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Original Communication

Serum Magnesium and Adiposity in Saudi Adults: Sex-Stratified Analysis

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Abstract

Background: Obesity affects 23.1% of Saudi adults aged ≥15 years and alters the bioavailability of micronutrients, such as Mg, essential for many physiological processes. This study assessed associations of serum and dietary Mg with obesity markers in Saudi adults. **Methods:** A cross-sectional study was conducted during 2021 to 2023 among 776 Saudi adults aged 18 to 64 years, including females (n = 490) and males (n = 286) recruited from community sites and clinics in Riyadh and Al Madinah, Saudi Arabia. Data included demographic characteristics, medical history, lifestyle factors (smoking and physical activity), anthropometric measurements [weight, height, body mass index (BMI), fat mass index (FMI), body fat percentage, waist and hip circumferences, waist–hip ratio, and waist–height ratio (WHtR)], biochemical parameters (serum Mg), and dietary intake (via a validated Saudi food frequency questionnaire, an Mg-specific food frequency questionnaire, and 24-h recall). Linear regression models were applied and were adjusted for age, smoking status, education, marital status, physical activity, and Mg from supplements. **Results:** Among females, higher BMI and FMI were associated with lower serum Mg, with coefficient expressed in 10^{-2} mg/dL units [BMI: –7.7, 95% confidence interval (CI): –14.2 to –1.2, $p = 0.02$; FMI: –4.5, 95% CI: –8.5 to –0.6, $p = 0.02$]. Larger hip circumference was also associated with lower serum Mg (expressed in 10^{-2} mg/dL) in females [–54.3, 95% CI: –91.5 to –17.1, $p = 0.001$]. No statistically significant associations between serum Mg and adiposity markers were observed among males. Dietary Mg showed limited associations. In males, higher hip circumference was associated with higher dietary Mg (mg/1000 kcal) [0.08, 95% CI: 0.04 to 0.14, $p = 0.02$]. In females, higher body fat percentage was associated with lower dietary Mg [–0.18, 95% CI: –0.31 to –0.09, $p = 0.03$]. No other adiposity markers were significantly associated with dietary Mg in adjusted models. **Conclusions:** Serum and dietary Mg captured distinct aspects of adiposity. Notably, lower serum Mg was associated with higher adiposity in females whereas no serum associations were observed in men. Further longitudinal and clinical studies with additional Mg biomarkers are warranted.

Keywords: magnesium; obesity; body mass index; waist circumference; waist–hip ratio

1. Introduction

Obesity is a chronic, multifactorial disease characterized by excess adiposity and elevated cardiometabolic risk [1]. In 2022, 16% of adults worldwide had obesity (~890 million individuals), and adult obesity has more than doubled since 1990 [2]. Saudi Arabia reflects this trend: the 2019 World Health Survey reported obesity in 20.2% of Saudi adults, with 38.2% being overweight [3]. Since 2019, obesity rose by 3.1% (from 20% to 23.1%) and overweight prevalence increased by 6.9% (from 38.2% to 45.1%) [4]. A meta-analysis of over 280 studies using waist circumference (WC) found that the estimated global central obesity prevalence was 41.5% among adults aged ≥15 years and 36.0%–45.5% in Saudi Arabia [5]. Furthermore, in the 2019 World Health Survey, abnormal waist circumference rose with age, from 23% at 15–29 years to 58% at 80 years and older, and was higher in women than men, 34% versus 27% [6].

Obesity results from interacting genetic, environmental, behavioral, and pharmacologic factors, including physical inactivity, psychological conditions, medications, and unhealthy dietary patterns (e.g., high-energy diets, excess processed foods, and poor-quality diets) [7,8,9,10,11]. Dietary micronutrient quality may modify obesity risk. Many adults fail to meet recommended intakes for key micronutrients, including Mg [12,13,14,15]. Mg supports energy metabolism, nucleic acid and protein synthesis, and ion transport, serving as a cofactor for at least 300 enzyme systems and likely over 600 reactions [16]. Serum Mg alone may underestimate deficiency because levels can appear normal despite low intracellular stores, complicating clinical and epidemiologic assessment [17]. Mg balance is affected by diet and medications: proton pump inhibitors, certain antibiotics, calcineurin inhibitors, cisplatin and other antineoplastics, and loop or thiazide diuretics can cause drug-induced hypomagnesemia, whereas high-phytate foods and some minerals at pharmacologic doses reduce absorption [18].

Mg is essential for insulin secretion and signaling. It also affects cardiometabolic pathways beyond insulin as

it acts as a natural calcium antagonist, relaxes vascular smooth muscle, improves endothelial nitric oxide availability, and modulates the renin–angiotensin–aldosterone system, which together lower vascular tone and blood pressure [19]. Furthermore, it reduces oxidative stress and NF-κB driven inflammation that link adiposity to atherogenesis. It also influences lipid handling and adipokine biology, with effects on fat storage and metabolic risk [20]. These pathways connect magnesium status to obesity-related hypertension, dyslipidemia, and cardiovascular disease. Thus, low Mg levels impair insulin receptor tyrosine kinase activity, decrease insulin sensitivity, and promote insulin resistance, a hallmark of metabolic syndrome [21].

Several mechanisms may explain why lower magnesium levels may be observed in individuals with obesity. First, magnesium intake may be lower when diets contain fewer magnesium-rich foods, such as green leafy vegetables, legumes, nuts, seeds, and whole grains, and more refined foods, because grain refining removes much of the magnesium-rich germ and bran [17]. Second, effective magnesium absorption may be lower even at similar reported intakes, because intestinal magnesium bioavailability varies with dose, food matrix, and dietary enhancers or inhibitors [17]. Third, obesity commonly coexists with insulin resistance and hyperinsulinemia, and human studies suggest that insulin can increase renal magnesium excretion; hyperglycemia can also increase urinary magnesium losses [22]. Together, these pathways suggest that lower magnesium in obesity may reflect a combination of lower intake, lower effective absorption, and greater renal losses rather than a single mechanism.

Although magnesium is mechanistically linked to insulin action, inflammation, vascular tone, and lipid metabolism [19,20,21], epidemiologic evidence relating magnesium status to adiposity remains inconsistent [23,24]. This heterogeneity likely reflects differences in study design, population characteristics, and how magnesium status is assessed. Serum magnesium represents a tightly regulated compartment that accounts for <1% of total body stores [17,25], whereas dietary intake estimates are prone to reporting error and do not capture absorption or losses. Evidence from Saudi Arabia and the broader Middle East is limited, and few studies have concurrently examined serum magnesium, dietary magnesium intake, detailed adiposity/body composition measures, and cardiometabolic biomarkers in the same cohort. Given the high and rising burden of obesity in Saudi Arabia and recent dietary shifts that may lower magnesium density [26], clarifying these relationships is important for generating hypotheses for longitudinal and intervention studies. This study aimed to evaluate sex-stratified associations of serum magnesium and dietary magnesium intake with general and central adiposity markers in Saudi adults.

2. Materials and Methods

2.1 Study Design

The study was part of an ongoing project on lifestyle scores and blood pressure among Saudi adults [27]. Data were collected at community sites in Riyadh and Al Madinah as well as clinics at the College of Applied Medical Sciences, King Saud University (KSU). The KSU Institutional Review Board approved the protocol (KSU-IRB-21-314), and written informed consent was obtained from all participants.

2.2 Recruitment

Participants were recruited through portable clinics established in Riyadh and Al madinah as well as at KSU Applied Medical Sciences clinics. Invitations were distributed via WhatsApp messages, posters on social media, brochures in malls, and posters displayed near clinics. Recruitment materials described the study's purpose, inclusion criteria, methods, setting, and potential benefits.

2.3 Eligibility Criteria

Inclusion criteria were Saudi adults aged ≥18 years. Exclusion criteria included pregnancy or lactation, use of medications that inhibit Mg absorption or increase Mg excretion, and medical conditions affecting Mg absorption, excretion, or redistribution (e.g., chronic or acute diarrhea, recent bariatric surgery, intestinal malabsorption, type 1 and 2 diabetes mellitus, kidney disease, inherited renal tubular disorders, hyperthyroidism, hypercalcemia, hyperaldosteronism, hungry bone syndrome, or acute pancreatitis). In total, 1104 adults were screened: 165 did not meet the inclusion criteria, 28 had incomplete data, and 35 withdrew. Ultimately, 876 participants were recruited, and 776 adults aged 18 to 64 years completed biochemical testing, including females (n = 490) and males (n = 286) (Fig. 1).

2.4 Sample Size

The sample size calculation was used to estimate the minimum required overall sample. Using an obesity prevalence of 23.1% (obesity prevalence in Saudi adults) [3], with d = 0.05 and Z = 1.96, the minimum required sample was 273 participants; after allowing 20% for nonresponse, the target sample was approximately 328. Recruitment continued beyond this minimum because the study was embedded within an ongoing community-based project and because additional participants were needed to offset exclusions, incomplete biochemical data, and attrition from the final analytic sample. The final sample was therefore larger than the calculated minimum. Sex-specific quotas were not prespecified at the design stage.

The adequacy of the final analytic sample for the regression analyses was evaluated using a sensitivity power analysis for linear multiple regression, fixed model, R2 increase, with alpha = 0.05 and 80% power [28]. Because the main analyses tested one adiposity marker at a time after

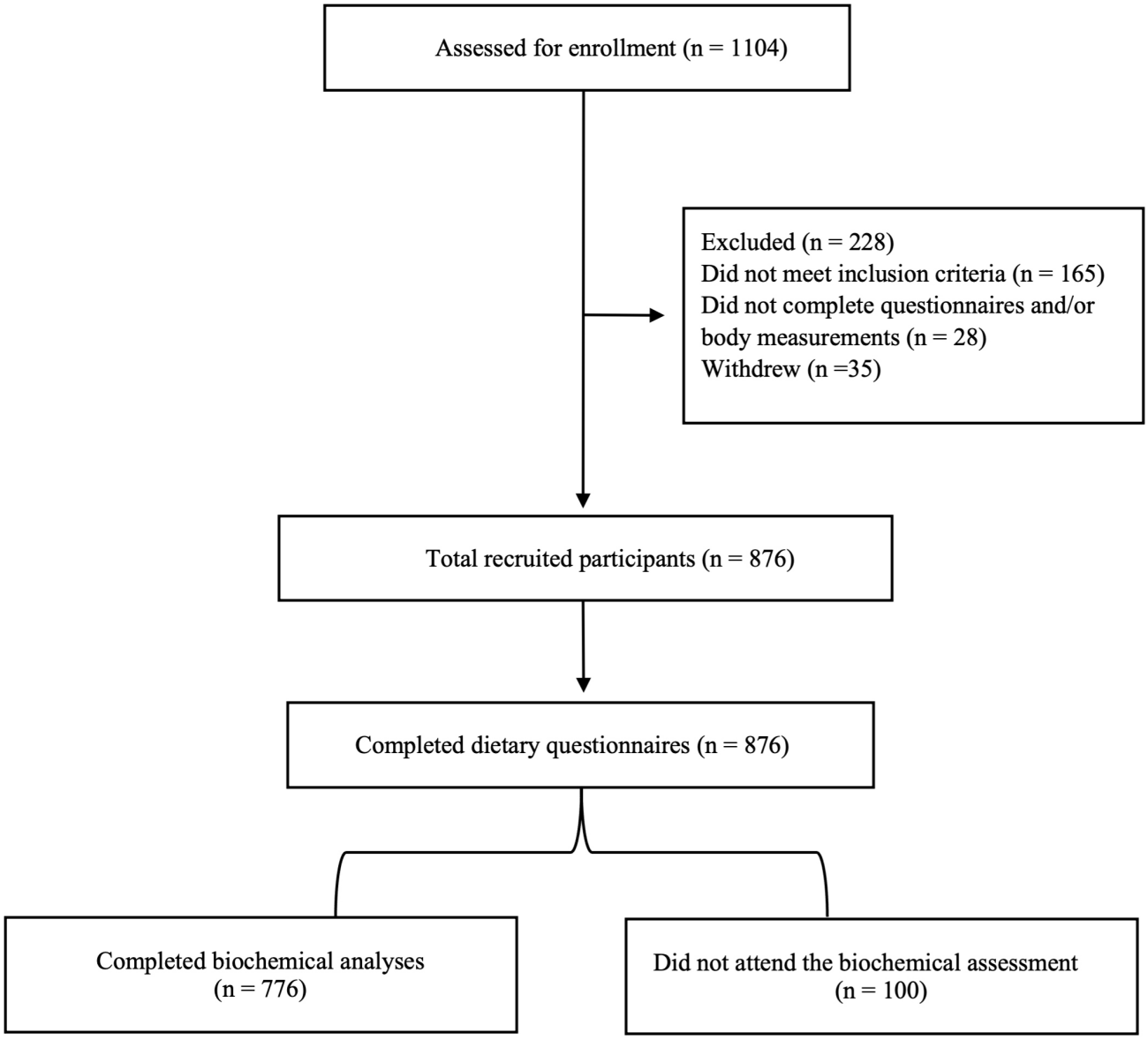

Fig. 1. Study enrollment flowchart.

covariate adjustment, the number of tested predictors was set to 1. The most conservative model included up to 10 total predictors, comprising the adiposity marker, age, smoking status, education, marital status, physical activity, and Mg from supplements. Under these assumptions, the overall sample (n = 776) could detect a minimum Cohen's f2 of 0.010, equivalent to a unique partial R2 of approximately 1.0%. The female-stratified sample (n = 490) could detect f2 = 0.016, partial R2 approximately 1.6%. The male-stratified sample (n = 286) could detect f2 = 0.028, partial R2 approximately 2.7%. Thus, the overall and female-stratified samples were adequately powered to detect effects at or below Cohen's conventional small-effect threshold (f2 = 0.02), whereas the male-stratified sample was powered to detect effects slightly larger than this threshold. Therefore, null findings in males should be interpreted cautiously.

2.5 Data Collection

Data were collected during November 2021–July 2023 (21 months). Trained dietitians conducted interviews at data collection sites. They completed protocol-based training in questionnaire administration and measurement standardization, using the World Health Organization STEPS [29] and National Health and Nutrition Examination Survey (NHANES) [30] procedure manuals as references. Training covered instrument flow, skip patterns, probing, role-play, and field certification. We pilot tested the questionnaire on ten adults to refine wording and timing. Field supervisors observed early interviews and provided corrective feedback.

Anthropometric measurements were obtained first, followed by structured, interviewer-administered questionnaires covering demographic, health, and lifestyle informa-

tion. Standardized procedures were used for anthropometric, blood pressure, and biochemical assessments.

2.6 Lifestyle Measures

2.6.1 Sociodemographic and Health History

Age, sex, birthplace, education, income, occupation, marital status, housing, smoking status, medication and supplement use, surgical history, and personal and family medical history were recorded.

2.6.2 Physical Activity

The validated Arabic Global Physical Activity Questionnaire [31] was used to assess occupational, transport-related, and discretionary or leisure-time physical activity, covering intensity, duration, and frequency.

2.6.3 Dietary Intake

Trained dietitians collected data during a structured interview using the validated Saudi Food and Drug Authority Food Frequency Questionnaire (SFDA-FFQ) [32] for dietary intake during the past year as well as two 24-h dietary recalls [33] to improve the accuracy of food intake estimations [34]. SFDA-FFQ, developed in Arabic, lists 133 food and beverage items with intake frequency (per day, week, or month) and includes questions regarding traditional Saudi food as well cooking fats and vitamin/mineral supplementation [32]. A validated Mg-specific FFQ (Mg-FFQ) with 33 Mg-rich food items was also administered to better capture intake of Mg-dense foods [35]. The 24-h dietary recalls, collected on two consecutive days (one in person on the recruitment day and one by phone), provided detailed quantitative intake data over the past 24 h. Food models and hand-size portion guides helped estimate portions and minimize portion misreporting. Nutrient intakes from the SFDA-FFQ and from the mean of the two 24-h recalls were compared as a quality check; correlations for macronutrients were high for protein ($r = 0.52$) and fat ($r = 0.61$), indicating good agreement between the assessment methods. Dietary Mg intake used in the present analyses was derived from the SFDA-FFQ (food sources). Dietary components were analyzed using ESHA's Food Processor Nutrition Analysis software® (Version 11.14, ESHA Research, Salem, OR, USA) with the US Department of Agriculture standard reference database. This software has an extensive food, beverage, and recipe database, and new recipes can be added based on their ingredients if a food item is unavailable. For Saudi recipes, data were derived from the Nutrition Assessment Guide for Saudi Arabia by the Obesity Research Center at KSU [36].

2.6.4 Calculation of Dietary Mg intake

Dietary magnesium intake used in the present analyses was derived from the validated SFDA-FFQ, which assessed usual food intake over the previous year. For each food item, the reported frequency of intake was converted to servings/day, multiplied by the standard portion size, and matched to nutrient composition data to calculate magnesium intake. Total dietary magnesium intake was expressed as mg/day. Food-derived Mg intake (mg/day) was calculated from the 133-item SFDA-FFQ by summing the item-specific Mg contribution for 85 food and beverage items with nonzero Mg values assigned in Food Processor Nutrition Analysis software, with local Saudi recipes entered when needed. Items with zero assigned Mg content or no reported consumption contributed 0 mg/day:

Dietary Mg (mg/day) = sum over items [frequency (servings/day) × portion (g/serving) × Mg content (mg/g)].

For the 24-h recalls, Mg intake for each recall day was calculated as the sum of Mg across all foods consumed that day, and the mean of the two days was computed for comparison with the FFQ-based estimate. The FFQ-derived dietary magnesium estimate showed a correlation with the mean 24-hour recall-derived magnesium estimate ($r = 0.5$). Mg from supplements was recorded separately and was not included in the food-derived dietary Mg calculation. However, Mg supplement use was adjusted for in the multivariable regression analyses.

2.6.5 Anthropometric and Body Composition Measurements

Measurements were recorded twice by trained dietitians using standardized methods; averages were used in the final analyses. Participants wore light clothing and no footwear. Height (cm) was measured using a digital height meter (cm). Body weight (kg) and body fat percentage were measured using a bioelectrical impedance analysis (BIA) scale (InBody technology), employing the TANITA BC-418 body composition analyzer (Tanita Corporation, Japan) after a minimum 2-hour period without food intake. Measurements were obtained during the scheduled study visit, either in the morning or afternoon depending on appointment time. BMI was calculated as weight (kg) / height (m^2). Fat mass index (FMI) was calculated as fat mass (kg) / height (m^2) [37,38,39]. WC, hip circumference (HC), and waist–hip ratio (WHR) were measured using a nonstretchable measuring tape. WC was measured at the midpoint between the lower rib margin and the top of the iliac crest. HC was measured around the greater trochanter. Measurements were taken with the participant standing upright and the tape tight around the body (without being constricting); if readings differed by >2 cm, a third measurement was taken, and the mean of the two closest values was used.

2.7 Biochemical Analyses

Fasting peripheral blood (~10 mL) was collected and centrifuged at 1200 ×g for 15 min and the serum was stored at −80 °C. All biochemical tests were performed in one session per participant after an 8−10 hour fast. Serum magnesium concentration was measured in fasting serum using an automated xylidyl blue colorimetric assay on a Diatron

P500 clinical chemistry analyzer (Diatron, Budapest, Hungary). Serum magnesium results were expressed in mg/dL.

Biochemical analyses were performed in the same clinical laboratory under standard quality assurance procedures. Commercial internal QC materials at two concentration levels were analyzed at the beginning of each analytical run and after calibration, reagent-lot changes, or maintenance. Runs were accepted only when QC values met predefined acceptance criteria; otherwise, calibration, troubleshooting, and repeat analysis were undertaken. Calibration and calibration verification were performed according to the manufacturer's instructions. The laboratory also participated in external proficiency testing. Assay imprecision was summarized as CV (%) = 100 × SD / mean from repeated QC measurements, and bias, when available, was estimated relative to the assigned target or peer-group mean [40].

2.8 Statistical Analysis

Analyses were performed using IBM SPSS software v28 (Armonk, New York, USA). Participants were categorized according to serum magnesium concentration as <1.8 mg/dL and ≥1.8 mg/dL. *p*-values were calculated using independent-samples *t*-tests for continuous variables and Pearson chi-square tests for categorical variables. Potential effect modification by sex was assessed using formal interaction terms in the adjusted linear regression models.

Spearman rank correlation coefficients were calculated to assess sex-stratified bivariate associations between serum Mg concentration and adiposity markers (BMI, WC, FMI, HC, WHR, body fat percentage, and WHtR). Correlation coefficients and two-sided *p* values were reported.

Linear regression was used to examine associations between serum and dietary Mg and a 1 SD increase in each adiposity marker (BMI = 6, FMI = 4.7, WC = 17.2, HC = 13.8, WHR = 0.13, body fat percent = 10.5, WHtR = 0.1). Models were adjusted for age, smoking status, education, marital status, physical activity, Mg from supplements, and energy intake when dietary Mg was included. We checked linear model assumptions. For normality, we inspected histograms and Q-Q plots of standardized residuals and ran the Shapiro–Wilk test. We assessed multicollinearity using variance inflation factors and tolerance, and reviewed condition indices with variance-decomposition proportions [41].

3. Results

3.1 Participant Characteristics by Serum Mg Status

In total, 776 adults were analyzed, including 474 and 302 with serum Mg levels ≥1.8 and <1.8 mg/dL, respectively. The two groups did not differ significantly by age, sex, marital status, education level, smoking status, or energy intake. For obesity-related markers, participants with serum Mg <1.8 mg/dL had significantly higher WC, FMI, WHR, body fat percentage, and WHtR. No significant differences were observed for BMI or hip circumference (Table 1).

3.2 Correlations

Sex-stratified correlation analyses showed that serum magnesium was inversely correlated with several adiposity markers in females but not in males. In females, weak inverse correlations were observed for BMI, r = –0.10, p = 0.02, FMI, r = –0.09, p = 0.04, and hip circumference, r = –0.21, p = 0.01. In males, no statistically significant correlations were identified between serum magnesium and any adiposity marker (Table 2).

3.3 Association of Serum Magnesium With Adiposity Markers

Sex-interaction testing showed evidence of interaction for several adiposity indicators. Serum magnesium interaction *p*-values were significant for BMI, p = 0.04, FMI, p = 0.03, and hip circumference, p = 0.04. Dietary magnesium interaction *p*-values were significant for FMI, p = 0.04, and reached the conventional threshold for BMI and hip circumference, both p = 0.05. No significant interactions were observed for waist circumference, WHR, body fat percentage, or WHtR, with serum magnesium interaction *p*-values ranging from 0.08 to 0.81 and dietary magnesium interaction *p*-values ranging from 0.10 to 0.85. Therefore, sex-stratified estimates are presented.

Among males, none of the adiposity markers were significantly associated with serum Mg (all $p \geq 0.05$). Among females, higher BMI and FMI were each associated with lower serum Mg, with results expressed in 10^{-2} mg/dL units (mean difference for BMI: −7.7, 95% confidence interval [CI] = −14.2 to −1.2, p = 0.02; FMI: −4.5, 95% CI = −8.5 to −0.6, p = 0.02). Larger HC was also associated with lower serum Mg (results expressed in 10^{-2} mg/dL) in females (mean difference: −54.3, 95% CI = −91.5 to −17.1, p = 0.001). No other markers showed significant relations in females (all $p \geq 0.06$; Table 3).

3.4 Associations Between Dietary Mg and Adiposity Markers

In males, higher HC was associated with higher dietary Mg (mg/1000 kcal) (mean difference: 0.08, 95% CI = 0.04 to 0.14, p = 0.02), whereas other markers showed no significant associations (all $p \geq 0.05$). In females, higher body fat percentage was associated with lower dietary Mg (mean difference: −0.18, 95% CI = −0.31 to −0.09, p = 0.03). No other markers had significant associations (all $p \geq 0.05$; Table 4).

4. Discussion

This cross-sectional study examined serum Mg, dietary Mg, and multiple adiposity markers in Saudi adults. In sex-stratified models, females showed inverse associations between serum Mg and BMI, FMI, and hip circum-

Table 1. Descriptive statistics according to serum Mg levels in Saudi adults, (n = 776)[1] .

Variables	Serum Mg <1.8 mg/dL	Serum Mg ≥1.8 mg/dL	*p* value
N	302	474	
Age (years)	30.2 (10.6)	31.1 (11.5)	0.27
Male	34.5	39.8	0.13
Marital status			
Single	31.9	38.1	0.06
Married	57.2	56.7	
Divorced	10.9	5.2	
Education			
High school	36.1	32.9	0.40
Bachelor	48.9	53.6	
Graduate	15.0	13.5	
Smoker			
Yes	14.2	11.4	0.23
Serum Mg (mg/dL)	1.31 (0.43)	2.00 (0.22)	<0.001
Energy intake (kcal/day)	4089 (1266)	3980 (1400)	0.25
Dietary Mg (mg/day)	471 (133)	440 (135)	0.10
Dietary Mg (mg/1000 kcal)	118.8 (43.7)	119.3 (39.2)	0.88
Obesity markers			
BMI (kg/m^2)	27.0 (6.0)	26.4 (6.1)	0.17
WC (cm)	84.6 (16.2)	78.3 (14.5)	<0.001
FMI	10.1 (4.7)	9.4 (4.7)	0.04
HC (cm)	105.58 (13.48)	103.75 (13.93)	0.07
WHR	0.90 (0.09)	0.78 (0.13)	<0.001
Body fat percentage	36.0 (10.3)	33.5 (9.0)	0.005
WHtR	0.55 (0.10)	0.49 (0.09)	<0.01

[1] Data are mean (SD) or %. *p*-values were calculated using independent-samples *t*-tests for continuous variables and Pearson chi-square tests for categorical variables.
Abbreviations: BFP, body fat percentage; BMI, body mass index; FMI, fat mass index; HC, hip circumference; WC, waist circumference; WHR, waist–hip ratio; WHtR, waist-to-height ratio.

Table 2. Sex-stratified Spearman correlations between serum magnesium concentration and adiposity markers in Saudi adults, (n = 776)[1] .

Variable	Males		Females	
	r	*p* value	r	*p* value
BMI (kg/m^2)	–0.07	0.18	–0.10	0.02
WC (cm)	–0.05	0.32	–0.08	0.06
FMI	–0.02	0.45	–0.09	0.04
HC (cm)	0.05	0.73	–0.21	0.01
WHR	–0.02	0.74	–0.03	0.49
Body fat percentage	–0.09	0.09	–0.08	0.06
WHtR	–0.06	0.28	–0.06	0.15

[1] Data are Spearman's coefficients and corresponding *p* values.
Abbreviations: BMI, body mass index; WC, waist circumference; FMI, fat mass index; HC, hip circumference; WHR, waist–hip ratio; WHtR, waist-to-height ratio.

ference, whereas no statistically significant serum Mg associations were observed among males. Dietary Mg showed few associations, with a positive association with hip circumference in males and an inverse association with body fat percentage in females. Overall, these findings suggest sex-stratified patterns in the association between Mg status and adiposity, and they highlight that serum and dietary Mg may reflect different aspects of Mg biology.

4.1 Serum Mg and Adiposity

The inverse associations observed in females align with reports showing lower serum Mg in individuals with greater adiposity. Studies in Canadian and South Asian females reported reduced serum Mg among those with obesity and insulin resistance, with weaker or absent findings in males, consistent with the female-stratified pattern observed in the present study [42]. Rotter et al. [43] reported inverse associations of serum Mg with BMI, WC, and WHR in older men, although estimates varied across cohorts. Other research, including studies from China, reported positive or null results, underscoring heterogeneity by setting

Table 3. Mean differences in serum Mg levels per 1 SD increase in obesity markers in Saudi adults, (n = 776)[1] .

Variable	Males			Females		
	Difference, 10^{-2} mg/dL	95% CI	*p*	Difference, 10^{-2} mg/dL	95% CI	*p*
BMI (kg/m^2)	−0.9	(−8.1, 6.4)	0.81	−7.7	(−14.2, −1.2)	0.02
WC (cm)	2.3	(−50.0, 54.6)	0.93	−37.5	(−80.1, 5.0)	0.08
FMI	−1.4	(−5.7, 2.8)	0.51	−4.5	(−8.5, −0.6)	0.02
HC (cm)	3.2	(−31.5, 37.9)	0.86	−54.3	(−91.5, −17.1)	0.001
WHR	0.01	(−0.02, 0.9)	0.91	0.01	(−0.02, 0.8)	0.42
Body fat percentage	−8.6	(−29.6, 12.3)	0.42	−16.9	(−34.6, 0.8)	0.06
WHtR	0.01	(−0.08, 0.9)	0.89	0.04	(−0.01, 0.6)	0.23

[1] Data are presented as mean difference (95% CI) in serum magnesium concentration per 1 SD increase in each adiposity marker, expressed as 10^{-2} mg/dL (BMI = 6, FMI = 4.7, WC = 17.2, HC = 13.8, WHR = 0.13, fat percent = 10.5, WHtR = 0.1). Models were adjusted for age, smoking status, education, marital status, physical activity, Mg from supplements.
Abbreviations: CI, confidence interval; BMI, body mass index; WC, waist circumference; FMI, fat mass index; HC, hip circumference; WHR, waist–hip ratio; WHtR, waist-to-height ratio.

Table 4. Mean differences in dietary Mg levels per 1 SD increase in obesity markers in Saudi adults, (n = 776)[1] .

Variable	Males			Females		
	Difference	95% CI	*p*	Difference	95% CI	*p*
BMI (kg/m^2)	0.09	(−0.03, 0.12)	0.07	0.08	(−0.03, 0.12)	0.34
WC (cm)	0.10	(−0.02, 0.20)	0.11	0.01	(−0.10, 0.10)	0.66
FMI	0.05	(−0.02, 0.10)	0.24	0.05	(–0.01, 0.08)	0.06
HC (cm)	0.08	(0.04, 0.14)	0.02	0.08	(−0.10, 0.10)	0.89
WHR	0.06	(−0.03, 0.09)	0.45	0.04	(−0.04, 0.08)	0.70
Body fat percentage	−0.15	(−0.4, 0.10)	0.42	−0.18	(−0.31, −0.09)	0.03
WHtR	0.04	(−0.01, 0.07)	0.21	0.05	(−0.03, 0.09)	0.66

[1] Data are presented as mean difference (95% CI) per 1 SD increase in each adiposity marker (BMI = 6, FMI = 4.7, WC = 17.2, HC = 13.8, WHR = 0.13, fat percent = 10.5, WHtR = 0.1). Models were adjusted for age, smoking status, education, marital status, physical activity, and Mg from supplements.
Abbreviations: CI, confidence interval; BMI, body mass index; WC, waist circumference; FMI, fat mass index; HC, hip circumference; WHR, waist–hip ratio; WHtR, waist-to-height ratio.

and design [44]. Biologically, Mg supports insulin receptor signaling and post receptor processes. Low intracellular Mg impairs insulin activity, promotes insulin resistance, and favors fat accumulation, especially in metabolically active depots. Sex differences in body fat distribution may also have contributed to the observed findings. Women generally have a higher body fat percentage and relatively greater gluteofemoral and subcutaneous fat accumulation, whereas men tend to have greater visceral fat deposition. These depot-specific differences may influence how adiposity relates to circulating magnesium [45]. However, because we did not directly measure regional fat depots, this interpretation should be considered tentative.

Although the inverse associations between serum magnesium and adiposity markers were observed only in women, the mechanisms underlying this sex-specific pattern remain uncertain. Experimental studies suggest that estrogen can influence TRPM6-mediated magnesium transport, and some clinical reports have linked estrogen status and hormonal contraceptive exposure with circulating magnesium levels [46,47]. However, our study did not measure menopausal status, reproductive hormones, oral contraceptive use, or hormone therapy. Therefore, any estrogen-related explanation remains speculative and should be interpreted cautiously. Future studies should directly measure these factors to clarify whether they contribute to sex-specific associations between magnesium status and adiposity.

4.2 Dietary Mg and Adiposity

Dietary Mg showed few associations with adiposity, contrasting with cohorts where higher Mg intake correlated with lower BMI and WC, such as Mexican adults and US individuals in NHANES data based on quantile regression [48]. Three factors may explain these differences. First, dietary assessment remains challenging; even with combined FFQs and repeated 24-h recalls, reporting errors persist. Second, residual confounding is likely, as Mg-rich

foods covary with energy intake, fiber levels, and overall diet quality [24]. Third, our relatively young sample may weaken diet–body composition associations. A fourth factor is diet and water in Saudi Arabia. Diets have shifted toward refined grains, sweets, and ultra-processed foods, with lower intakes of fruits, vegetables, fish, nuts, and dairy, patterns that track with higher adiposity and lower magnesium density per calorie [26,49]. This can weaken or confound diet/Mg adiposity links when Mg-rich foods co-occur with higher energy intake or poor overall quality. In addition, much drinking water is desalinated and often contains little magnesium, although national utilities have begun remineralization programs, so background Mg exposure from water can vary by region and time [50,51]. These cultural and environmental features may blunt simple associations between reported Mg intake and adiposity. These factors suggest the need for caution when interpreting weak dietary relationships from a single study.

4.3 Dietary Mg and Circulatory Mg

Dietary magnesium is the primary exogenous source of Mg and therefore upstream of the extracellular (serum) Mg pool. After ingestion, Mg is absorbed along the gastrointestinal tract via two complementary mechanisms: (i) a passive paracellular pathway driven by electrochemical gradients (predominantly in the small intestine) and (ii) an active transcellular pathway mediated by epithelial Mg channels (e.g., TRPM6/7), which becomes relatively more important when luminal Mg is low [17,47,52]. Fractional absorption is adaptive and non-linear; it increases under low habitual intake and decreases when intake is high, limiting large diet-induced fluctuations in serum Mg [52]. Importantly, the amount absorbed depends not only on intake but also on bioavailability, which is influenced by the food matrix and other exposures (e.g., phytate/oxalate-rich patterns, gastrointestinal conditions, or medications such as proton pump inhibitors that can reduce Mg absorption) [18,53].

Once absorbed, Mg enters the extracellular fluid and circulatory compartment, but serum Mg is not a simple proxy for dietary intake because whole-body Mg homeostasis is tightly regulated [17,25]. Most total body Mg is stored in bone and intracellular compartments, while only a small fraction is present in blood, allowing buffering of short-term intake variation [17]. The kidney is the principal regulator of circulating Mg: filtered Mg is largely reabsorbed along the nephron, and urinary excretion can be rapidly adjusted to conserve Mg during low intake or to increase Mg losses when intake is high [53,54]. As a result, serum Mg may remain within the reference range despite chronically low intake and depleted stores, and tends to fall mainly when deficiency is prolonged or when renal/gastrointestinal losses exceed compensatory mechanisms [25].

These physiological features help interpret the different patterns observed for dietary versus serum Mg in our study. Dietary Mg estimated from FFQs captures usual exposure to Mg-rich foods and broader diet quality, whereas serum Mg reflects the current extracellular pool shaped by absorption efficiency, renal handling, redistribution between compartments, inflammation, and medication-related losses [17,18]. Therefore, the unadjusted dietary Mg values in Table 1 should not be interpreted as evidence of higher bioavailable Mg intake among participants with low serum Mg. Dietary Mg and serum Mg provide complementary, not interchangeable, measures and may be jointly informative for understanding Mg-related metabolic risk.

4.4 Limitations and Strengths

The key strengths of this study include a large, well-characterized, community-based sample of Saudi adults, assessment of both serum and dietary Mg levels, and comprehensive evaluation of adiposity and body composition indices. Excluding individuals with medical conditions affecting Mg metabolism strengthened internal validity. Repeated measurements, standardized protocols, and the use of portion models also improved measurement quality.

This study's limitations include the cross-sectional design, which precludes causal inference. Additionally, serum Mg is an imperfect biomarker, as circulating Mg represents ≤1% of body Mg [25,52,53]. The gold-standard Mg loading test, typically employed to identify hypomagnesemia [25] was not feasible in our work.

Furthermore, participants were recruited from community sites and clinics in Riyadh and Al Madinah, and the sample was not designed to be nationally representative. Therefore, the findings should be interpreted as applying to adults recruited from these two urban settings, and generalizability to the wider Saudi population is limited. In addition, body composition was assessed using BIA rather than more precise methods such as DXA or CT/MRI. Although BIA is practical for epidemiologic field studies, DXA provides more accurate estimates of whole-body fat and lean mass, while CT/MRI more precisely assesses visceral adiposity. As for anthropometrics, although standard adult criteria were applied across participants aged 18 to 64 years and age was included as a covariate in the adjusted models, body composition changes with age, particularly in relation to lean mass and fat distribution. Therefore, the use of uniform adult criteria may not fully capture age-related heterogeneity, and future studies should consider age-stratified analyses or age-specific reference values, especially for body-composition indices such as FMI. Moreover, although formal sex-interaction testing supported sex-stratified interpretation for the outcomes, the sex-stratified results should still be interpreted with caution. Men accounted for 37% of the sample, and sex-specific quotas were not prespecified. This imbalance may have reduced precision and statistical power in the male-stratified analyses. Therefore, the lack of statistically significant serum Mg associations in males should be considered inconclu-

sive rather than evidence of a true absence of association. Future studies should use sex-balanced recruitment to confirm whether these sex differences persist. Finally, dietary intake data collected by FFQ and 24-hour recall remain subject to recall error and misestimation, and the frequency of specific Mg-rich food groups, such as whole grains, nuts, legumes, and dark-green vegetables, was not analyzed separately.

4.5 Implications and Next Steps

Our findings suggest that lower serum Mg is linked to higher adiposity in females, whereas dietary Mg shows minimal associations in either sex. Prospective studies and intervention trials in Saudi populations should test whether improving Mg status reduces central adiposity or enhances insulin sensitivity. Future research should incorporate intracellular or functional Mg biomarkers, such as Mg loading tests, and employ repeated dietary assessments with objective recovery markers. Mechanistic studies should also explore sex-specific pathways, including fat distribution and insulin regulation.

To test these pathways, use designs that can separate direction and mechanism. First, run a double-blind randomized trial in Saudi adults with central adiposity and low serum or ionized Mg. Give oral magnesium, for example 300 to 400 mg elemental per day, versus placebo for 16 to 24 weeks. Stratify by sex. Also, add a short crossover feeding study. Provide isocaloric dietary plans that differ only in magnesium density for 4 weeks per period with a washout. Finally, complement trials with a longitudinal cohort that repeats diet, biomarkers, and adiposity every 3 to 6 months, then model bidirectional links. Include a genetic component to enable causal inference using variants in magnesium transport loci.

5. Conclusions

Overall, this cross-sectional study found evidence of sex modified selected associations between Mg status and adiposity markers in Saudi adults. In women, lower serum magnesium was associated with higher BMI, fat mass index, and hip circumference. In men, no significant associations were observed between serum magnesium and adiposity markers. Dietary magnesium intake showed weaker and inconsistent associations with adiposity and body composition. Because participants were recruited from community sites and clinics in Riyadh and Al Madinah, these findings may not be generalizable to all Saudi adults. Longitudinal and clinical studies incorporating additional biomarkers of magnesium status are warranted to confirm these findings and clarify underlying mechanisms.

Availability of Data and Materials

All data reported in this paper will be shared by the lead contact upon request.

Author Contributions

Conceptualization: SAM, GSA; Data curation: FA, MA; Formal analysis: FA; Funding acquisition: SAM, GSA; Investigation: FA, MA; Methodology: FA, SAM, MA, GSA; Project administration: SAM, GSA; Resources: SAM, GSA; Software: FA; Supervision: SAM, GSA; Validation: FA, MA; Visualization: FA; Writing, original draft: FA, MA; Writing, review and editing: FA, SAM, MA, GSA. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study adhered to the Declaration of Helsinki. The KSU Institutional Review Board approved the protocol (KSU-IRB-21-314), and written informed consent was obtained from all participants.

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Conflicts of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work the authors used ChatGPT-3.5 in order to check spelling and grammar. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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