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Journal of Integrated Human Health Sciences

ISSN-3143-8814

Research Article

Low Serum Magnesium and Albuminuria Despite Preserved eGFR in U.S. Adults: NHANES 2021–2023

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Received date: 02 Apr 2025: **Accepted date:** 21 Apr 2025: **Published date:** 28 Apr 2025

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Abstract

Background: Low serum magnesium has been linked to diabetes, vascular dysfunction, and incident kidney disease, but its association with albuminuria in the contemporary general U.S. population is less clear.

Objectives: This investigation focused on serum magnesium, UACR, and eGFR among subjects with preserved kidney function.

Methods: We included adults aged ≥20 years (NHANES August 2021–August 2023) with serum magnesium, urinary albumin-to-creatinine ratio (UACR), creatinine-derived estimated glomerular filtration rate (eGFR), and required covariates. The main analysis was limited to eGFR ≥60 mL/min/1.73 m² to lower confounding by advanced filtration impairment. UACR indicates that albuminuria ≥30 mg/g The analysis controlled for variables such as age, sex, race/ethnicity, body mass in dex, diabetes, high blood pressure, and smoking and family income-to-poverty ratio.

Results: A total of 4850 adults with preserved eGFR were included in the descriptive cohort. 4215 had complete covariate data for the primary model. In patients with serum magnesium <1.7 mg/dL, 32.1% had a weighted prevalence of albuminuria, while the figures were 9.9%, 6.1%, and 5.6% for serum levels of 1.7-1.9 mg/dL, 2.0-2.1 mg/dL and ≥2.2 mg/dL respectively. When mg/dL are compared with ≥2.2 mg, that was associated with 2.55-fold higher adjusted albuminuria prevalence. (95% CI 1.61–4.04). Each increase of 0.1 mg/dL in magnesium levels was linked with a lower likelihood of albuminuria by 13.3%+++. Adults who have diabetes and those who do not have it also showed associations. Magnesium diebetes interaction was not significant. We saw persistence of the results when excluding self-reported kidney disease and when restricting to eGFR ≥90.

Conclusions: Lower serum magnesium was linked to albuminuria even at normal glomerular filtration. The findings from the cross-sectional analysis suggest that serum magnesium could be a marker of early renal or systemic microvascular stress, however they do not confirm that magnesium deficiency causes albuminuria or that supplementation will prevent kidney disease.

Keywords: Magnesium, Albuminuria, UACR, Kidney Disease, Microvascular Injury, NHANES

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Introduction

Magnesium is an essential intracellular cation and enzymatic cofactor involved in energy metabolism, ion transport, vascular tone, glucose homeostasis and cellular signaling. The human body regulates the concentration of magnesium in association with intestinal absorption, renal handling, and distal nephron that influences the systemic balance of magnesium [1,2]. Magnesium is not measured routinely in many ambulatory sectors despite its broad physiology and modest disturbances remain asymptomatic.

Due to low concentrations that occur with diabetes, hypertension, insulin resistance, inflammation, cardiovascular disease, interest in magnesium has increased. In a JAMA article, other researchers in Huichol, with contemporary NHANES 2021–2023 data, found that mean serum magnesium is lower in U.S. adults with diabetes, hypertension and chronic kidney disease than in metabolically healthy adults [3]. Studies conducted on experiments and patients mention that lesser availabilities of magnesium are associated with endothelial dysfunction, oxidative stress, inflammatory signaling, and impaired microvascular integrity. They can also plausibly interfere with glomerular albumin permeability [1,4].

Measures of albuminuria are particularly useful for studying this possibility, as they can pinpoint kidney or systemic vascular injury, even when filtration is apparently preserved. UACR provides prognostic information in addition to eGFR and is an independent marker for the subsequent failure of the kidney, cardiovascular event and mortality [57]. As such, restricting a study to preserved eGFR may aid in differentiating an early albuminuric phenotype from major changes in Mg handling that supervene with more advanced loss of filtration.

Although previous studies have reported inverse associations between serum magnesium and albuminuria, most were performed in patients with diabetes and selected... In Chinese studies, diabetic patients with microalbuminuria have lower magnesium [8,9] and 2024 paper reported substantially more albuminuria in adults with type 2 diabetes and hypomagnesemia [10]. Additionally, the ARIC study found low serum magnesium to be associated with incident CKD and end-stage kidney disease with long-term follow-up of adults with baseline eGFR ≥60 mL/min/1.73 m² [11].

The study at hand investigated whether there is any association between serum magnesium and albuminuria using data from NHANES 2021–2023. In order to lessen the likelihood that low filtration per se was driving magnesium concentrations, the main analysis was restricted to adults with eGFR ≥60 mL/min/1.73 m². We also examined severe albuminuria, used UACR as a continuous variable, analysed diabetes-specific associations, and conducted sensitivity analyses among adults with eGFR ≥90 and with no previous kidney diagnosis.

Materials and Methods

Study design and population

This cross-sectional investigation employed public data from the National Center for Health Statistics, NHANES August 2021–August 2023. A complex multistage probability design is utilized by NHANES to target the U.S. population. Adults who are above 20 years. Pregnant subjects were omitted. The initial analytical group needed serum

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magnesium, serum creatinine, UACR, eGFR ≥60 mL/min/1.73 m², and the survey phlebotomy weight. Prespecified covariates were additionally required for complete-case regression.

Serum magnesium

The NHANES standard biochemical profile (LBXMAGN) contains serum magnesium which is quantified in mg/dL. Managing laboratory-specific measurement difficulties is a part of centralized laboratory measurement quality monitoring and reporting [12]. We modeled magnesium continuously per 0.1 mg/dL and in clinically interpretable categories: <1.7, 1.7–1.9, 2.0–2.1, and ≥2.2 mg/dL. The category of <1.7 mg/dL is clearly low according to modern U.S. reference intervals [3].

Kidney measures

An analysis of spot urine levels gives the urinary albumin-to-creatinine ratio. The definition of albuminuria is UACR ≥30 mg/g severely albuminuria UACR ≥300 mg/g Serum creatinine the 2021 CKD-EPI creatinine equation without race [13] was utilized to determine eGFR. Only patients with eGFR greater than or equal to 60 mL/min/1.73 m² were included as primary study population. We performed a sensitivity analysis that restricted participants to eGFR ≥90.

Covariates

The applied model took into account age, gender, race / ethnicity, weight, diabetes, high blood pressure, current cigarette smoking and family income / poverty ratio. A diagnosis of diabetes was classi&ed as per self-reported physician diagnosis or HbA1c ≥6.5%. According to the definition, the self-reported diagnosis or mean measured systolic blood pressure of ≥130 mmHg or diastolic blood pressure ≥80 mmHg was hypertension. They calculate mean blood pressure from available NHANES measures.

Statistical analysis

The NHANES phlebotomy weight (WTPH2YR), strata and primary sampling units were used in the analyses. Weighted averages and descriptive statistics were calculated across magnesium categories. To determine the prevalence ratios and 95% confidence intervals for albuminuria, survey-weighted modified Poisson regression with design-based variance estimation was employed. Magnesium was modeled continuously per 0.1 mg/dL and categorically reference of ≥2.2 mg/dL. A complementary analysis was performed using a survey-weighted linear model of log-transformed UACR. We tested for effect modification by diabetes using a multiplicative interaction term. A sensitivity analysis of adult patients that reported weak or failing kidneys and limited the cohort to eGFR ≥90 mL/min/1.73 m². Statistical significance was defined as $p < 0.05$.

Results

Serum magnesium distribution and albuminuria

A total of 4850 adults with preserved eGFR and measured serum magnesium comprised the descriptive cohort. The clinically interpretable concentration bands were more informative than equal-sized quartiles due to 0.1 mg/dl reporting of magnesium values. Around 3.1% of the weighted population had magnesium levels less than 1.7 mg/dL.

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38.6% had magnesium concentrations between 1.7–1.9 mg/dL. 45.9% had levels of 2.0–2.1. Lastly, 12.4% of the population had concentrations 2.2 mg/dL or more (Table 1).

Serum magnesium	Unweighted (N)	Weighted population, %	Albuminuria, %
<1.7 mg/dL	189	3.1	32.1
1.7–1.9 mg/dL	1841	38.6	9.9
2.0–2.1 mg/dL	2200	45.9	6.1
≥2.2 mg/dL	620	12.4	5.6

Table 1. Serum magnesium distribution and albuminuria

Concentration gradient was strong in Albuminuria Weighted prevalence was 32.1% among adults with a magnesium level of <1.7 mg/dL, compared to 9.9% with a level of 1.7–1.9 mg/dL and so on. Similarlysevere increase in albuminuria was seen in this case (Figure 1).

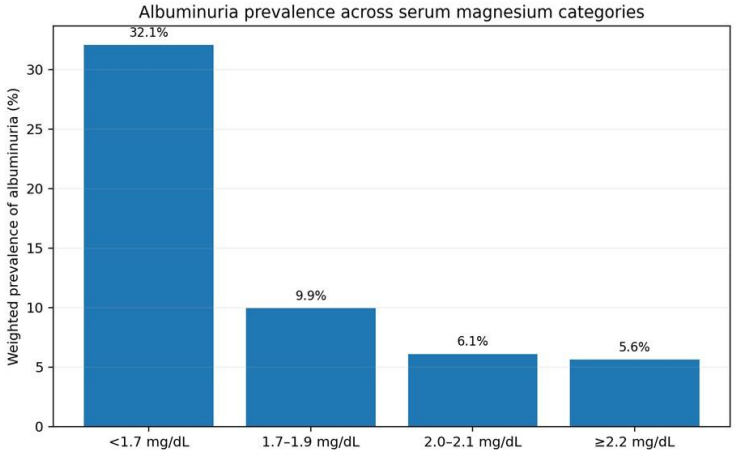


Figure 1. Survey-weighted prevalence of albuminuria by serum magnesium category among adults with eGFR ≥60 mL/min/1.73 m²

Adjusted association with albuminuria

In the complete-case model (n=4215) adjustment for demographics, metabolites, vascular, smoking and socioeconomics found lower magnesium remain associated with albuminuria. Lower concentrations of magnesium, compared to magnesium greater than or equal to 2.2 mg/dL, were associated with 2.55-fold higher prevalence of albuminuria. The prevalence of albuminuria in the 1.7–1.9 mg/dL category showed a directional association (PR 1.54, 95% CI 0.96–2.47; p=.073) whereas 2.0–2.1 mg/dL category was not different from reference (Table 2, Figure 2).

Magnesium category	Adjusted PR	95% CI	p value
<1.7 mg/dL	2.55	1.61–4.04	<.001
1.7–1.9 mg/dL	1.54	0.96–2.47	0.073
2.0–2.1 mg/dL	1.12	0.60–2.09	0.709
≥2.2 mg/dL	1.00	1.00–1.00	Reference

Table 2. Adjusted association with albuminuria

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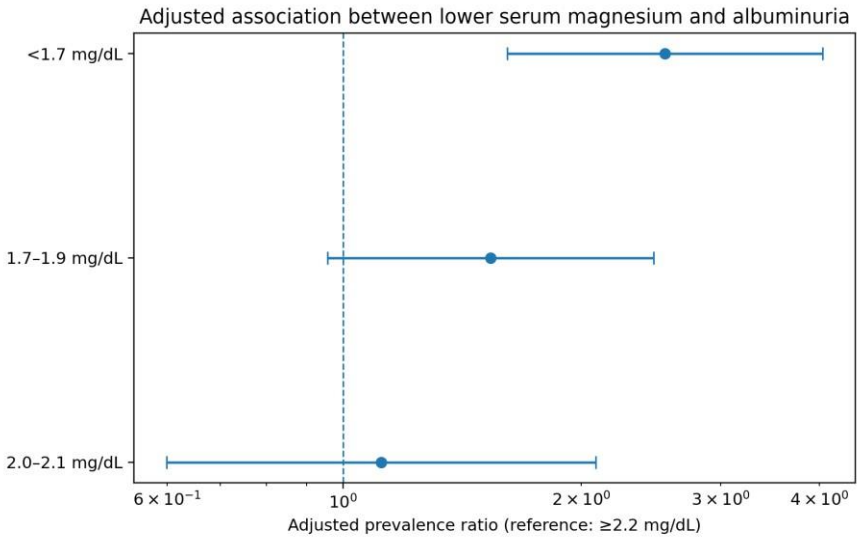


Figure 2. Adjusted prevalence ratios for albuminuria across serum magnesium categories. Reference: ≥2.2 mg/dL

When serum magnesium was modeled to increase 0.1 mg/dL, albuminuria's adjusted prevalence was 13.3% lower (PR 0.867, 95% CI 0.833–0.903; p<0.001). The relationship showed up for severely raised albuminuria too (PR 0.822, 95% CI 0.761–0.889; p<0.001). In the continuous UACR model, each 0.1 mg/dL higher magnesium was associated with a lower log UACR (β=-0.058; p=0.001), equivalent to an approximately 5.6% lower geometric-mean UACR.

Diabetes-stratified and sensitivity analyses

Diabetes was not the only thing affected inversely. For adults without diabetes, the adjusted PR for obtaining a 0.1 mg/dL higher magnesium was 0·844 (95% CI 0·793–0·898; p<0.001). The PR was 0.877 (95% CI 0.829–0.929; p<0.001) among adults with diabetes. The interaction of magnesium and diabeteswas not statistically significant (p=0.290), implying that the association was similar throughout glycemic groups (Figure 3).

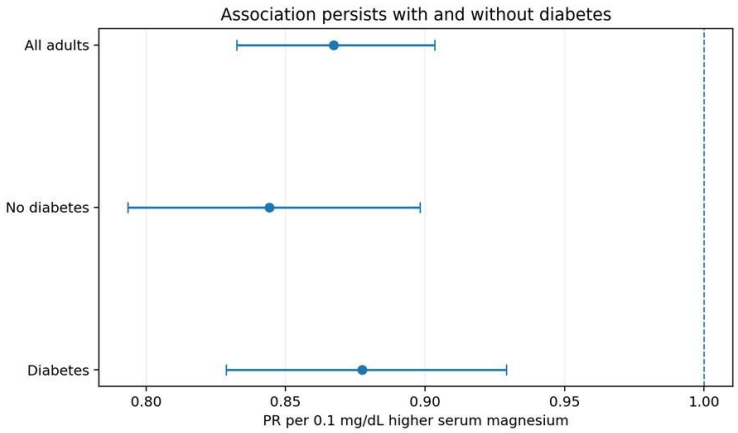


Figure 3. Adjusted association of serum magnesium with albuminuria overall and stratified by diabetes status

The findings were also strong against stricter kidney definitions. After excluding people who indicated having been told they had weak or failing kidneys, the PR per 0.1 mg/dL higher magnesium was 0.871 (95% CI 0.835–0.908;

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p<.001). The correlation remained substantial with eGFR scores more than or equal to 90 mL/min/1.73 m² (PR 0.860; 95% CI 0.802 to 0.923; p<.001).

Discussion

This study helps in understanding contemporary US adults with preserved eGFR, where a lower magnesium level led to albuminuria risk. Within the examined concentration range, a graded relationship was established, particularly evident at evidently low magnesium concentrations. Compared with those at serum magnesium levels ≥2.2 mg/dL (1.3), those with serum magnesium levels <1.7 mg/dL (1.8) had more than doubled adjusted prevalence of albuminuria, while every 0.1 mg/dL higher magnesium level was associated with around 13% lower prevalence of albuminuria.

It is important to restrict interpretation to eGFR ≥60. The kidneys have a vital role in the homeostasis of magnesium, and advanced kidney dysfunction can alter circulating magnesium concentrations [1,4]. By excluding adults with obvious filtration impairment, the analysed reduces – but cannot eliminate – the probability that the correlation simply reflects reduced renal clearance or advanced CKD. The fact that those with eGFR ≥90 and without prior kidney diagnosis still exhibit the finding suggests magnesium may track with earlier renal (and/or systemic microvascular) phenotype.

The results show that it may also be applicable to non-diabetic conditions Xu and others [9] observed significantly greater microalbuminuria in diabetic adults, whose serum magnesium was lower. More than 3,000 patients with diabetes were evaluated. More than 3,000 diabetic patients were studied by Lu et al. [8]. The authors, Eker and Ataoğlu [10], mentioned that hypomagnesemia is associated with albuminuria more than three times in type 2 diabetes. Our evaluation suggests that this relationship is not restricted to diabetes: in both diabetic and non-diabetic adults, the inverse association remained statistically significant, while formal testing for interaction indicated no meaningful heterogeneity by diabetes status.

Research outcomes are also consistent with longitudinal evidence. In ARIC study, low serum magnesium at baseline predicted incident chronic kidney disease (CKD) and end-stage kidney disease (ESKD) with a median follow-up of over two decades in the population with baseline eGFR ≥60 mL/min/1.73 m² [11]. This potential connection does not establish cause and effect. Yet, this finding along with albuminuria findings suggests that magnesium may be associated with renal risk before clinically-advanced filtration loss.

There are numerous biological pathways that could link low magnesium to albumin leakage. Magnesium assists endothelial stability, vascular tone, insulin signalling, and cellular redox balance [1,2]. Induced magnesium deficiency causes free radical signaling and damages endothelial barriers. The connection between low magnesium and increased UACR can be linked with systemic endothelial stress, owing to the dependence of glomerular albumin permeability on an unbroken endothelial–basement–podocyte membrane filtration barrier. A lack of magnesium might also enhance insulin resistance, sympathetic and renin–angiotensin activity, as well as vascular stiffness. This condition may consequently augment intraglomerular hemodynamic stress.

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Nonetheless, the relationship should not be taken to mean that magnesium supplementation prevents albuminuria. Findings from cross-sectional studies cannot establish if low magnesium causes glomerular injury, if early renal disease alters magnesium balance or if both are caused by common causes like diet, diabetes severity, diuretic use etc. According to Halawa and colleagues [14], only small intervention studies are available in diabetic nephropathy reporting the effect of magnesium supplementation on microalbuminuria, with variable results and not powerful enough to recommend population treatment.

Our findings are particularly valuable at this time as a 2026 analysis of the same NHANES cycle provided updated reference intervals for U.S. serum magnesium. Furthermore, it showed lower mean magnesium among adults with diabetes, hypertension, and CKD [3].

Today's analysis deals with a different question; it does not define reference values for a population but examines whether magnesium is related to albuminuria while filtration is still preserved. Magnesium distribution being lower at gradient shows that kidney and vascular phenotyping could be relevant when serum magnesium is unexpectedly low in a clinical setting.

Highly significant laboratory finding is albuminuria itself. The contemporary guidance on chronic kidney disease considers urine albumin-to-creatinine ratios (UACRs) and estimated glomerular filtration rates (eGFRs) as complementary dimensions of kidney risk. Large meta-analyses show that albuminuria independently predicts kidney failure, cardiovascular events and mortality [5,6,7]. Before the eGFR drops, we can establish modifiable or informative correlates of albuminuria. This show benefits for risk detection even without causality being established.

There are many strengths in this study. We utilize a recent nationally representative sample, objective serum magnesium and HbA1c measurements, measured blood pressure, UACR, the 2021 race-free CKD-EPI equation, and the NHANES complex survey design. Limiting the main cohort to preserved eGFR and repeating the analysis at eGFR ≥90 tackles an important source of reverse association. The study also examined magnesium continuously, categorically and UACR as binary and continuous outcome.

There are essential bounds. Since NHANES is cross-sectional, temporality cannot be established. Serum magnesium does reflect some total-body magnesium; however, because most magnesium is intracellular or in bone, it is an imperfect measure. UACR and magnesium were both reported at a one off examination, and albuminuria may be transient; for a clinical CKD the requirement is chronicity [6]. Magnesium status may be confounded by detailed medication classes–especially diuretics, proton-pump inhibitors and magnesium-containing supplements–that were not incorporated. Factors related to diet and the gastrointestinal system may still play a role. Ultimately, while low magnesium is significantly associated with the outcome, the precision of any detailed subgroup analyses is limited by the relatively small sample size of patients with concentrations <1.7 mg/dl.

The data demonstrates a consistent correlation between lower serum magnesium levels and albuminuria in adults with preserved eGFR. The observation aligns with previous findings on diabetes and long-term kidney complications; however, it should be considered a marker-relationship and a hypothesis for future mechanistic and prospective studies rather than evidence for a supplementation therapy.

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Conclusions

In NHANES 2021–2023, lower serum magnesium was independently associated with a higher prevalence of albuminuria among U.S. adults with eGFR ≥60 mL/min/1.73 m². Lower serum magnesium (<1.7 mg/dL) were associated with roughly 2.6-fold higher adjusted albuminuria prevalence. Moreover, the inverse was applicable to adults with and without diabetes. Furthermore, it was after excluding known kidney disease. In addition, it was in adults with eGFR ≥90. Low serum magnesium may indicate early renal or systemic microvascular stress, according to the findings of these studies. There is a need for prospective investigations that involve repeated measurements of magnesium and UACR, medication information, and randomized supplementation where appropriate to determine causation and clinical utility.

Declarations

Ethics statement. NHANES protocols were approved by the National Center for Health Statistics Research Ethics Review Board, and participants provided informed consent. The present study used publicly available de-identified data.

Data availability. NHANES August 2021–August 2023 data are publicly available through the U.S. Centers for Disease Control and Prevention/National Center for Health Statistics.

Funding. No specific funding was received for this secondary analysis.

Conflict of interest. The authors declare no conflict of interest.

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