JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

DOI: https://doi.org/10.61919/e6wqfj52

Review Article

Effect of Dietary Sodium Restriction (<2 g/day) on Blood Pressure Variability and Proteinuria Levels in Patients with Stage 2–3 chronic kidney disease: A Structured Narrative Review

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"Cite this Article" Received: 13 December 2025; **Accepted**: 04 March 2026; **Published**: 30 March 2026

Author Contributions: Concept: SAE; Design: SAE, MKH; Data Collection: MR, RKM, AM; Analysis: SAE, MKH, MR; Drafting: SAE, MKH, MR, RKM, AM **Ethical Approval**: Ethical approval was not required for this study because it is a systematic review based exclusively on previously published studies and publicly available data. No human participants, patient records, or identifiable personal information were directly involved in this research **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest. **Funding**: No external funding; **Data Availability**: All data analyzed during this study are included within the published article and its referenced sources. Additional information related to study selection and extracted data may be made available by the corresponding author upon reasonable request **Acknowledgments**: NA

ABSTRACT

Background: Excess dietary sodium contributes to extracellular volume expansion, hypertension, proteinuria, and reduced responsiveness to renin–angiotensin–aldosterone system inhibition in chronic kidney disease. However, its specific relationship with blood pressure variability remains uncertain. Objective: To critically synthesize evidence concerning dietary sodium restriction below 2 g/day and its effects on blood pressure variability, mean blood pressure, proteinuria, albuminuria, and related renal outcomes in adults with stage 2–3 chronic kidney disease. Methods: A structured narrative review was undertaken using literature identified through PubMed/MEDLINE, Scopus, Web of Science, the Cochrane Library, Embase, and Google Scholar. Controlled dietary interventions, longitudinal cohorts, observational studies, guidelines, and clinically relevant mechanistic literature were evaluated. Intervention evidence was interpreted separately from observational sodium-exposure studies, and mean blood pressure was distinguished from formally measured blood pressure variability. Results: Controlled trials generally demonstrated that sodium restriction reduced office and ambulatory blood pressure, extracellular fluid volume, proteinuria, and albuminuria. One crossover trial reported approximate reductions of 10 mmHg in systolic and 4 mmHg in diastolic blood pressure, while behavioural sodium-reduction support produced short-term reductions in ambulatory and office blood pressure and urinary protein excretion. Sodium restriction also enhanced the antiproteinuric effects of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers. Higher urinary sodium excretion was frequently associated with CKD progression and cardiovascular outcomes in observational cohorts, particularly among patients with greater proteinuria. Direct evidence regarding standardized blood pressure variability indices was limited. Conclusion: Sodium restriction is supported as an adjunctive strategy for improving mean blood pressure control and reducing proteinuria in non-dialysis CKD, but its independent effects on blood pressure variability and long-term clinical outcomes require further investigation. Keywords: Chronic kidney disease; dietary sodium restriction; salt restriction; blood pressure variability; hypertension; proteinuria; albuminuria; renin–angiotensin–aldosterone system.

INTRODUCTION

Chronic kidney disease (CKD) is a major global health concern associated with progressive deterioration of kidney function, cardiovascular morbidity, premature mortality, and increasing healthcare

expenditure. CKD stages 2 and 3 represent a clinically important period during which renal impairment is established but potentially modifiable factors can still be addressed to slow further disease progression. Among these factors, hypertension and proteinuria are particularly important because both are independently associated with declining glomerular filtration rate, cardiovascular events, and progression to kidney failure (1,2).

Dietary sodium intake has a central role in blood pressure regulation, extracellular fluid balance, vascular function, and renal haemodynamics. Excess sodium consumption promotes extracellular volume expansion, arterial stiffness, endothelial dysfunction, and increased systemic and intraglomerular pressure. These effects may be more pronounced in CKD because impaired renal sodium excretion increases susceptibility to volume overload and salt-sensitive hypertension (3,4). Persistent intraglomerular hypertension can disrupt the glomerular filtration barrier and increase urinary protein loss, while prolonged exposure of renal tubules to filtered proteins may contribute to inflammation, interstitial fibrosis, and progressive nephron injury (5,6).

Proteinuria is therefore both a marker of glomerular damage and a potential mediator of CKD progression. Reductions in urinary protein or albumin excretion are generally associated with improved renal prognosis, particularly when accompanied by effective blood pressure control. Renin–angiotensin–aldosterone system inhibitors remain central to the treatment of hypertension and proteinuria in CKD; however, their therapeutic effects may be influenced by dietary sodium intake. High sodium consumption may attenuate the antihypertensive and antiproteinuric effects of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers, whereas sodium restriction may enhance their renoprotective response.

In addition to mean blood pressure, variability in blood pressure over time has emerged as a potentially important cardiovascular and renal risk indicator. Blood pressure variability refers to within-person fluctuations measured over different time intervals, including beat-to-beat, ambulatory, day-to-day, and visit-to-visit variation. Depending on the study design, it may be quantified using the standard deviation, coefficient of variation, average real variability, variability independent of the mean, or related indices. Increased blood pressure variability has been associated with vascular injury, target-organ damage, cardiovascular events, and deterioration of renal function independently of average blood pressure in several clinical populations (7,8).

Mean blood pressure and blood pressure variability are nevertheless distinct outcomes. A dietary intervention may reduce average systolic or diastolic blood pressure without necessarily reducing the magnitude of fluctuations between measurements. Conversely, improved consistency of blood pressure readings should not be inferred solely from a reduction in the mean value. This distinction is particularly important in CKD research because studies frequently use terms such as blood pressure control, ambulatory blood pressure, blood pressure stability, and blood pressure variability interchangeably despite measuring different physiological and statistical constructs.

Clinical guidelines recommend limiting sodium intake in adults with CKD, commonly to less than 2 g of sodium per day, equivalent to approximately 5 g of salt (9). Sodium restriction is intended to improve blood pressure control, reduce extracellular fluid expansion, and complement pharmacological renoprotection. Sustained adherence may, however, be difficult because of processed-food consumption, hidden sodium in packaged foods, cultural dietary practices, limited access to dietetic counselling, and the practical difficulty of estimating daily sodium intake.

Evidence from hypertensive populations has consistently shown that lowering dietary sodium can reduce systolic and diastolic blood pressure, particularly among individuals with hypertension or salt sensitivity (11,12). Research conducted in CKD populations has similarly suggested that sodium restriction may improve blood pressure control and reduce urinary protein excretion. Clinical studies have reported that lower sodium intake may enhance the antiproteinuric effect of angiotensin-

converting enzyme inhibition and improve the renal haemodynamic response to renin–angiotensin–aldosterone system blockade (18,19). Randomized dietary interventions have also demonstrated reductions in ambulatory blood pressure and extracellular fluid volume among patients with moderate CKD (20).

Observational evidence has provided additional information regarding the relationship between sodium exposure and renal outcomes. Studies using dietary assessments or urinary sodium excretion have reported associations between higher sodium intake, poorer blood pressure control, increased cardiovascular risk, and faster deterioration of kidney function (16,17,21). These findings support the biological and clinical rationale for sodium reduction but require cautious interpretation because observational associations may be influenced by confounding, reverse causation, medication use, disease severity, nutritional status, and measurement error. Such studies should therefore be distinguished from controlled interventions evaluating the effects of prescribed sodium restriction.

The interaction between dietary sodium and renin–angiotensin–aldosterone system inhibition is especially relevant in stage 2–3 CKD. Studies have indicated that sodium restriction may potentiate the albuminuria-lowering effects of losartan and other renoprotective therapies, whereas excessive sodium intake may reduce the response to pharmacological treatment (18,19,22). Dietary modification may consequently function as an important adjunct to medication rather than as an isolated intervention.

Despite the apparent benefits of sodium reduction, the available literature remains heterogeneous. Studies differ in CKD stage, underlying renal diagnosis, baseline blood pressure, concurrent medication, prescribed sodium target, achieved sodium intake, method of dietary assessment, intervention duration, and outcome definition. Proteinuria may be assessed using 24-hour urinary protein excretion, urine protein-to-creatinine ratio, albumin excretion, or urine albumin-to-creatinine ratio. Similarly, some studies report office or ambulatory mean blood pressure, while relatively few directly evaluate formally defined measures of blood pressure variability.

Previous reviews and meta-analyses have generally supported dietary salt reduction for lowering blood pressure and proteinuria in CKD, but the specific relationship between sodium restriction and blood pressure variability remains less clearly established (15). The available evidence is further complicated by the tendency to interpret reductions in average blood pressure as evidence of improved variability. A clinically useful review must therefore distinguish direct measurements of blood pressure variability from broader indicators of blood pressure control and separately interpret intervention studies and observational evidence.

This structured narrative review examines the relationship between dietary sodium restriction below 2 g/day and blood pressure variability, mean blood pressure, proteinuria, and albuminuria in adults with stage 2–3 CKD. It also considers the interaction between sodium intake and renin–angiotensin–aldosterone system inhibitor therapy, potential effects on renal-function decline, biological mechanisms, clinical implications, and limitations of the available evidence. Particular emphasis is placed on distinguishing true blood pressure variability from changes in average blood pressure and on interpreting controlled dietary interventions separately from observational sodium-exposure studies.

MATERIAL AND METHODS

A structured narrative-review approach was used to examine clinical evidence concerning dietary sodium restriction, blood pressure variability, proteinuria, and related renal outcomes in adults with stage 2–3 CKD. This approach was selected because the literature included heterogeneous randomized trials, crossover studies, non-randomized interventions, prospective cohorts, observational analyses, guideline documents, and mechanistic studies that differed substantially in design, population, sodium assessment, follow-up duration, and outcome measurement. The purpose of the review was to provide a

critical and clinically focused synthesis rather than an exhaustive systematic review or pooled meta-analysis.

Relevant literature was identified through structured searches of PubMed/MEDLINE, Scopus, Web of Science, the Cochrane Library, Embase, and Google Scholar. The search emphasized publications from January 2015 to March 2025 to capture contemporary clinical evidence. Earlier landmark trials, mechanistic studies, major reviews, and clinical guidelines were also retained when they were necessary to explain the development of sodium-restriction recommendations, the interaction between sodium intake and renin–angiotensin–aldosterone system blockade, or the biological relationship between sodium, blood pressure, and proteinuria.

Search terms were organized around four principal concepts: chronic kidney disease, sodium intake, blood pressure, and urinary protein excretion. Population-related terms included “chronic kidney disease,” “CKD stage 2,” “CKD stage 3,” “moderate chronic kidney disease,” and “renal impairment.” Exposure and intervention terms included “dietary sodium restriction,” “low sodium diet,” “salt restriction,” “sodium reduction,” “dietary sodium intake,” and “urinary sodium excretion.” Blood pressure terms included “hypertension,” “ambulatory blood pressure,” “blood pressure variability,” “visit-to-visit variability,” and “blood pressure control.” Renal outcome terms included “proteinuria,” “albuminuria,” “urinary protein excretion,” “estimated glomerular filtration rate,” “renal outcomes,” and “CKD progression.” Boolean operators were used to combine related terms, and reference lists of relevant studies and reviews were examined to identify additional publications.

Studies and other sources were selected on the basis of their clinical relevance, methodological contribution, and direct relationship to the review objectives. Particular attention was given to investigations involving adults with stage 2 or stage 3 CKD, although mixed-stage CKD studies were considered when they provided relevant information applicable to early-to-moderate disease. Evidence derived exclusively from dialysis-dependent populations, kidney transplant recipients, paediatric populations, or patients with kidney failure was not used as the principal basis for conclusions concerning stage 2–3 CKD.

Priority was given to studies evaluating prescribed dietary sodium reduction, low-sodium dietary counselling, achieved sodium intake, or sodium exposure measured through urinary excretion. Controlled dietary interventions were considered the most direct evidence of treatment effect. Observational studies examining urinary sodium excretion or reported sodium intake were used to evaluate exposure–outcome relationships and longer-term clinical associations. These two evidence types were interpreted separately because an association between higher sodium exposure and adverse outcomes does not by itself establish that a sodium-reduction intervention will produce the corresponding clinical benefit.

The principal outcomes of interest were blood pressure variability and proteinuria or albuminuria. Blood pressure variability was interpreted only when a study reported a defined measure of within-person fluctuation, such as the standard deviation, coefficient of variation, average real variability, variability independent of the mean, or another stated ambulatory, day-to-day, or visit-to-visit variability index. Reductions in office blood pressure, mean ambulatory blood pressure, nighttime blood pressure, or systolic blood pressure were considered indicators of blood pressure lowering rather than direct evidence of reduced blood pressure variability unless an explicit variability measure was reported.

Proteinuria-related outcomes included 24-hour urinary protein excretion, urine protein-to-creatinine ratio, urinary albumin excretion, and urine albumin-to-creatinine ratio. Because these measures are not directly interchangeable, findings were interpreted according to the measurement reported in each study rather than combined into a single quantitative estimate. Secondary outcomes included mean systolic and diastolic blood pressure, ambulatory blood pressure, extracellular fluid volume, estimated

glomerular filtration rate, CKD progression, cardiovascular risk indicators, and modification of treatment response to angiotensin-converting enzyme inhibitors or angiotensin receptor blockers.

The evidence was organized into clinically relevant themes. These included the physiological effects of sodium in CKD, effects of sodium reduction on mean blood pressure, evidence concerning blood pressure variability, effects on proteinuria and albuminuria, interactions with renin–angiotensin–aldosterone system inhibition, associations with renal-function decline, barriers to dietary adherence, and implications for clinical practice. Randomized and non-randomized interventions were considered separately from observational studies, and findings were interpreted in relation to study design, population, sodium-assessment method, duration of follow-up, and outcome definition.

Formal meta-analysis was not performed because the reviewed studies varied substantially in their sodium targets, achieved intake, comparator conditions, clinical populations, outcome measures, follow-up periods, and statistical reporting. Numerical findings from individual studies were considered where they were clearly reported, but results were not averaged across heterogeneous studies and no unreported effect estimates, confidence intervals, p-values, or participant-level data were reconstructed. Apparent differences between studies were interpreted qualitatively rather than treated as statistically established subgroup effects.

The methodological strengths and limitations of the literature were evaluated narratively. Greater interpretive weight was given to controlled interventions with clearly defined sodium targets, objective assessment of sodium intake, appropriate comparator conditions, and complete reporting of blood pressure or proteinuria outcomes. Observational findings were interpreted more cautiously because of possible residual confounding, reverse causation, incomplete dietary assessment, variation in urinary sodium collection, medication effects, and differences in baseline kidney function. The review also considered whether conclusions were based on direct measurements of the outcome of interest or inferred from related but distinct clinical indicators.

As this review used information from published studies and publicly available sources, it did not involve recruitment of participants, access to identifiable patient information, or collection of new clinical data. Ethical approval and individual informed consent were therefore not required.

RESULTS

Overview of the Evidence

The literature identified for this structured narrative review comprised randomized and crossover dietary interventions, behavioural sodium-reduction trials, prospective cohort studies, and observational analyses based predominantly on urinary sodium excretion. The studies differed considerably in CKD stage, underlying renal diagnosis, baseline proteinuria, antihypertensive treatment, sodium target, method of estimating sodium intake, follow-up duration, and outcome measurement. Controlled interventions generally evaluated short-term changes in mean blood pressure, ambulatory blood pressure, extracellular fluid volume, proteinuria, or albuminuria. Observational studies more commonly examined associations between urinary sodium excretion and CKD progression, cardiovascular outcomes, or mortality.

The evidence was therefore synthesized according to five principal themes: effects on mean blood pressure, evidence concerning blood pressure variability, effects on proteinuria and albuminuria, interaction with renin–angiotensin–aldosterone system inhibition, and associations with kidney-function decline and cardiovascular outcomes. Intervention findings were interpreted separately from observational associations because the latter cannot independently establish the clinical effect of prescribing a low-sodium diet.

Effects of Sodium Restriction on Mean Blood Pressure

Controlled intervention studies generally demonstrated that reducing sodium intake lowered mean systolic and diastolic blood pressure in patients with CKD. In a randomized crossover trial involving 20 adults with hypertensive stage 3–4 CKD, McMahon et al. compared low- and high-sodium dietary periods and reported mean reductions of approximately 10 mmHg in systolic blood pressure and 4 mmHg in diastolic blood pressure during sodium restriction. Sodium reduction was also associated with decreased extracellular fluid volume, albuminuria, and proteinuria, suggesting that the antihypertensive effect was accompanied by favourable changes in volume status and renal haemodynamics (20).

Meuleman et al. evaluated a behavioural self-management intervention incorporating education, motivational interviewing, coaching, and self-monitoring among 138 patients with moderately impaired kidney function. At three months, the intervention reduced urinary sodium excretion by a mean of 30.3 mmol/24 hours compared with regular care. It also reduced daytime ambulatory diastolic blood pressure by 3.4 mmHg, office diastolic blood pressure by 5.2 mmHg, and urinary protein excretion by 0.4 g/24 hours. At six months, the differences in sodium excretion and ambulatory blood pressure were no longer statistically evident, although reductions in office systolic and diastolic blood pressure and protein excretion remained detectable. These findings indicate that behavioural support can produce clinically relevant changes but that maintaining sodium restriction over time remains difficult (25).

A prospective randomized trial by Trakarnvanich et al. assigned 194 patients with CKD stages 1–3 to a low-salt intervention or usual dietary intake for three months. The low-salt group demonstrated greater reductions in systolic and diastolic blood pressure and a smaller short-term decline in estimated glomerular filtration rate than the control group. However, the achieved separation in urinary sodium excretion was modest and was not consistently significant between groups, requiring cautious interpretation of the renal-function findings (36).

Prospective observational evidence also supports an association between reduced sodium exposure and better blood pressure control. Nerbass et al. studied 1,607 adults with an estimated glomerular filtration rate of 30–59 mL/min/1.73 m² in primary care. Participants whose estimated sodium intake decreased from more than 100 mmol/day to 100 mmol/day or less over one year experienced a larger reduction in mean arterial pressure than those whose sodium intake increased above this threshold. Improvements in albuminuria were mainly observed among participants who had albuminuria at baseline (24).

Table 1. Principal Intervention and Longitudinal Evidence on Sodium Reduction in CKD

Study	Design and population	Sodium intervention or comparison	Principal findings	Interpretation
Slagman et al. (18)	Randomized crossover trial; 52 patients with non-diabetic proteinuric nephropathy receiving background ACE inhibition	Low-sodium target of 50 mmol/day compared with regular-sodium target of 200 mmol/day, with or without additional angiotensin receptor blockade	Achieved urinary sodium excretion was approximately 106 versus 184 mmol/day. Residual proteinuria decreased from 1.68 g/day during regular sodium intake and ACE inhibition to 0.85 g/day during low sodium intake; combined low sodium intake and angiotensin receptor blockade reduced it further to 0.67 g/day	Sodium restriction markedly enhanced antiproteinuric therapy and produced a larger reduction than adding angiotensin receptor blockade during regular sodium intake
McMahon et al. (20)	Randomized crossover trial; 20 adults with hypertensive stage 3–4 CKD	Low-sodium versus high-sodium dietary periods	Mean systolic and diastolic blood pressure decreased by approximately 10 and 4 mmHg, respectively; extracellular fluid volume, albuminuria, and proteinuria also decreased	Strong short-term physiological evidence, although the sample was small and included stage 4 CKD
Meuleman et al. (25)	Open randomized controlled trial; 138 patients with moderately reduced kidney function	Regular care versus behavioural sodium-restriction support	At three months, sodium excretion decreased by 30.3 mmol/24 hours, daytime ambulatory diastolic pressure by 3.4 mmHg, office diastolic pressure by 5.2 mmHg, and protein excretion by 0.4 g/24 hours. Some effects attenuated at six months	Demonstrates modest short-term effectiveness and the difficulty of sustaining dietary adherence
Nerbass et al. (24)	Prospective primary-care cohort; 1,607 adults with eGFR 30–59 mL/min/1.73 m²	Change in estimated sodium intake over one year	Reduction from more than 100 to 100 mmol/day or less was associated with greater improvement in mean arterial pressure; albuminuria improved primarily in participants with baseline albuminuria	Supports real-world benefit but remains observational and susceptible to confounding
Trakarnvanich et al. (36)	Prospective open-label randomized trial; 194 adults with CKD stages 1–3	Low-salt diet for three months versus usual diet	Blood pressure reductions favoured the low-salt group, and short-term eGFR decline was smaller than in controls; separation in urinary sodium excretion was limited	Contemporary supportive evidence, but short follow-up and modest sodium separation limit conclusions about CKD progression

ACE, angiotensin-converting enzyme; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

Evidence Concerning Blood Pressure Variability

Direct evidence that sodium restriction reduces formally measured blood pressure variability was substantially more limited than evidence concerning mean blood pressure. Most sodium-reduction trials reported office blood pressure, mean ambulatory blood pressure, daytime or nighttime blood pressure, or overall blood pressure control. These outcomes are clinically important but do not independently establish a reduction in blood pressure variability.

A valid assessment of blood pressure variability requires repeated measurements and an explicitly reported variability index, such as the standard deviation, coefficient of variation, weighted standard deviation, average real variability, or variability independent of the mean. The principal dietary intervention studies included in this review did not consistently report these measures. Consequently, reductions in mean systolic or diastolic pressure observed during sodium restriction should not be reclassified as reductions in blood pressure variability.

Observational research confirms that blood pressure variability has prognostic importance in CKD and is associated with cardiovascular and renal outcomes independently of mean blood pressure (7,8). However, this evidence does not demonstrate that restricting sodium intake below 2 g/day directly reduces variability. Real-world analyses have also suggested substantial heterogeneity in individual salt sensitivity, meaning that changes in sodium intake do not produce uniform within-person changes in blood pressure.

The most defensible interpretation is therefore that sodium restriction improves mean blood pressure and may improve the stability of blood pressure through better volume regulation, but direct evidence of an independent effect on standardized blood pressure variability remains insufficient. This outcome should be presented as an important evidence gap rather than as an established benefit.

Table 2. Distinction Between Reported Blood Pressure Outcomes

Outcome category	Common measurement	Evidence in sodium-restriction literature	Interpretation
Office blood pressure	Mean systolic and diastolic pressure recorded during clinical visits	Frequently reported; generally decreases following sodium restriction	Supports an antihypertensive effect
Ambulatory mean blood pressure	Mean 24-hour, daytime, or nighttime systolic and diastolic pressure	Reported in several intervention studies; generally favours lower sodium intake	Provides stronger assessment than isolated office readings but does not itself measure variability
Nocturnal blood pressure pattern	Nighttime pressure or dipping status	Inconsistently reported	Potentially clinically relevant, but evidence is insufficient for firm conclusions
Visit-to-visit variability	Standard deviation, coefficient of variation, average real variability, or variability independent of the mean across visits	Rarely evaluated directly in sodium-restriction trials	Direct treatment effect remains uncertain
Short-term ambulatory variability	Standard deviation, weighted standard deviation, or average real variability within a 24-hour recording	Not consistently reported in the core dietary intervention studies	Mean ambulatory BP reduction must not be interpreted as variability reduction
General "blood pressure stability"	Qualitative or undefined description	Used inconsistently in some reports	Should not be treated as a measured BP variability outcome

BP, blood pressure.

Effects on Proteinuria and Albuminuria

The antiproteinuric effect of sodium restriction was among the most consistent findings across controlled dietary interventions. Slagman et al. demonstrated this interaction particularly clearly in patients with non-diabetic proteinuric nephropathy receiving background angiotensin-converting enzyme inhibition. During regular sodium intake, geometric mean residual proteinuria was 1.68 g/day. Adding angiotensin receptor blockade reduced proteinuria to 1.44 g/day, whereas introducing a low-sodium diet reduced it to 0.85 g/day. Combining low sodium intake with angiotensin receptor blockade reduced residual proteinuria further to 0.67 g/day. Thus, sodium restriction produced a larger antiproteinuric effect than adding a second renin–angiotensin system blocker during regular sodium intake (18).

McMahon et al. similarly found reductions in both proteinuria and albuminuria during low-sodium intake, together with decreased blood pressure and extracellular fluid volume (20). Meuleman et al. reported a mean between-group reduction in urinary protein excretion of 0.4 g/24 hours after three months and 0.3 g/24 hours after six months, despite attenuation of the effect on urinary sodium excretion and ambulatory blood pressure during follow-up (25).

These findings support the view that reduced sodium intake may influence urinary protein excretion through mechanisms extending beyond office blood pressure reduction alone. Lower extracellular fluid volume, reduced systemic and intraglomerular pressure, and improved responsiveness to renin–angiotensin–aldosterone system inhibition are plausible contributors. Nevertheless, proteinuria and albuminuria were measured using different methods across studies, including 24-hour urinary protein, albumin excretion, protein-to-creatinine ratio, and albumin-to-creatinine ratio. These outcomes should therefore be reported separately rather than combined into an unsupported overall percentage reduction.

Observational studies also demonstrate a positive association between urinary sodium and proteinuria. Higher urinary sodium excretion has been correlated with greater urinary protein loss even after adjustment for blood pressure, demographic characteristics, glycaemic measures, and antihypertensive medication. Such findings support a biological relationship but remain unable to establish causality because proteinuria, kidney function, dietary intake, medication use, and urinary sodium measurement may influence one another.

Interaction With Renin–Angiotensin–Aldosterone System Inhibition

The evidence indicates that dietary sodium intake modifies the response to angiotensin-converting enzyme inhibitors and angiotensin receptor blockers. High sodium intake can blunt the expected reductions in blood pressure and proteinuria during renin–angiotensin–aldosterone system blockade, whereas sodium restriction can restore or enhance these responses.

The randomized crossover findings of Slagman et al. demonstrated that reducing sodium intake was more effective in lowering residual proteinuria than adding angiotensin receptor blockade to established angiotensin-converting enzyme inhibition during regular sodium intake (18). Earlier physiological studies similarly showed that dietary sodium influenced the renal and antihypertensive effects of pharmacological renin–angiotensin system blockade (19). Observational analyses have also associated higher sodium exposure with reduced antiproteinuric response and poorer renal outcomes among patients receiving these agents.

These findings suggest that sodium restriction should be regarded as a component of renoprotective treatment rather than as an optional lifestyle measure independent of medication. However, the magnitude of benefit is likely to vary according to baseline sodium intake, achieved dietary reduction, underlying renal disease, proteinuria severity, adherence, diuretic use, and the intensity of pharmacological blockade.

Kidney-Function Decline and Cardiovascular Outcomes

Evidence relating sodium intake to long-term CKD progression was less uniform than evidence concerning blood pressure and proteinuria. In the Chronic Renal Insufficiency Cohort, He et al. analysed repeated 24-hour urinary sodium measurements from 3,757 participants. Compared with participants in the lowest sodium-excretion quartile, those in the highest quartile had higher adjusted risks of CKD progression, all-cause mortality, and the composite of progression or mortality. The adjusted hazard ratio for CKD progression was 1.54, while the hazard ratios for all-cause mortality and the composite outcome were 1.45 and 1.43, respectively (23).

Using the same broader cohort framework, Mills et al. found that higher calibrated urinary sodium excretion was associated with cardiovascular events. Compared with the lowest sodium-excretion quartile, the highest quartile had an adjusted hazard ratio of 1.36 for the composite cardiovascular outcome, 1.34 for heart failure, and 1.81 for stroke. These findings support an association between higher habitual sodium exposure and cardiovascular risk among patients with CKD (31).

The KNOW-CKD study also reported an association between measured 24-hour urinary sodium excretion and CKD progression among patients with stages G3a–G5 CKD (26). Subsequent analysis

demonstrated that baseline proteinuria modified this relationship. Among participants with proteinuria of at least 0.5 g/day, each additional gram per day of urinary sodium excretion was associated with a 29% higher risk of an adverse kidney outcome, whereas sodium excretion was not clearly associated with the primary outcome among participants with lower proteinuria (27). This interaction suggests that patients with substantial proteinuria may be particularly susceptible to the adverse renal effects associated with high sodium exposure.

Ogata et al. used multiple 24-hour urine collections to reduce the measurement error associated with a single dietary assessment. Higher averaged urinary sodium excretion and a higher sodium-to-potassium ratio were associated with a faster decline in estimated glomerular filtration rate, whereas higher potassium excretion was associated with a more favourable trajectory. The authors nevertheless emphasized that observational associations could not determine whether changing sodium and potassium intake would slow kidney-function decline (28).

Not all observational evidence has demonstrated a consistent relationship. Fan et al. found no overall association between 24-hour urinary sodium excretion and kidney failure in non-diabetic CKD. Exploratory analyses suggested that the relationship varied according to baseline proteinuria and sodium-excretion level, but these findings were not sufficiently consistent to establish a uniform exposure–response relationship (17). Such discrepancies may reflect differences in study populations, sodium-measurement methods, proteinuria severity, medication use, kidney-function stage, residual confounding, or reverse causation.

Table 3. Observational Evidence on Sodium Exposure and Clinical Outcomes

Study	Population and exposure assessment	Outcome	Main finding	Interpretive limitation
Fan et al. (17)	Non-diabetic CKD; 24-hour urinary sodium excretion	Kidney failure and composite renal outcome	No overall association between sodium excretion and kidney failure; exploratory relationships varied by proteinuria and sodium level	Findings were subgroup-dependent and did not demonstrate a consistent overall association
He et al. (23)	3,757 CRIC participants; repeated 24-hour urinary sodium measurements	CKD progression and mortality	Highest versus lowest sodium-excretion quartile was associated with greater CKD progression, mortality, and composite risk	Observational design cannot establish that prescribed sodium reduction would reverse the risk
Nerbass et al. (24)	1,607 adults with eGFR 30–59 mL/min/1.73 m²; estimated change in sodium intake	Blood pressure, arterial stiffness, and albuminuria	Reduction to 100 mmol/day or less was associated with improved mean arterial pressure and improvement in albuminuria among albuminuric participants	Sodium intake was estimated rather than repeatedly measured through complete 24-hour collections
Kang et al. (26)	1,254 patients with CKD stages G3a–G5; measured 24-hour urinary sodium	CKD progression	Higher sodium excretion was associated with greater risk of progression	Includes advanced CKD stages and remains susceptible to residual confounding
Kim et al. (27)	967 patients with CKD stages G1–G5; 24-hour urinary sodium and protein	≥50% eGFR decline or kidney replacement therapy	Higher sodium excretion was more strongly associated with adverse outcomes among patients with proteinuria ≥0.5 g/day	Interaction finding requires confirmation and does not prove a treatment effect
Ogata et al. (28)	240 patients with CKD stages 3–5; multiple 24-hour urine collections	Annual eGFR decline	Higher averaged sodium excretion and sodium-to-potassium ratio were associated with faster eGFR decline	Retrospective cohort with a predominantly older and more advanced CKD population
Mills et al. (31)	3,757 CRIC participants; repeated calibrated urinary sodium measurements	Cardiovascular disease, heart failure, myocardial infarction, and stroke	Highest sodium-excretion quartile was associated with greater composite cardiovascular, heart-failure, and stroke risk	Association may reflect dietary pattern, comorbidity, or other unmeasured factors

CKD, chronic kidney disease; CRIC, Chronic Renal Insufficiency Cohort; eGFR, estimated glomerular filtration rate.

Integrated Interpretation of the Evidence

The most consistent direct evidence concerned reductions in mean blood pressure and urinary protein excretion. Controlled trials showed that clinically meaningful sodium restriction can lower office and ambulatory blood pressure, decrease extracellular fluid volume, and reduce proteinuria or albuminuria. The antiproteinuric response was particularly evident among patients already receiving angiotensin-converting enzyme inhibitors or angiotensin receptor blockers.

Evidence concerning blood pressure variability was considerably weaker. Although blood pressure variability is clinically important in CKD, the sodium-restriction trials generally did not report standardized variability measures. The available findings therefore support improved mean blood pressure control but do not establish that sodium restriction independently reduces short-term or visit-to-visit blood pressure variability.

Observational evidence generally associated higher urinary sodium excretion with CKD progression, cardiovascular outcomes, or greater proteinuria, but the results were not entirely uniform. Associations appeared stronger in patients with greater baseline proteinuria, suggesting that sodium exposure may interact with the severity of glomerular injury. Nevertheless, observational results cannot substitute for long-term randomized evidence.

Table 4. Outcome-Level Summary of the Narrative Evidence

Outcome	Overall direction of evidence	Strength of interpretation	Principal qualification
Mean systolic and diastolic blood pressure	Generally favours sodium restriction	Moderate	Mostly based on short-term interventions with variable adherence and sodium separation
Ambulatory blood pressure	Generally favours sodium restriction	Moderate	Benefits may attenuate when adherence declines
Blood pressure variability	Uncertain	Limited	Few studies report standardized variability indices; mean BP reduction is not equivalent to reduced BP variability
Proteinuria and albuminuria	Generally favours sodium restriction	Moderate	Measures and units differ across studies, preventing simple quantitative combination
Response to ACE inhibitors or ARBs	Sodium restriction appears to enhance treatment response	Moderate	Most compelling evidence is derived from proteinuric populations under closely controlled conditions
Extracellular fluid volume	Generally reduced during sodium restriction	Moderate	Primarily supported by short-duration physiological trials
eGFR decline and CKD progression	Observational evidence generally favours lower sodium exposure	Limited to moderate	Long-term randomized evidence is insufficient, and observational findings are not fully consistent
Cardiovascular outcomes	Higher sodium excretion is associated with greater cardiovascular risk	Limited to moderate	Evidence is observational and may be affected by residual confounding
Sustained dietary adherence	Frequently difficult to maintain	Consistent	Behavioural support improves short-term adherence, but effects may diminish over time

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

Taken together, the evidence indicates that reducing dietary sodium can improve mean blood pressure control and decrease proteinuria or albuminuria in patients with non-dialysis CKD. Sodium restriction also appears to enhance the therapeutic response to renin–angiotensin–aldosterone system inhibitors, particularly in patients with clinically important proteinuria. These findings are supported most directly by controlled short-term dietary interventions.

The evidence does not support an equally firm conclusion regarding blood pressure variability. Although sodium restriction may theoretically reduce pressure fluctuations by improving fluid balance and vascular loading, most intervention studies measured mean blood pressure rather than validated variability indices. The effect of sodium restriction on blood pressure variability should therefore remain an unresolved research question.

Long-term cohort studies generally associate higher urinary sodium excretion with CKD progression, cardiovascular events, and mortality, particularly in individuals with greater proteinuria. However, inconsistencies between cohorts and the limitations inherent in observational dietary assessment prevent these associations from being interpreted as definitive evidence that sodium restriction slows progression to kidney failure. Larger and longer randomized trials using repeated objective sodium measurements, standardized blood pressure variability indices, and clinically important renal and cardiovascular endpoints are required.

DISCUSSION

This structured narrative review indicates that reducing dietary sodium intake in adults with non-dialysis CKD generally improves mean blood pressure control, reduces proteinuria or albuminuria, and enhances the therapeutic response to renin–angiotensin–aldosterone system inhibition. These effects were most consistently demonstrated in controlled dietary interventions, particularly among patients with hypertension, clinically important proteinuria, or concurrent treatment with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. Observational studies also generally associated higher urinary sodium excretion with poorer renal and cardiovascular outcomes, although these relationships were not uniform and cannot establish treatment effects. In contrast to the evidence concerning mean blood pressure, the available literature did not provide sufficiently direct or consistent

evidence that sodium restriction independently reduces formally measured blood pressure variability. The findings therefore support sodium reduction as a component of blood pressure and proteinuria management in CKD while identifying blood pressure variability and long-term kidney outcomes as areas of continuing uncertainty.

The observed reduction in mean blood pressure is consistent with the established physiological relationship between sodium intake, extracellular fluid volume, vascular resistance, and salt-sensitive hypertension. Patients with CKD have a reduced capacity to excrete an acute or sustained sodium load, which can promote extracellular volume expansion and increase cardiac output and systemic arterial pressure. Chronic sodium excess may also contribute to arterial stiffness, endothelial dysfunction, and altered neurohormonal regulation, thereby maintaining hypertension even when overt peripheral oedema is absent (3,4). These mechanisms help explain why the blood pressure response to sodium reduction may be greater in CKD than in individuals with preserved kidney function.

The findings of McMahon et al. provide particularly direct evidence of this effect. In their randomized crossover trial, sodium restriction among adults with hypertensive stage 3–4 CKD reduced mean systolic and diastolic blood pressure by approximately 10 and 4 mmHg, respectively, while also reducing extracellular fluid volume, albuminuria, and proteinuria (20). The simultaneous improvement in volume status and blood pressure supports a haemodynamic mechanism rather than a change attributable solely to office measurement variability. A separate randomized crossover trial conducted by Saran et al. similarly found that sodium restriction reduced 24-hour systolic blood pressure, urinary sodium excretion, body weight, and measures of hydration among patients with stage 3–4 CKD (14). The consistency of the blood pressure response across these crossover studies strengthens the inference that sodium reduction can produce a clinically meaningful antihypertensive effect in moderate CKD.

The magnitude of blood pressure reduction observed in these individual trials is also broadly consistent with previous quantitative syntheses. Garofalo et al. reported that lower salt intake reduced both clinic and ambulatory systolic and diastolic blood pressure across randomized trials involving adults with non-dialysis CKD (15). The effect was evident despite differences in CKD stage, sodium targets, intervention duration, and background antihypertensive therapy. Earlier trials in hypertensive populations, including the DASH-Sodium study, similarly demonstrated a graded blood pressure response to lower sodium intake, with larger reductions among participants with hypertension or increased salt sensitivity (11,12). The present synthesis therefore extends an established antihypertensive principle to patients with CKD, in whom sodium sensitivity may be amplified by impaired renal excretion and volume regulation.

The clinical relevance of these blood pressure reductions should not be underestimated. Hypertension is both a cause and a consequence of CKD, and sustained elevation of systolic pressure contributes to cardiovascular disease, glomerular hypertension, and progressive loss of kidney function (2). Even modest reductions in mean blood pressure may therefore have meaningful implications when maintained over time. However, most sodium-restriction trials were short and primarily assessed intermediate outcomes. Consequently, reductions in blood pressure cannot automatically be translated into proven reductions in kidney failure, cardiovascular events, or mortality. The intervention evidence is strongest for physiological and surrogate outcomes and remains less definitive for long-term clinical endpoints.

The behavioural trial by Meuleman et al. adds an important implementation perspective. A structured intervention involving education, motivational interviewing, coaching, and self-monitoring reduced sodium excretion, daytime ambulatory diastolic blood pressure, office blood pressure, and urinary protein excretion at three months (25). Some differences attenuated by six months, indicating that initial dietary change may be achievable but difficult to sustain. This pattern is highly relevant to routine practice because sodium restriction depends on repeated daily decisions rather than a single clinical intervention. Hidden sodium in processed foods, limited nutritional literacy, cultural dietary patterns,

family meal preparation, cost, food availability, and uncertainty regarding food labels may all undermine sustained adherence.

The prospective findings of Nerbass et al. also support the importance of maintained dietary change in real-world care. Adults with early-to-moderate CKD who reduced estimated sodium intake to approximately 100 mmol/day or less experienced greater improvement in mean arterial pressure than participants whose intake increased above this level. Improvements in albuminuria were most apparent among participants who had albuminuria at baseline (24). Although this study was observational and used estimated rather than repeated measured sodium excretion, it suggests that clinically relevant improvements are possible outside tightly controlled feeding studies. It also indicates that the greatest renal benefit may occur in patients with an identifiable treatment target, such as elevated blood pressure or albuminuria, rather than uniformly across all patients with CKD.

The findings concerning proteinuria and albuminuria were similarly persuasive, particularly in patients receiving renin–angiotensin–aldosterone system inhibitors. Proteinuria is not only a marker of glomerular injury but may also contribute to disease progression through tubular uptake of filtered proteins, inflammatory signalling, oxidative stress, and interstitial fibrosis (5,6). Interventions that reduce proteinuria may therefore indicate an improvement in intraglomerular haemodynamics and glomerular barrier stress, even when short-term estimated glomerular filtration rate remains unchanged.

Slagman et al. provided the clearest demonstration of the interaction between sodium intake and pharmacological renoprotection. In patients with non-diabetic proteinuric nephropathy receiving maximal angiotensin-converting enzyme inhibition, reducing sodium intake lowered residual proteinuria more substantially than adding an angiotensin receptor blocker during regular sodium intake. Combining sodium restriction with angiotensin receptor blockade produced the lowest residual proteinuria (18). This result suggests that persistent high sodium intake can create a state of relative resistance to renin–angiotensin system blockade and that dietary sodium reduction may restore part of the expected therapeutic response.

Earlier work by Vogt et al. similarly demonstrated that dietary sodium intake modifies the antiproteinuric and antihypertensive effects of pharmacological renin–angiotensin system blockade (19). Lambers Heerspink et al. also reported that moderation of dietary sodium potentiated the renal effects of losartan among patients with proteinuric kidney disease (22). Considered together, these studies indicate that sodium intake is not merely an independent lifestyle exposure but a determinant of the effectiveness of established pharmacological treatment. A patient who continues to consume a high-sodium diet may experience a smaller reduction in proteinuria despite apparently appropriate medication selection and dosing.

Several mechanisms may explain this interaction. High sodium intake promotes extracellular fluid expansion and maintains systemic and intraglomerular pressure, potentially counteracting the haemodynamic effects of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers. Sodium loading may also alter renal vascular responsiveness and contribute to aldosterone breakthrough or incomplete suppression of sodium-retaining pathways. Conversely, sodium restriction reduces volume-dependent pressure, decreases glomerular capillary stress, and may improve the haemodynamic response to renin–angiotensin system inhibition (13,18,19). These mechanisms are consistent with the concurrent reductions in blood pressure, volume status, albuminuria, and proteinuria observed in controlled dietary trials.

The proteinuria response was not completely uniform across studies. McMahon et al. observed reductions in both proteinuria and albuminuria during low-sodium intake (20), while Meuleman et al. reported a reduction in urinary protein excretion that persisted longer than some of the changes in sodium excretion and ambulatory blood pressure (25). In contrast, Saran et al. reported clinically

important improvements in blood pressure and hydration but did not demonstrate a statistically clear change in the albumin-to-creatinine ratio (14). These differences may reflect variation in baseline proteinuria, underlying kidney disease, intervention duration, achieved sodium separation, medication use, sample size, and the precision of urinary protein measurements.

The method used to assess urinary protein may further contribute to heterogeneity. Twenty-four-hour protein excretion, urine protein-to-creatinine ratio, urinary albumin excretion, and urine albumin-to-creatinine ratio measure related but non-identical outcomes. Albuminuria may be more sensitive to glomerular permeability, whereas total protein measurements may capture albumin and non-albumin proteins. Biological day-to-day variation and incomplete urine collection can also influence apparent treatment responses. For this reason, the findings support an overall antiproteinuric direction of effect but do not justify combining all reported outcomes into a single unqualified percentage reduction.

A major contribution of the present review is the distinction between mean blood pressure and blood pressure variability. The reviewed intervention studies consistently evaluated office or ambulatory mean blood pressure, whereas relatively few directly quantified within-person variability using validated indices. Blood pressure variability may be assessed over beat-to-beat, 24-hour, day-to-day, or visit-to-visit intervals, and each timescale reflects different physiological and behavioural influences. Common measures include the standard deviation, coefficient of variation, average real variability, and variability independent of the mean. A reduction in average systolic pressure does not necessarily imply a reduction in any of these variability measures.

Previous literature supports the prognostic relevance of blood pressure variability in renal disease. Greater variability has been associated with albuminuria, vascular injury, renal-function decline, cardiovascular events, and mortality independently of mean blood pressure in several cohorts (7,8). Home blood pressure studies have also linked day-to-day variation with CKD progression, while visit-to-visit variability has been associated with adverse cardiovascular outcomes in predialysis CKD populations. These observations provide a strong rationale for investigating blood pressure variability as a therapeutic target.

However, evidence that sodium restriction directly modifies blood pressure variability remains limited. The biological hypothesis is plausible: reduced sodium exposure may stabilize extracellular fluid volume, reduce fluctuations in vascular loading, improve arterial compliance, and lessen episodic pressure changes related to variable dietary intake. Nevertheless, the core intervention studies primarily reported mean office or ambulatory blood pressure. Descriptions such as improved blood pressure control, lower nighttime pressure, or greater stability should not be interpreted as reductions in variability unless a defined variability statistic was calculated. The present findings therefore support sodium restriction for lowering mean blood pressure but do not establish an independent effect on blood pressure variability.

This distinction is important because mean pressure and variability may have overlapping but different prognostic implications. Some studies have found that visit-to-visit variability predicts renal or cardiovascular outcomes independently of mean pressure, whereas others have reported that mean on-treatment systolic pressure is a stronger or more consistent predictor of renal outcomes. Differences in measurement frequency, follow-up duration, medication adherence, antihypertensive drug class, comorbidity, and statistical adjustment may explain these conflicting results. Future dietary trials should prospectively define blood pressure variability as an outcome rather than deriving it opportunistically from a small number of measurements.

The evidence relating sodium exposure to long-term CKD progression was less consistent than the short-term intervention evidence. In the Chronic Renal Insufficiency Cohort, higher repeated urinary sodium excretion was associated with increased risks of CKD progression, mortality, and combined adverse outcomes (23). Kang et al. similarly reported that the highest category of measured 24-hour urinary

sodium excretion was associated with a greater risk of CKD progression in the KNOW-CKD cohort (26). These studies benefit from clinically important endpoints and objective urinary assessment, but their observational designs prevent firm causal interpretation.

The analysis by Kim et al. provides an important refinement by suggesting that the association between sodium exposure and adverse kidney outcomes depends partly on baseline proteinuria. Higher sodium excretion was more strongly associated with progression among participants with proteinuria of at least 0.5 g/day, whereas the relationship was not clear among those with lower proteinuria (27). This interaction is biologically plausible because patients with greater glomerular injury may be more susceptible to the adverse haemodynamic effects of sodium loading. It is also consistent with the stronger antiproteinuric effect of sodium restriction observed in proteinuric populations receiving renin–angiotensin system blockade.

Ogata et al. strengthened exposure assessment by averaging multiple 24-hour urine collections rather than relying on a single baseline measurement. Higher averaged sodium excretion and a higher sodium-to-potassium ratio were associated with faster decline in estimated glomerular filtration rate, whereas higher potassium excretion was associated with a more favourable trajectory (28). Repeated urine collections reduce random day-to-day measurement error and better approximate habitual intake. Nevertheless, the study included patients with more advanced CKD, and the authors appropriately recognized that observational associations do not prove that changing sodium or potassium intake will alter renal decline.

Other cohorts have produced less consistent findings. Fan et al. found no overall association between 24-hour urinary sodium excretion and kidney failure among patients with non-diabetic CKD, although exploratory analyses suggested interactions with baseline proteinuria and sodium level (17). Smyth et al. similarly did not identify a consistent association between estimated sodium excretion and major renal outcomes in a high-cardiovascular-risk population with and without CKD (16). These discrepant findings caution against assuming a simple linear relationship between sodium excretion and kidney failure across all CKD populations.

Several explanations may account for these inconsistencies. Urinary sodium excretion reflects recent intake but may vary substantially between days, particularly when based on a single collection. Incomplete urine collection can produce additional error. Reduced sodium excretion in advanced CKD may reflect poor appetite, lower energy intake, frailty, diuretic use, or more severe illness rather than successful dietary restriction. Reverse causation is therefore possible, especially when patients with advanced disease reduce food intake or receive intensive dietary advice. Statistical adjustment may not fully account for differences in kidney function, proteinuria, blood pressure, medication, diabetes, body composition, or socioeconomic circumstances.

The sodium-to-potassium balance may also be clinically relevant. A diet high in sodium is frequently low in fruits, vegetables, and other potassium-containing foods, although potassium intake must be individualized in patients at risk of hyperkalaemia. Ogata et al. observed opposing associations of urinary sodium and potassium excretion with estimated glomerular filtration rate decline (28). These findings suggest that sodium may sometimes function as a marker of the broader dietary pattern rather than as an isolated exposure. However, the present review focused on sodium restriction and cannot determine the independent or combined effects of potassium modification.

Observational cardiovascular evidence further supports caution regarding high sodium exposure in CKD. Mills et al. found that higher urinary sodium excretion was associated with increased risks of cardiovascular events, particularly heart failure and stroke, in the Chronic Renal Insufficiency Cohort (31). This relationship is clinically plausible because sodium-driven volume expansion, hypertension, arterial stiffness, and left ventricular loading may be especially consequential in CKD, where cardiovascular risk is already elevated. Nevertheless, as with renal outcomes, observational associations

cannot confirm that dietary sodium reduction will produce corresponding reductions in cardiovascular events.

The short duration of most intervention trials remains a central limitation. Previous reviews have noted that sodium-restriction studies in CKD have generally lasted weeks or a few months, which is sufficient to evaluate urinary sodium, blood pressure, extracellular volume, and proteinuria but inadequate for kidney failure, major cardiovascular events, hospitalization, or mortality (15,32). The more recent trial by Trakarnvanich et al. reported a smaller short-term decline in estimated glomerular filtration rate and greater blood pressure reduction among participants assigned to a low-salt diet (36). However, the three-month follow-up and modest separation in urinary sodium excretion limit the extent to which these findings can establish protection against long-term CKD progression.

A short-term change in estimated glomerular filtration rate also requires careful interpretation. Haemodynamic interventions can alter intraglomerular pressure and produce changes in estimated filtration that do not necessarily represent structural kidney injury or protection. A slower decline over several weeks cannot be assumed to predict sustained preservation of kidney function, particularly when baseline and follow-up creatinine measurements are affected by hydration, dietary intake, medication, and biological variability. Longer trials with repeated kidney-function measurements and clinical endpoints are therefore needed.

The present findings have several practical implications. Dietary sodium restriction should be integrated with pharmacological management, especially among patients with uncontrolled hypertension, extracellular volume expansion, or clinically important proteinuria. The evidence suggests that sodium reduction may increase the effectiveness of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers rather than merely adding a small independent lifestyle benefit (18,19,22). Dietary assessment should therefore be considered when a patient demonstrates persistent hypertension or proteinuria despite apparently appropriate renoprotective treatment.

A uniform instruction to “avoid salt” is unlikely to produce sustained adherence. Effective implementation requires translation of the sodium target into practical food choices, identification of processed and restaurant foods, interpretation of nutritional labels, modification of culturally important recipes, and involvement of family members responsible for meal preparation. Repeated reinforcement, dietitian involvement, home blood pressure monitoring, and feedback based on urinary sodium measurements may support adherence. The attenuation of benefit observed after the active behavioural intervention in Meuleman et al. demonstrates that maintenance strategies are as important as initial education (25).

Objective monitoring may be useful, but no single method is ideal. Twenty-four-hour urinary sodium excretion is generally more informative than dietary recall or spot urine estimation, yet it is burdensome and susceptible to incomplete collection. Repeated collections improve reliability but may be impractical in routine care. Spot urine equations are more convenient but may produce biased estimates at the individual level. Clinical interpretation should therefore combine dietary history, blood pressure, volume status, urinary protein, medication use, and, where feasible, objective sodium assessment.

The review has several strengths. It distinguishes controlled sodium-reduction interventions from observational sodium-exposure studies, thereby avoiding the assumption that an association automatically represents a treatment effect. It also separates mean blood pressure from blood pressure variability and proteinuria from albuminuria, reducing the risk of combining clinically related but methodologically distinct outcomes. The synthesis integrates haemodynamic, renal, behavioural, and long-term observational evidence and interprets the findings according to study design rather than relying solely on the direction of reported results.

The limitations of the evidence base must nevertheless be recognized. Many intervention studies were small, short, and unable to blind participants to dietary allocation. Sodium targets and achieved reductions varied considerably, and some studies enrolled mixed CKD stages rather than exclusively stage 2–3 disease. Background antihypertensive treatment, diuretic use, underlying kidney diagnosis, baseline sodium intake, proteinuria severity, and dietary counselling differed across studies. Outcomes were assessed using heterogeneous methods, and direct measures of blood pressure variability were uncommon.

The structured narrative-review design also has inherent limitations. Although the literature search was organized around predefined clinical concepts and priority was given to methodologically relevant studies, the review was not intended to provide an exhaustive systematic search, formal risk-of-bias scoring, or pooled quantitative estimate. Selection and interpretation of studies may therefore be influenced by the availability and clinical relevance of the published evidence. The conclusions should be understood as a critical synthesis rather than a formal estimate of treatment efficacy.

Future randomized trials should focus specifically on adults with stage 2–3 CKD and use a clearly defined sodium target, objective repeated assessment of achieved intake, and sufficient follow-up to assess sustainability. Blood pressure outcomes should include mean office and ambulatory measurements together with prespecified variability indices such as standard deviation, coefficient of variation, average real variability, and variability independent of the mean. Proteinuria and albuminuria should be reported using standardized units and repeated measurements, and analyses should account for baseline proteinuria, kidney diagnosis, medication, volume status, and adherence.

Longer studies should evaluate whether sustained sodium restriction alters estimated glomerular filtration rate trajectory, kidney failure, cardiovascular events, hospitalization, quality of life, and mortality. Stratified analyses are needed to determine whether benefit is greatest among patients with high baseline sodium intake, uncontrolled hypertension, substantial proteinuria, diabetes, salt sensitivity, or concurrent renin–angiotensin–aldosterone system inhibition. Trials should also assess potential harms, including symptomatic hypotension, excessive volume depletion, nutritional compromise, and reduced dietary quality.

Overall, the current evidence most strongly supports dietary sodium restriction as a means of reducing mean blood pressure and urinary protein excretion and enhancing the response to renin–angiotensin–aldosterone system inhibitors in non-dialysis CKD. The evidence is less conclusive for blood pressure variability and long-term progression to kidney failure. Sodium restriction should therefore be presented as an evidence-supported component of comprehensive CKD management, while claims that it independently stabilizes blood pressure variability or prevents major renal and cardiovascular events should remain appropriately qualified.

CONCLUSION

Dietary sodium restriction below 2 g/day appears to be a clinically valuable component of comprehensive management for adults with stage 2–3 chronic kidney disease, particularly those with hypertension, extracellular volume expansion, proteinuria, or albuminuria. The available intervention evidence consistently supports reductions in mean office and ambulatory blood pressure, urinary protein excretion, and extracellular fluid volume, while also indicating that lower sodium intake can enhance the antihypertensive and antiproteinuric effects of renin–angiotensin–aldosterone system inhibitors. Observational evidence further suggests that higher habitual sodium exposure may be associated with faster kidney-function decline and greater cardiovascular risk, especially among patients with substantial baseline proteinuria, although these associations remain vulnerable to confounding and cannot establish treatment efficacy. Evidence concerning blood pressure variability is substantially less developed because most studies assessed mean blood pressure rather than standardized indices of within-person variability. Sodium restriction should therefore be recommended as an adjunct to

pharmacological renoprotection and individualized dietary counselling, while claims regarding independent improvement in blood pressure variability or prevention of kidney failure and cardiovascular events should remain cautious. Longer randomized trials using repeated objective sodium measurements, standardized blood pressure variability metrics, consistent proteinuria assessments, and clinically important renal and cardiovascular outcomes are required.

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