company, edn. 3, 9, p. 524 (1987).
- H. Özgünes, K. Kayakirilmaz, F.B. Ataç, O. Karabacak and M.S. Beksaç, Gynecol. Obstet. Reprod. Med., 1, 20 (1995).
- R.E. Olson, H.P. Broqusit, C.O. Chichester, W.J. Darby, A.C. Kolbye and R.M. Stalvey, Magnesium in Present Knowledge in Nutrition, The Nutrition Foundation Inc. Washington DC, USA, edn. 5, pp. 422-438 (1984).
- 29. O. Köksal and K. Kayakirilmaz, Doga Tip ve Ecz. D., 11, 359 (1987).
- Y. Song, H. Sesso, J. Manson, N. Cook, J. Buring and S. Liu, *Am. J. Cardiol.*, 98, 1616 (2006).
- 31. K. Kayakirilmaz, Gida, 14, 243 (1989).
- 32. H. Kesteloot and J.V. Joosens, J. Hum. Hypertens, 4, 527 (1990).
- A. Ascherio, E.B. Rimm, E.L. Giovannucci, G.A. Colditz, B. Rosner, W.C. Willett, F. Sacks and M.J. Stampfer, *Circulation*, 86, 1475 (1992).
- 34. M.R. Joffres, D.M. Reed and K. Yano, Am. J. Clin. Nutr., 45, 469 (1987).
- K.E. Charlton, M. Phil, K. Steyn, N.S. Levitt, J.V. Zulu, B. Cur, D.R.N. Jonathan, F.J. Veldman and J.H. Nel, *Nutrition*, 21, 39 (2005).
- M.S. Townsend, V.L. Fulgoni, J.S. Stern, S. Adu-Afarwuah and D.A. McCarron, Am. J. Hypertens., 18, 261 (2005).
- 37. R.L. Watson and H.G. Langford, J. Clin. Nutr., 23, 290 (1970).