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Functional and Novel Foods

Association Between Dietary Acid Load and Kidney Function in Patients with Stage 4–5 Chronic Kidney Disease: A Cross-Sectional Study in Padang, Indonesia

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ABSTRACT

Background: Chronic Kidney Disease (CKD) is characterized by a progressive and irreversible decline in glomerular filtration rate (GFR), accompanied by disruptions in acid–base homeostasis, both of which may be exacerbated by an elevated dietary acid load (DAL). Persistent metabolic acidosis resulting from high DAL has been implicated in the acceleration of renal functional deterioration, muscle catabolism, and bone demineralization in patients with advanced CKD. Notwithstanding the growing body of evidence linking DAL to CKD progression in other settings, research systematically examining the influence of dietary acid burden in individuals with advanced-stage CKD remains limited in the Indonesian context.

Aims: This study aimed to investigate the association between DAL and indices of kidney function among patients with stage 4–5 CKD.

Patients and Methods: A cross-sectional analytical study was conducted at Dr. M. Djamil General Hospital, Padang, Indonesia, involving 80 patients diagnosed with stage 4–5 CKD. Dietary intake was assessed through two non-consecutive 24-hour dietary recall interviews, and DAL was estimated utilizing the validated Net Endogenous Acid Production (NEAP) formula. Bivariate analyses and multivariate binary logistic regression were employed to examine associations between DAL tertiles and renal function parameters, adjusting for potential confounders.

Results: Higher DAL was associated with lower GFR and elevated serum urea and creatinine concentrations. Following full multivariate adjustment, individuals in the highest DAL tertile demonstrated a significantly increased odds of reduced GFR relative to those in the lowest tertile (OR: 8.55; 95% CI: 1.63 – 44.79; $p = 0.017$). Statistically significant dose–response trends were additionally observed for elevated serum urea ($p = 0.023$) and serum creatinine ($p = 0.032$) across increasing DAL tertiles, indicating a consistent and progressive relationship between dietary acid burden and markers of renal functional impairment.

Conclusions: These findings indicate that an elevated dietary acid burden may constitute a significant and modifiable contributing factor to the decline in kidney function observed in patients with advanced CKD. Dietary modification aimed at reducing acid load—particularly through optimization of the balance between animal- and plant-derived protein sources and the enhancement of dietary potassium intake—may offer a clinically feasible and evidence-based nutritional strategy to attenuate metabolic acidosis and decelerate CKD progression. Prospective longitudinal studies with larger sample sizes are warranted to further elucidate the differential effects of animal- versus plant-based protein consumption on DAL and to establish causal directionality in this association.

Keywords: Chronic Kidney Disease; Dietary Acid Load; Net Endogenous Acid Production; Glomerular Filtration Rate; Kidney Function; Metabolic Acidosis.

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1 INTRODUCTION

Chronic Kidney Disease (CKD) is a condition defined by kidney damage or diminished kidney function, characterized by a glomerular filtration rate of below 60 mL/min/1.73 m² persisting for three months or more (Stevens *et al.*, 2024a). CKD currently constitutes a major global public health concern, with a significant increase in incidence, prevalence, and mortality (Huang *et al.*, 2025; Qin *et al.*, 2024). The burden of disease is further exacerbated by an increase in CKD risk factors, such as hypertension and type 2 diabetes mellitus (Foreman *et al.*, 2018). Findings from the Global Burden of Disease (GBD) study indicate that CKD accounted for 1.5 million deaths in 2021. In addition, the Disability-Adjusted Life Years (DALY) attributable to CKD have revealed an increasing trend, rising by 10.4%.

Consequently, CKD has risen in rank among the leading causes of death by age, from 18th in 1990 to 11th in 2021 (Guo *et al.*, 2025; Naghavi *et al.*, 2024). A recent study estimates that CKD affects approximately 9.5% of the global population, corresponding to roughly 850 million individuals worldwide (Bello *et al.*, 2024). According to the 2023 Indonesia Health Survey, the national prevalence of CKD is 0.18%, whereas in West Sumatra, the prevalence is slightly higher, at 0.23% (Kemenkes, 2023).

Kidney disease increases the risk of mortality, morbidity, disability, reduced quality of life, and cardiovascular events, and imposes substantial economic consequences at both the individual and health-system levels (Faucon, 2025; Francis *et al.*, 2024). The rising prevalence of CKD is accompanied by

a considerable economic burden, that increases substantially with disease severity, becoming particularly significant once patients reach stage G5 or end-stage renal disease (Jha *et al.*, 2023; Masajtis-Zagajewska *et al.*, 2024).

CKD is generally progressive and irreversible, affecting various metabolic pathways, including disturbances in acid-base balance that result in metabolic acidosis (Wieërs *et al.*, 2024). Metabolic acidosis occurs in approximately 20% of patients with CKD (Adamczak & Surma, 2021) and its incidence increases as kidney function declines, especially when the GFR falls below 30–40 mL/min/1.73 m² (Bushinsky *et al.*, 1999; Hamidianshirazi & Ekramzadeh, 2021). Metabolic acidosis can lead to dysfunction of various systems and organs, as well as to the progression of CKD (Adamczak & Surma, 2021). Furthermore, individuals with metabolic acidosis face an increased risk of muscle mass loss, heightened inflammatory responses, alterations in insulin, leptin, and growth hormone activity, and a greater likelihood of mortality (Scialla & Anderson, 2013; Vincent-Johnson *et al.*, 2023).

Dietary intake constitutes the principal source of the acid load that the kidneys must eliminate to maintain the acid-base homeostasis. Endogenous acid production originates from dietary amino acids and the total quantity of alkali absorbed in the gastrointestinal tract (Vincent-Johnson *et al.*, 2023). Accordingly, the control of dietary acid load may play a role in CKD prevention. High dietary acid consumption is directly associated with an increased risk of CKD and declining kidney function (Silva *et al.*, 2021). Toba *et al.* (2019) reported that Dietary Acid Load (DAL) is associated with CKD progression, with higher DAL scores constituting a risk factor for CKD development. Similarly, Rusdi *et al.* (2023) observed that DAL is associated with glomerular filtration rate, urea, and creatinine levels. These findings suggests that DAL plays a significant role in both the development and severity of the disease. Elevated DAL is also directly associated with an increased risk of kidney stone formation and reduced urine pH (Mofrad *et al.*, 2021; Rule *et al.*, 2009) and each 1 mmol/L reduction in serum bicarbonate has been associated with a 3% increase in the risk of developing renal stones (Tangri *et al.*, 2023).

Substantial observational evidence indicates that DAL is associated with albuminuria and the progression of kidney disease through the mechanisms involving ammonium toxicity and acid-base imbalance (Raphael, 2024; Wieërs *et al.*, 2024). However, research specifically targeting individuals with stage 4–5 CKD remains limited in Indonesia. Understanding the dietary contribution to acid production is therefore essential, as reducing DAL may help protect patients with CKD from chronic metabolic acidosis and its potential systemic and organ-level consequences, including accelerated disease progression. This study aims to

investigate the association between dietary acid load (DAL) and kidney function among patients with stage 4–5 chronic kidney disease at Dr. M. Djamil General Hospital, Padang, Indonesia.

2 SUBJECTS AND METHODS

2.1 Study Design and Population

This *cross-sectional* study was conducted between July and August 2025 at Dr. M. Djamil Hospital, Padang City, West Sumatra. The study sample comprised 80 patients diagnosed with chronic kidney disease, recruited employing a consecutive sampling technique whereby all eligible patients meeting the inclusion criteria were enrolled. The inclusion criteria were as follows: male and female patients diagnosed with CKD by a physician, willingness to participate as indicated by signing an informed consent form, and the ability of the patient and/or family to communicate effectively. The exclusion criteria comprised withdrawal from the study, death during the study period, and incomplete data on eGFR, urea, creatinine, albumin, or hemoglobin. The minimum required sample size was 71 participants, calculated applying the following formula (Hulley *et al.*, 2013):

$$N = \left(\frac{(Z\alpha + Z\beta)}{C} \right)^2 + 3$$

Where:

- n = minimum sample size
- Z α = confidence level = 1,96
- Z β = standard normal deviate = 0,8416
- r = 0,327 (Rusdi *et al.*, 2023)
- C = $0,5 \cdot \ln \left[\frac{(1+r)}{(1-r)} \right] = 0.3395$

2.2 Data Collection

Data collected in this study consisted of respondent characteristics, nutritional status, dietary intake, and kidney function. Respondent characteristics included age, sex, educational attainment, employment status, comorbidities, smoking status, and physical activity and were obtained through direct interviews with patients and/or their families. Age was categorized as either ≤ 60 years or > 60 years (WHO, 2025) and sex was categorized as male and female. Educational attainment was classified into two groups: a lower level comprising respondents with no formal schooling or those who had completed elementary or junior high school, and a higher level, comprising respondents who had completed senior high school or tertiary education. Employment status was categorized as employed or unemployed. Data on comorbidities included the presence or absence of hypertension and diabetes mellitus, and smoking status was categorized as smoker and non-smoker. Nutritional

status data was assessed through direct measurement of respondents' weight and height.

Dietary intake data were collected through direct interviews with patients and/or their families employing a 24-hour recall method administered on two occasions (2×24-hour recall). A food photograph book (*buku foto makanan*) published by the Indonesian Ministry of Health was used to assist in portion estimation during the interviews. The data collected comprised the type and frequency of foods and beverages consumed, together with estimated portion sizes expressed in household measures, which were subsequently converted into gram weights. These dietary intake data were subsequently utilized to calculate each respondent's Dietary Acid Load (DAL), which required protein and potassium intake as input variables. All reported food items were converted into gram weights and analyzed for protein and potassium content employing Nutrisurvey 2007; nutrient values for packaged foods and beverages were obtained directly from their nutrition labels. Protein and potassium intakes were expressed in grams and potassium intake was subsequently converted from grams to milliequivalents (mEq) using the conversion factor 1 mEq = 39.1 mg potassium.

DAL was estimated through the Net Endogenous Acid Production (NEAP) equation, with DAL categorized into tertile (Huang *et al.*, 2025; Koor *et al.*, 2024). The NEAP equation is expressed as follows:

$$NEAP(mEq/d) = 5 \left[\frac{Protein(gr/day)}{Kalium(mEq/day)} \right] - 10.2$$

Renal function data consisting of eGFR, urea, creatinine, albumin, and hemoglobin, were obtained during routine visits to the Internal Medicine Outpatient Polyclinic at the time of data collection. For inpatients, renal function data were obtained during hospitalization in the Internal Medicine Inpatient Ward. Renal function categories were defined to the following predetermined limits: GFR < 15 mL/min/1.73 m² (Stevens *et al.*, 2024b), urea > 50 mg/dL (Loscalzo *et al.*, 2022), and creatinine > 5.0 mg/dL (Loscalzo *et al.*, 2022).

2.3 Statistical Analysis

Data analysis comprised univariate, bivariate, and multivariate procedures. Univariate analysis was adopted to describe each dependent and independent variables, with results presented in tables and figures. Continuous variables were reported as means with standard deviations, while categorical variables were presented as frequencies and percentages (n, %). A *p*-value < 0.05 was considered statistically significant. Bivariate analysis involved chi-square cross-tabulation and Fisher's exact test to assess associations between dependent and independent variables, while independent *t*-tests and Mann–Whitney U tests were applied to compare nutritional status, physical and clinical indicators,

and dietary intake among participants. Multivariate regression analysis was conducted to determine the effect of dietary acid load (DAL) on kidney function among patients with chronic kidney disease.

2.4 Ethical Consideration

The study protocol was reviewed and received ethical approval from the Health Research Ethics Committee, RS M. Djamil Padang (No. DP.04.03/D.XVI.10.1/186/2025). Prior to data collection, comprehensive information regarding the study objectives and methodological procedures was provided to all invited participants, and written informed consent was obtained from each individual prior to enrollment.

3 RESULTS

3.1 Characteristics of The Study Participants

The study population consisted of 80 patients with chronic kidney disease (CKD) at Dr. M. Djamil General Hospital, Padang. Table 1 presents the principal characteristics of the participants, stratified according to renal function based on glomerular filtration rate (GFR), urea, and creatinine, with reference thresholds derived from the KDIGO guidelines (Stevens *et al.*, 2024b) and Harrison's Principles of Internal Medicine (Loscalzo *et al.*, 2022). Significant difference in age distribution were observed according to GFR (*p* = 0.013) and creatinine (*p* = 0.001), whereas no such difference was observed according to urea (*p* = 0.438). A significant difference in the prevalence of hypertension was likewise observed according to GFR (*p* = 0.036), with the majority of patients with a history of hypertension exhibiting a GFR > 15 mL/min/1.73 m². This finding is consistent with the established role of hypertension as a major risk factor in CKD progression. Patients with advanced renal failure, defined by GFR < 15 mL/min/1.73 m² and creatinine > 5 mg/dL indicators, were considerably more prevalent among those with a history of hypertension than among those without. No significant differences were observed in the distribution of sex, educational attainment, history of diabetes mellitus, or smoking status across the three renal function categories.

Nutritional status, as reflected by mean BMI, differed significantly across groups (*p* = 0.025). Patients with early-stage renal failure exhibited a higher mean BMI, approaching the overweight category, whereas those with advanced renal failure displayed a mean BMI within the normal range. This may be attributable to the weight loss commonly observed in patients with more severe, longer-standing renal impairment.

Specifically, the group with GFR ≥ 15 mL/min/1.73 m² had a higher mean BMI (25.20 ± 4.98 kg/m²) than the group with GFR < 15 mL/min/1.73 m² (22.20 ± 3.94 kg/m²).

Table 1. Profile of CKD Subjects Characterized by Demography Factors, Morbidity & Vital Signs

Variables	Kidney function, n (%)		P-value	Kidney function, n (%)		P-value	Kidney function, n (%)		P-Value
	GFR>15	GFR<15		Urea<50	Urea>50		Creatinine <5	Creatinine >5	
Age group									
< 60 years	49 (81.7)	11 (18.3)	0.013**	30 (50.0)	30 (50.0)	0.438 ^a	17 (28.3)	43 (71.7)	0.001***
> 60 years	10 (50)	10 (50)		12 (60.0)	8 (40.0)		14 (70.0)	6 (30.0)	
Sex									
Male	30 (66.7)	15 (33.3)	0.169 ^a	20 (44.4)	25 (55.6)	0.102 ^a	17 (37.8)	28 (62.2)	0.840 ^a
Female	29 (82.9)	6 (17.1)		22 (62.9)	13 (37.1)		14 (40.0)	21 (60.0)	
Educational level									
High	36 (67.9)	17 (32.1)		31 (58.8)	22 (41.5)	0.133 ^a	21 (39.6)	32 (60.4)	0.822 ^a
Low	23 (85.2)	4 (14.8)	0.164 ^a	11 (40.7)	16 (59.3)		10 (37.0)	17 (63.0)	
HBP history									
No	10 (52.6)	9 (47.4)		10 (52.6)	9 (47.4)	0.990 ^a	11 (57.9)	8 (42.1)	0.05**
Yes	49 (80.3)	12 (19.7)	0.036**	32 (52.5)	29 (47.5)		20 (32.8)	41 (67.2)	
DM history									
No	37 (78.7)	10 (21.3)		21 (44.7)	26 (55.3)	0.095 ^a	16 (34.0)	31 (66.0)	
Yes	22 (66.7)	11 (33.3)	0.343 ^a	21 (63.6)	12 (36.4)		15 (45.5)	18 (54.5)	0.302 ^a
Smoking									
No	53 (75.7)	17 (24.3)		39 (55.7)	31 (44.3)	0.180 ^b	28 (40.0)	42 (60.0)	0.733 ^a
Yes	6 (60)	4 (40)	0.442 ^b	3 (30.0)	7 (70.0)		3 (30.0)	7 (70.0)	
Nutritional Status, mean (SD)	25.20 (4.98)	22.20 (3.94)	0.025**	23.21 (4.24)	22.75 (4.64)	0.476 ^c	23.78 (4.93)	22.48 (4.03)	0.307 ^c
Systolic blood pressure, mean (mmHg)	128.43 (20.16)	142.69 (21.25)	0.012**	139.33 (22.38)	138.53 (21.39)	0.664 ^c	133.23 (20.54)	142.57 (21.96)	0.087 ^c
Diastolic blood pressure, mean (mmHg)	76.14 (13.40)	81.83 (12.84)	0.089 ^d	79.00 (13.46)	81.82 (12.80)	0.342 ^d	76.16 (13.87)	82.98 (12.07)	0.023**
Body temperature (°C)	36.54 (0.51)	36.50 (0.37)	0.830 ^e	36.43 (0.25)	36.61 (0.52)	0.075 ^e	36.50 (0.44)	36.52 (0.38)	0.537 ^e
Heart rate (bpm)	84.57 (13.45)	85.61 (14.32)	0.773 ^d	82.50 (13.17)	88.47 (14.42)	0.057 ^d	82.74 (15.39)	86.98 (12.97)	0.190 ^d
Respiratory rate (%)	19.29 (2.00)	20.42 (3.23)	0.079 ^e	19.36 (2.30)	20.97 (3.44)	0.003**	19.48 (2.20)	20.53 (3.35)	0.128 ^e

Note: *Indicates a significant result (p -Value<0.05); **Indicates a significant result (p -value<0.001); a chi-square; b fisher's exact test; c Mann Whitney test; d independent t -test; Continuous values indicate as Mean $\pm$ SD, categorical values as n (%).

Clinical and physical parameters obtained from medical record generally did not reveal significant differences between groups stratified by GFR, urea, or creatinine. However, mean systolic blood pressure was significantly lower in the GFR group > 15 (128.43 ± 20.16 mmHg) than in the GFR < 15 group (142.69 ± 21.25 mmHg; $p = 0.012$). Diastolic blood pressure and pulse rate also exhibit significant differences according to creatinine-based categories ($p = 0.023$ and $p = 0.035$, respectively). These findings suggest specific physiological alterations among individuals with CKD, arising from reduced GFR and the accumulation of urea and creatinine.

3.2 Dietary Acid Load

Mean dietary intake values across groups are presented in Table 2. Participants with elevated urea levels exhibited significantly lower DAL values than those with normal urea levels (69.34 ± 44.97 mEq/day vs. 93.15 ± 43.34 mEq/day, $p < 0.05$). Similarly, participants with elevated creatinine levels exhibited lower DAL values than those with normal creatinine levels (72.42 ± 42.40 mEq/day vs. 96.72 ± 46.78 mEq/day, $p < 0.05$). No significant differences in protein or potassium intake were observed across the corresponding categories.

Protein intake did not differ significantly between renal function groups for any of the three indicators. Mean protein intake was comparable between the GFR > 15 and GFR < 15 groups (40.01 ± 25.40 g/day vs. 33.85 ± 22.89 g/day). Similarly, no significant differences in protein intake were observed between groups defined by urea or creatinine levels ($p > 0.05$). Potassium intake likewise did not differ significantly between groups with early- and advanced-stage renal impairment across all three renal function indicators ($p > 0.05$).

Conversely, DAL demonstrated a more consistent association with renal function parameters. Although the difference in DAL between the GFR > 15 and GFR < 15 groups did not reach statistical significance ($p = 0.070$), a trend toward higher DAL values was observed among participants with relatively preserved renal function. Significant differences in DAL were observed between early-

and advanced-stage renal failure when stratified by urea ($p = 0.006$) and creatinine ($p = 0.022$), with lower mean DAL values evident in more severe cases. Collectively, these findings indicate that DAL is significantly associated with biochemical indicators of kidney function, namely urea and creatinine, whereas protein and potassium intake considered individually revealed no such association. This suggests that dietary acid load, as a comprehensive dietary construct, may more accurately reflect renal function than single-nutrient intake alone.

3.3 Effect of Dietary Acid Load on Kidney Function

The odds ratios (ORs) with 95% confidence intervals (CIs) for reduced kidney function across tertiles of DAL are summarized in Table 3. For the GFR parameter, an increased risk of impaired kidney function was identified following adjustment for several risk factors. In Model 1, the highest DAL tertile was associated with a 3.15-fold higher risk (95% CI: 0.88–11.22) compared with the lowest tertile. In Model 2, following additional adjustment for educational level and smoking status, the risk of reduced GFR in the highest DAL tertile was 2.79 (95% CI: 0.77–10.15). In Model 3, following further adjustment for history of hypertension and diabetes mellitus, the risk of reduced GFR reached statistical significance, increasing to 8.55 (95% CI: 1.63–44.79) in the highest tertile of DAL. A significant trend was additionally observed, indicating that higher dietary acid load was associated with an increased risk of reduced GFR (p -value = 0.017).

A significant increase in risk was likewise observed in the urea parameter. Subjects in the second DAL tertile exhibited a higher risk of elevated urea than those in the first tertile across all models, with ORs of 5.80 (95% CI: 1.36–24.72), 5.44 (95% CI: 1.24–23.76), and 5.52 (95% CI: 1.17–26.14) in Models 1, 2, and 3, respectively. Although the risk associated with the third tertile did not reach statistical significance for urea, the trend test remained significant across all three models ($p = 0.011$, $p = 0.010$, and $p = 0.023$, respectively).

Table 2. Dietary Intake (Mean $\pm$ SD) of Non ESRD Subjects and ESRD Subjects

Variables	Kidney function, n (%)			Kidney function, n (%)			Kidney function, n (%)		
	GFR >15	GFR <15	<i>p</i> -value	Urea < 50	Urea > 50	<i>p</i> -value	Creatinine < 5	Creatinine > 5	<i>p</i> -value
Protein (gr)	40.01 (25.40)	33.85 (22.89)	0.312	35.51 (17.25)	35.40 (29.27)	0.482	37.11 (23.26)	34.41 (23.94)	0.505
Potassium (mEq)	27.69 (30.32)	24.92 (23.37)	0.827	21.13 (14.16)	30.64 (32.90)	0.421	24.24 (25.58)	26.54 (25.13)	0.407
Dietary Acid Load (DAL)	100.21 (53.25)	75.31 (40.86)	0.070	93.15 (43.34)	69.34 (44.97)	0.006*	96.72 (46.78)	72.42 (42.40)	0.022*

Table 3. Logistic Regression Models' Odds Ratios of Chronic Kidney Disease (CKD) Across Tertile Categories of DAL

Model	Dietary Acid Load (DAL)			<i>p</i> -value
	Tertile 1	Tertile 2	Tertile 3	
GFR				
Model 1 ^a	1.00	4.77 (1.01 - 22.42)	3.15 (0.88 - 11.22)	0.091
Model 2 ^b	1.00	4.97 (0.99 - 24.84)	2.79 (0.77 - 10.15)	0.111
Model 3 ^c	1.00	10.33 (1.58 - 67.26)	8.55 (1.63 - 44.79)	0.017
Urea				
Model 1 ^a	1.00	5.80 (1.36 - 24.72)	0.78 (0.25 - 2.43)	0.011
Model 2 ^b	1.00	5.44 (1.24 - 23.76)	0.64 (0.19 - 2.11)	0.010
Model 3 ^c	1.00	5.52 (1.17 - 26.14)	0.79 (0.22 -2.84)	0.023
Creatinine				
Model 1 ^a	1.00	7.06 (1.41 - 35.39)	1.65 (0.50 - 5.37)	0.056
Model 2 ^b	1.00	6.73 (1.33 -34.06)	1.62 (0.49 -5.35)	0.067
Model 3 ^c	1.00	11.48 (1.84 - 71.49)	2.63 (0.70 - 9.78)	0.032

Note: ^a Adjusted for age, sex; ^b Additionally adjustment for educational level, smoking; ^c Additionally adjustment for HBP history and DM history.

For the creatinine parameter, an increased risk was observed in both the second and third DAL tertiles, with the highest risk consistently observed in the second tertile across all models. In Model 1, the OR for the second tertile was 7.06 (95% CI: 1.41–35.39), remaining elevated in Model 2 at 6.73 (95% CI: 1.33–34.66), and increasing further in Model 3 to 11.48 (95% CI: 1.84–71.49). In the third tertile, the direction of association likewise suggested an increased risk, although the estimates did not reach statistical significance. A significant trend was observed, indicating that higher DAL tertiles were associated with an increased risk of elevated creatinine levels (*p* for trend = 0.032).

4 DISCUSSION

This study demonstrated a decline in kidney function among individuals with a higher dietary acid load. This positive association remained significant after adjustments for age, sex, educational level, smoking status, history of hypertension and history of diabetes mellitus.

These findings are consistent with those of [Banerjee *et al.* \(2015\)](#), who reported that DAL in the upper range is associated with an increased risk of end stage renal disease (ESRD). Increased dietary acid excretion may contribute to kidney injury. The National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (KDOQI) clinical practice guidelines for nutrition in CKD currently recommend reducing DAL in adults with CKD through increased consumption of fruits and vegetables, with the aim of slowing further loss of kidney function ([Ikizler *et al.*, 2020](#)). Dietary intake can influence the body's acid-base balance through the supply of acid or alkaline precursors ([Banerjee *et al.*, 2015](#)). [Kanda *et al.* \(2014\)](#) similarly reported that higher NEAP was associated with an increased risk of CKD progression among older adults with chronic kidney disease. Dietary acid load may contribute to the decline in GFR by

reducing serum bicarbonate concentrations; elevated dietary protein intake enhances renal endothelin production, and distal nephron acidification is mediated, in part, through increased endothelin activity ([Kanda *et al.*, 2014](#)).

Animal protein constitutes an acidic precursor food that is metabolized into acid waste products, and, owing to its high phosphorus content, further contributes to the acidity of body fluids ([Remer & Manz, 1994, 1995](#); [Silva *et al.*, 2021](#)). A high DAL may therefore act as a risk factor for CKD through intrarenal mechanisms that promote renal injury and a progressive GFR decline. Diets with a higher acid load stimulate increased ammonium production by tubular cells as a compensatory response to the excess tubular hydrogen ions, a process that contributes to tubular hypertrophy and glomerular hyperfiltration. The resulting rise in intratubular hydrogen ion concentration, accompanied by increased production of endothelin-1, angiotensin II, and aldosterone, may ultimately reduce GFR when acid-producing foods are consumed in excess and over prolonged periods ([Osuna-Padilla *et al.*, 2019](#); [Rodrigues *et al.*, 2018](#)).

The present study demonstrated a significant association between DAL and kidney function among patients with stage 4–5 CKD, suggesting that a higher dietary acid burden is associated with poorer renal function in this population. These findings are consistent with previous observational studies reporting that higher dietary acid load is associated with reduced kidney function and accelerated CKD progression ([Silva *et al.*, 2021](#)). The observed association is biologically plausible and may be explained by several pathophysiological mechanisms. As the kidneys are principally responsible for maintaining acid-base homeostasis, declining kidney function increases the acidification burden placed on the residual nephrons ([Raphael, 2019](#); [Silva *et al.*, 2021](#)). Individuals with normal kidney function or preserved GFR are able to excrete acid in

amounts commensurate with dietary intake, whereas those with impaired kidney function or reduced GFR have limited excretory capacity owing to an already diminished filtration rate. In early-stage CKD, the kidneys initially adapt to increased acid retention; however, this compensatory response eliminates only a fraction of the dietary acid load, and this insufficient adaptation leads over time to disturbances in acid–base balance (Wesson *et al.*, 2011). Increased acidification demands may also elevate ammonia production and injure residual nephrons. In animal models of CKD, acid–base imbalance has been demonstrated to stimulate production of angiotensin II, aldosterone, and endothelin-1 and increased ammonia formation, potentially promoting inflammation and fibrosis (Joshi *et al.*, 2021). Furthermore, a diet high in DAL may reduce blood pH toward the lower limit of the normal range, which, in the absence of adequate homeostatic compensation or dietary modification, can result in chronic metabolic acidosis (Rusdi *et al.*, 2023).

Collectively, these findings demonstrate an association between higher dietary acid load and poorer kidney function among patients with CKD, suggesting that dietary acid load may represent an essential target for nutritional management. Prospective and interventional studies are nevertheless required to determine whether dietary modification can reduce metabolic acidosis and improve kidney outcomes. Dietary modification represents a promising strategy to lowering non-volatile acid load and protecting individuals with CKD from the adverse effects of metabolic acidosis (Vincent-Johnson *et al.*, 2023). Public health strategies aimed at improving dietary quality at the population may improve CKD outcomes, including among the large populations of individuals with early- to moderate-stage CKD who remain undiagnosed or unaware of their condition. The benefits and potential risks of reducing dietary acid load as a secondary prevention strategy in early- to moderate-stage CKD warrant rigorous evaluation in future studies.

5 CONCLUSIONS

The present study assessed dietary acid load among patients with chronic kidney disease based on reported food intake. The findings indicate that higher DAL may be associated with greater impairment of renal function, as reflected by GFR, urea, and creatinine parameters. These results suggest that modifying dietary acid intake could help improve kidney function and mitigate metabolic acidosis, thereby potentially preventing subsequent acidosis-related adverse outcomes in CKD. Further research is required to clarify the differential effects of animal- versus plant-based protein on dietary acid load. Clinical and public health strategies emphasizing improved dietary quality, at both the population and individual levels, may support better kidney

function and reduce the risk of metabolic acidosis–related complications.

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