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Sodium Intake and Six-Month Kidney Function in Adults with Diabetic Kidney Disease: An Observational Study

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ABSTRACT: Dietary management is an important component of diabetic kidney disease (DKD) care, although evidence linking habitual nutrient intake with short-term kidney-function trajectories remains limited. We examined the associations of protein, fat, carbohydrate, and sodium intake with six-month kidney function in adults with DKD. This observational analytic study included 50 adults with type 2 diabetes and chronic kidney disease. Habitual dietary intake was assessed using a semiquantitative food-frequency questionnaire supported by 24-hour dietary recall. Six-month estimated glomerular filtration rate (eGFR) was modeled with adjustment for baseline eGFR. Separate linear regression models evaluated continuous dietary exposures, while sodium intake was additionally examined using a predefined threshold of <2300 versus ≥2300 mg/day. The cohort predominantly had advanced CKD, with 76.0% classified as G4–G5 and 96.0% having urinary albumin-to-creatinine ratio ≥30 mg/g. Mean sodium intake was 2541±742 mg/day. Continuous protein, fat, carbohydrate, and sodium intake were not associated with six-month eGFR. In contrast, sodium intake ≥2300 mg/day was associated with a lower six-month eGFR after adjustment for baseline eGFR, age, sex, HbA1c, and systolic blood pressure (B=−8.82 mL/min/1.73 m²; 95% CI −16.22 to −1.43; p=0.021). Adjusted mean six-month eGFR was 31.57 mL/min/1.73 m² for sodium intake <2300 mg/day and 22.75 mL/min/1.73 m² for intake ≥2300 mg/day. The association was attenuated but remained directionally consistent after exclusion of influential observations (B=−5.46; 95% CI −10.89 to −0.02; p=0.049). These findings identify a clinically relevant categorical association between sodium intake and short-term kidney function in DKD, while not supporting a linear dose-response relationship. Larger prospective studies with repeated dietary and objective sodium assessment are warranted.

1. INTRODUCTION

Diabetic kidney disease (DKD) is a major complication of type 2 diabetes and an important cause of kidney failure, while chronic kidney disease in people with diabetes also increases cardiovascular risk. Its phenotype is heterogeneous: albuminuria and estimated glomerular filtration rate (eGFR) do not necessarily progress in parallel [1,2]. Management therefore combines disease-modifying pharmacotherapy with lifestyle and nutritional care [1].

Nutritional management in DKD requires balancing kidney protection with nutritional status. Current guidelines favor individualized dietary care, controlled protein intake, and sodium restriction [3]. KDOQI recommends sodium <2.3 g/day in CKD to improve blood pressure and volume control and reduce proteinuria [4]. Evidence for sodium is strongest for

intermediate outcomes. Sodium restriction reduces blood pressure and albuminuria in DKD [5], whereas a Cochrane review found lower blood pressure but no clear change in GFR, with low-certainty evidence and few participants with reduced kidney function [6]. In broader CKD populations, pooled data suggested fewer composite renal events but no significant improvement in eGFR decline, with substantial heterogeneity [7]. A single-arm study in diabetes and early CKD similarly reduced self-reported sodium intake and blood pressure without improving albuminuria [8].

Evidence for protein, carbohydrate, and fat intake is less uniform; nutrient source and quality may be as important as quantity [9]. Indonesian national survey data underscore the local relevance of CKD and its association with diabetes but do not address longitudinal diet–kidney function relationships [10]. This study examined dietary protein, fat, carbohydrate, and sodium intake in relation to six-month kidney function in adults with DKD, including a prespecified analysis using a 2300-mg/day sodium threshold.

2. MATERIALS AND METHODS

2.1 Study design and participants

This observational analytic study used a retrospective longitudinal assessment of kidney function and was conducted at RSUP Dr. Wahidin Sudirohusodo, Makassar, Indonesia, from May to June 2026. Adults aged ≥18 years with type 2 diabetes mellitus and chronic kidney disease documented in the medical record were eligible. Participants underwent current clinical, anthropometric, dietary, and laboratory assessments, while kidney-function measurements obtained approximately six months earlier were retrieved from medical records. A total of 50 participants with complete data were included in the analysis.

Patients with type 1 diabetes mellitus, acute kidney injury, malignancy, pregnancy or lactation, or incomplete dietary or laboratory data required for the analysis were excluded. The term diabetic kidney disease (DKD) was used for the study population because participants had established type 2 diabetes and chronic kidney disease under nephrology care; the analysis did not independently establish the etiologic attribution of CKD beyond the clinical diagnosis.

2.2 Dietary assessment

Habitual dietary intake during the preceding six months was assessed using a semiquantitative food-frequency questionnaire, with a 24-hour dietary recall used as supporting dietary information. Daily nutrient intake was summarized as protein (g/day and g/kg body weight/day), fat (g/day), carbohydrate (g/day), sodium (mg/day), fiber (g/day), and potassium (mg/day).

The primary dietary exposures were protein, fat, carbohydrate, and sodium intake. For regression analyses, coefficients were expressed per 0.1 g/kg/day of protein, 10 g/day of fat, 10 g/day of carbohydrate, and 500 mg/day of sodium. Sodium intake was also examined categorically using the predefined threshold of <2300 versus ≥2300 mg/day. Protein intake was categorized as <0.8, 0.8–1.2, and >1.2 g/kg/day for descriptive and sensitivity analyses.

2.3 Kidney-function assessment and outcomes

Serum urea and creatinine were available at baseline and approximately six months later. Estimated glomerular filtration rate (eGFR) was derived using the 2021 CKD-EPI creatinine equation. GFR categories were defined as G1 ≥90, G2 60–89, G3a 45–59, G3b 30–44, G4 15–29, and G5 <15 mL/min/1.73 m².

For the present analysis, six-month eGFR was modeled as the primary continuous outcome, with baseline eGFR included as a covariate. Changes in urea, creatinine, and eGFR were calculated as the six-month value minus the baseline value. DKD progression was defined as worsening of GFR category accompanied by a ≥25% decline in eGFR from baseline. Because only six participants met this composite endpoint, progression was summarized descriptively and was not modeled using multivariable logistic regression.

2.4 Clinical and nutritional covariates

Covariates included age, sex, body mass index, diabetes duration, systolic and diastolic blood pressure, hemoglobin, HbA1c, serum albumin, and urinary albumin-to-creatinine ratio (UACR). UACR was classified as <30, 30–300, or >300 mg/g. The Prognostic Nutritional Index (PNI) was calculated as:

PNI = 10 × serum albumin (g/dL) + 0.005 × total lymphocyte count (/mm³).

2.5 Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics version 30.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro–Wilk test. Normally distributed variables are presented as mean ± standard deviation, non-normally distributed variables as median (interquartile range), and categorical variables as frequency and percentage.

Changes in serum urea, creatinine, and eGFR between baseline and six months were evaluated using the Wilcoxon signed-rank test. Six-month eGFR was used as the primary continuous outcome in regression analyses, with baseline eGFR included as a covariate to account for initial kidney function.

Associations of protein, fat, carbohydrate, and sodium intake with six-month eGFR were examined using separate ordinary least-squares linear regression models to limit overfitting given the sample size. Continuous dietary exposures were expressed per 0.1 g/kg/day of protein, 10 g/day of fat, 10 g/day of carbohydrate, and 500 mg/day of sodium and were adjusted for baseline eGFR, age, and sex. Sodium intake was additionally evaluated using the predefined threshold of <2300 versus ≥2300 mg/day. The categorical sodium model was further adjusted for HbA1c and systolic blood pressure.

Adjusted six-month eGFR according to sodium category was estimated using the general linear model and reported as estimated marginal means with 95% confidence intervals. A sodium-by-sex interaction was examined as an exploratory effect-modification analysis. Multicollinearity was assessed using tolerance and variance inflation factors. Model influence and residual diagnostics included standardized residuals, leverage values, and Cook's distance. A sensitivity analysis was performed after excluding two influential observations identified by Cook's distance >1 and absolute standardized residuals >2.5. All tests were two-sided, and $p < 0.05$ was considered statistically significant.

3. RESULTS AND DISCUSSION

3.1 Participant characteristics

A total of 50 adults with diabetic kidney disease were included. The mean age was 54.8 ± 10.7 years, and 25 participants (50.0%) were female. The cohort predominantly had advanced kidney disease, with 38 participants (76.0%) classified as CKD G4–G5 and 48 (96.0%) having UACR ≥30 mg/g. Median baseline eGFR was 15.5 (11.0–29.5) mL/min/1.73 m². Mean protein intake was 1.17 ± 0.43 g/kg/day, and mean sodium intake was 2541 ± 742 mg/day (Table 1).

Table 1. Baseline clinical and dietary characteristics of the study participants (n=50)

Characteristic	Value
Clinical characteristics	
Age, years	54.8 ± 10.7
Female sex	25 (50.0)
BMI, kg/m²	17.92 (17.33–19.35)
Diabetes duration, years	4.50 (2.00–8.50)
Systolic blood pressure, mmHg	145.0 (134.5–156.0)
Diastolic blood pressure, mmHg	78.5 (68.0–83.3)
Hemoglobin, g/dL	9.30 (8.30–10.53)
HbA1c, %	6.04 (5.58–6.72)
Albumin, g/dL	3.36 ± 0.70
Prognostic Nutritional Index	41.50 ± 8.63
Baseline eGFR, mL/min/1.73 m²	15.5 (11.0–29.5)

Advanced CKD (G4–G5)	38 (76.0)
UACR ≥30 mg/g	48 (96.0)
Dietary intake	
Protein, g/day	52.94 ± 20.68
Protein, g/kg/day	1.17 ± 0.43
Fat, g/day	44.45 (31.03–59.70)
Carbohydrate, g/day	242.25 (156.05–282.55)
Fiber, g/day	9.30 (6.50–14.43)
Sodium, mg/day	2541 ± 742
Potassium, mg/day	1161.1 (988.0–1359.4)

Note: Data are presented as mean ± SD, median (IQR), or n (%), as appropriate.

3.2 Six-month kidney-function changes

At six months, median serum creatinine decreased from 3.65 to 2.85 mg/dL (p=0.037). Median eGFR increased from 15.5 to 22.5 mL/min/1.73 m², although the change was not statistically significant (p=0.080). Eight participants (16.0%) experienced an eGFR decline ≥25%, 11 (22.0%) had worsening GFR stage, and six (12.0%) met the composite definition of DKD progression (Table 2).

Table 2. Changes in kidney-function measures over six months

Outcome	Baseline	6 months	Change	P value
Urea, mg/dL	68.5 (38.8–101.3)	51.0 (34.0–73.5)	−3.5 (−33.8 to 14.8)	0.140
Creatinine, mg/dL	3.65 (2.12–5.32)	2.85 (2.24–3.52)	−0.10 (−1.64 to 0.20)	0.037
eGFR, mL/min/1.73 m²	15.5 (11.0–29.5)	22.5 (14.0–28.8)	1.0 (−3.0 to 10.3)	0.080
eGFR decline ≥25%	—	8 (16.0)	—	—
Worsening GFR stage	—	11 (22.0)	—	—
Composite DKD progression	—	6 (12.0)	—	—

Note: Continuous data are presented as median (IQR). P values were obtained using Wilcoxon signed-rank tests. Change was calculated as the 6-month value minus the baseline value.

3.3 Dietary intake and six-month eGFR

Continuous protein, fat, carbohydrate, and sodium intake were not independently associated with six-month eGFR after adjustment for baseline eGFR, age, and sex (all p>0.05) (Table 3). In contrast, sodium intake ≥2300 mg/day was associated with lower six-month eGFR after additional adjustment for HbA1c and systolic blood pressure (B=−8.82 mL/min/1.73 m²; 95% CI −16.22 to −1.43; p=0.021).

Table 3. Associations of dietary intake with six-month eGFR

Dietary exposure	Adjusted B (95% CI), mL/min/1.73 m²	P value
Protein, per 0.1 g/kg/day	−0.23 (−1.10 to 0.64)	0.596
Fat, per 10 g/day	−0.31 (−1.60 to 0.97)	0.624
Carbohydrate, per 10 g/day	−0.17 (−0.57 to 0.23)	0.400

Sodium, per 500 mg/day	−1.06 (−3.59 to 1.48)	0.406
Sodium ≥2300 vs <2300 mg/day	**−8.82 (−16.22 to −1.43)**	**0.021**

Note: Continuous exposure models were adjusted for baseline eGFR, age, and sex. The categorical sodium model was additionally adjusted for HbA1c and systolic blood pressure. Negative coefficients indicate lower six-month eGFR.

The sodium-by-sex interaction was not statistically significant (p=0.120). In sensitivity analysis excluding two influential observations, the association was attenuated but remained in the same direction (B=−5.46; 95% CI −10.89 to −0.02; p=0.049). Adjusted mean six-month eGFR was 31.57 mL/min/1.73 m² (95% CI 25.86–37.28) among participants consuming <2300 mg/day and 22.75 mL/min/1.73 m² (95% CI 18.34–27.16) among those consuming ≥2300 mg/day (Figure 1).

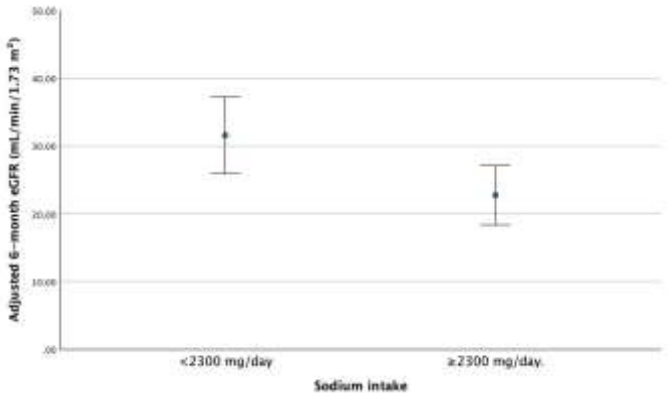

Figure 1. Adjusted six-month estimated glomerular filtration rate according to sodium intake category. Points represent estimated marginal means and error bars represent 95% confidence intervals. Estimates were adjusted for baseline eGFR, age, sex, HbA1c, and systolic blood pressure.

3.4 Discussion

The principal finding of this study was that participants with sodium intake ≥2300 mg/day had a lower adjusted six-month eGFR than those consuming <2300 mg/day. The adjusted difference was −8.82 mL/min/1.73 m² and remained in the same direction, although attenuated, after exclusion of two influential observations. In contrast, sodium analyzed as a continuous exposure was not associated with six-month eGFR, and no independent associations were observed for protein, fat, or carbohydrate intake. The pattern therefore does not establish a linear sodium–eGFR relationship or a biological threshold, but identifies a clinically relevant categorical association that merits further investigation. Contemporary CKD guidance continues to incorporate dietary sodium modification within comprehensive kidney-protective care while emphasizing individualized nutritional management [1,11].

The sodium finding is consistent with the rationale underlying existing dietary recommendations, although previous evidence is considerably stronger for blood pressure and albuminuria than for preservation of GFR. In DKD, Chen et al. (2022) found that sodium restriction reduced systolic and diastolic blood pressure and urinary albumin excretion [5]. The Cochrane review by Hodson and Cooper (2023) similarly reported lower blood pressure with sodium reduction but no clear change in GFR; importantly, evidence certainty was low and most participants had normal or only mildly impaired kidney function [6]. Shi et al. (2022), examining a broader CKD population, reported fewer composite renal events with low-salt diets but no significant improvement in the rate of eGFR decline [7]. Dietary counseling in adults with diabetes and early CKD also reduced reported sodium intake and blood pressure without improving albuminuria [8]. These differences in populations, outcomes, and follow-up help explain why the relation between sodium exposure and longitudinal kidney function remains less settled than its effects on blood pressure.

The categorical result should not be interpreted as evidence that 2300 mg/day represents a biological breakpoint. The cutoff was defined a priori and has a clinical basis: KDOQI recommends sodium intake below 2.3 g/day in CKD, whereas KDIGO 2024 recommends <2 g/day [4,11]. Dietary sodium is also difficult to quantify precisely at the individual level. Judge et al.

(2021) noted that dietary recalls may be useful for population-level estimation but are less suited to precise estimation of an individual's habitual intake [12]. CKD introduces additional challenges, including altered sodium handling and recall-related measurement error [13]. Such error could weaken a continuous association and should temper interpretation of the categorical contrast.

The absence of an association between protein intake and six-month eGFR is compatible with uncertainty in the intervention literature. Li et al. (2021) reported slower GFR decline and lower proteinuria with protein intake <0.8 g/kg/day in a meta-analysis of nine trials [14]. In contrast, the more recent Cochrane review of eight studies concluded that the effect of low-protein diets on GFR decline remains uncertain, with low or very low certainty of evidence and substantial problems with adherence [15]. Protein source may also be relevant: Eckert et al. (2022) found weak evidence favoring diets lower in animal or red-meat protein in short-term crossover studies, whereas parallel trials were not significant [16]. Together with evidence emphasizing nutrient quality rather than quantity alone [9], these findings caution against interpreting the present null protein association as evidence that protein management is unimportant. This is particularly relevant in a cohort with predominantly advanced CKD and relatively low BMI, where dietary restriction should be individualized to nutritional status; protein-energy wasting in nondialysis CKD has itself been associated with adverse long-term outcomes [17].

The null findings for carbohydrate and fat quantity warrant similar caution. Broader CKD literature suggests that whole-diet characteristics may carry information not captured by absolute macronutrient intake. Quintela et al. (2021) found that dietary patterns rich in fruits, vegetables, and fiber were generally associated with more favorable CKD outcomes, although the underlying studies were observational [18]. He et al. (2021) likewise reported a lower risk of CKD with healthier dietary patterns and a higher risk with Western-type patterns, but these data primarily concern CKD occurrence rather than short-term progression of established DKD [19]. Consistent with this distinction, a meta-analysis of randomized lifestyle interventions found improvements in creatinine, albuminuria, and blood pressure without a significant pooled improvement in eGFR [20].

Several limitations should frame interpretation. The study was single-center and included only 50 participants, most with advanced CKD; only six met the composite progression definition, preventing reliable multivariable modeling of that endpoint. Kidney function was represented by two measurements over approximately six months, while habitual intake was assessed by semiquantitative FFQ supported by dietary recall rather than repeated objective sodium measurements. Medication exposure, changes in clinical management, volume status, and other unmeasured factors may have introduced residual confounding, and previous dietary counseling could have influenced reported intake. Even urinary sodium methods have important limitations in CKD: Kurzhagen et al. (2024) demonstrated appreciable bias when spot urine equations were compared with measured 24-hour sodium excretion [21]. Nevertheless, adjustment for baseline eGFR and relevant clinical covariates, formal influence diagnostics, and sensitivity analysis provide additional support for the internal consistency of the categorical sodium finding.

The present results therefore support further investigation of sodium exposure in DKD rather than a causal conclusion that intake ≥2300 mg/day accelerates disease progression. Larger prospective studies with repeated dietary or urinary sodium assessments, longer eGFR trajectories, and detailed treatment data are needed to determine whether this categorical association is reproducible and clinically meaningful [11,13].

4. CONCLUSION

In adults with diabetic kidney disease, sodium intake ≥2300 mg/day was associated with a lower adjusted six-month eGFR compared with intake <2300 mg/day. This association persisted in sensitivity analysis, although its magnitude was attenuated after exclusion of influential observations. In contrast, sodium analyzed continuously and intakes of protein, fat, and carbohydrate were not associated with six-month eGFR. These findings support further evaluation of sodium exposure as a potentially relevant dietary factor in DKD but do not establish a causal effect or a biological threshold. Dietary management should remain individualized, particularly in patients with advanced CKD. Larger prospective studies with repeated dietary assessment, objective sodium measurements, and longer eGFR follow-up are warranted.

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AUTHOR CONTRIBUTIONS

RRPAH: Conceptualization, Investigation, Data Curation, Formal Analysis, Visualization, and Writing – Original Draft.

HR: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

AYS: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

NAD: Methodology, Supervision, Validation, and Writing – Review & Editing.

A: Methodology, Supervision, Validation, and Writing – Review & Editing.

NR: Methodology, Supervision, Validation, and Writing – Review & Editing.

All authors critically reviewed the manuscript, approved the final version, and agreed to be accountable for the accuracy and integrity of the work.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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ETHICS STATEMENT

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Research Ethics Committee of Hasanuddin University, Makassar, Indonesia (Approval No. 547/UN4.6.4.5.31/PP36/2026; Protocol No. UH26030420; April 21, 2026). Written informed consent was obtained from all participants before enrollment. Participation was voluntary, and participant confidentiality and anonymity were maintained throughout the study.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional restrictions.

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