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Narrative Review on Dietary Modulation of Inflammation in Nephrotic Syndrome and Chronic Kidney Disease

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Abstract

Purpose: The purpose of this study is to investigate the role of chronic inflammation in the progression of nephrotic syndrome (NS) and chronic kidney disease (CKD), with a focus on mechanisms including glomerular injury, proteinuria-induced tubular NF-κB activation, oxidative stress, uremic toxin accumulation, gut dysbiosis, and their contribution to cardiovascular complications and progression to end-stage renal disease (ESRD).

Methodology: Literature searches from inception to December 20, 2025, were performed on PubMed, google scholar, and the Since Direct using key words such as chronic kidney disease, inflammatory markers, nephrotic syndrome were related terms. All relevant studies in the review focusing on proinflammatory, inflammatory biomarkers included if sufficient data were available.

Findings: Anti-inflammatory dietary strategies in CKD and NS patients show potential to lower proinflammatory marker levels. Larger, multicenter randomized controlled trials (RCTs) are needed to establish efficacy, safety, and quality-of-life benefits.

Unique Contribution to Theory, Policy and Practice: This review uniquely contributes by integrating dietary modulation into the inflammation-driven pathophysiology of CKD and NS, offering a mechanistic, clinically relevant, and policy-informing framework for nutrition-based interventions.

Keywords: *Nephrotic Syndrome, Chronic Kidney Disease, Inflammation, Oxidative Stress, Gut Dysbiosis, Omega-3 PUFAs, Mediterranean Diet*

Introduction

Nephrotic syndrome (NS) and chronic kidney disease (CKD) are both characterized by chronic low-grade systemic inflammation that plays a central role in disease progression and complications. In NS, clinical manifestations such as proteinuria, hypoalbuminemia, edema, and dyslipidemia are accompanied by inflammatory processes contributing to podocyte injury, glomerular damage, and renal fibrosis. Proteinuria further amplifies tubular inflammation through activation of NF-κB signaling, oxidative stress, and immune cell recruitment [1]. In CKD, inflammation is driven by uremic toxin accumulation, oxidative stress, gut dysbiosis, and recurrent infections, leading to persistent elevation of inflammatory markers including CRP, IL-6, and TNF-α, which are strongly associated with disease progression, cardiovascular complications, and increased mortality [2]. Oxidative stress reinforces inflammation via excessive ROS production, creating a self-perpetuating cycle that accelerates renal fibrosis and functional decline. Both NS and CKD share overlapping mechanisms in which inflammation and oxidative stress mutually reinforce each other, contributing to worsening renal function, cardiovascular disease (CVD), and progression to end-stage renal disease (ESRD) [3]. Additional pathogenic factors, including RAAS activation, insulin resistance, and endothelial dysfunction, further exacerbate these outcomes. Dietary factors also influence disease progression; while protein restriction may slow CKD progression, it may increase risk of malnutrition. Emerging evidence suggests that anti-inflammatory dietary patterns, including reduced red meat intake and increased dietary fiber, may decrease pro-inflammatory cytokines and oxidative stress, although clinical evidence remains inconsistent, particularly for specific supplements such as curcumin [4].

Aim of the Review

This narrative review aims to synthesize current evidence on the role of inflammation in NS and CKD progression and to evaluate the effectiveness of anti-inflammatory dietary interventions in reducing oxidative stress and pro-inflammatory biomarkers in these populations.

Review Objectives

1. The review systematically summarizes inflammatory mechanisms in NS from glomerular proteinuria to uremic inflammation in CKD driven by toxins, gut dysbiosis, and oxidative stress.
2. To summarize the major dietary factors implicated in modulating inflammatory pathways relevant to NS and CKD.
3. To evaluate the evidence for specific dietary patterns (e.g., Mediterranean, plant-based) or individual nutrients (e.g., omega-3 fatty acids, antioxidants) in ameliorating inflammation in these conditions.
4. Emphasize the growing evidence linking diet to inflammatory pathways.

5. To identify current challenges, synthesizing current evidence and highlighting gaps in research and future research directions in this field.

Methodology

Search Strategy and Selection Criteria

A comprehensive literature search was conducted using electronic databases, including PubMed, Google Scholar, the Cochrane Database of Systematic Reviews, ClinicalTrials.gov, and ScienceDirect. The search covered studies published from database inception through December 20, 2025. Relevant articles were identified using combinations of the following keywords and Medical Subject Headings (MeSH) terms: “chronic kidney disease,” “nephrotic syndrome,” “anti-inflammatory diet,” and CKD nutrition anti-inflammatory. Articles were screened and selected based on predefined inclusion and exclusion criteria, focusing on studies that evaluated the relationship between inflammation, dietary interventions, and clinical outcomes in patients with CKD and nephrotic syndrome.

Eligibility Criteria

Studies were included if they met the following criteria:

- Conducted in adult populations (≥18 years) diagnosed with nephrotic syndrome (NS) or chronic kidney disease (CKD).
- Evaluated dietary or nutritional interventions, particularly those with antioxidant or anti-inflammatory properties.
- Reported outcomes related to oxidative stress and pro-inflammatory biomarkers, including C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α).

A systematic search was conducted in PubMed, ScienceDirect , and Google Scholar for studies published between 2010 and 2025. Keywords included 'nephrotic syndrome', 'chronic kidney disease', 'inflammation', 'dietary intervention', 'omega-3 fatty acids', 'antioxidants', 'plant-based diet', and 'Mediterranean diet'. Original research articles, systematic reviews, and meta-analyses in English were included, while case reports and editorials were excluded. Excluded from this review were studies, research on pediatric patients (< 18 years old) , and non-English language articles.

Data Extraction and Synthesis: Key elements extracted included study design (e.g., RCTs, cohorts), participant profiles (NS/CKD stage, age, comorbidities), interventions (type, duration, components like omega-3s or plant-based diets), and outcomes (inflammatory markers, proteinuria, eGFR). Narrative synthesis grouped findings by dietary patterns (e.g., Mediterranean, low-protein, antioxidant-rich), noting consistency (stronger evidence for omega-3s reducing IL-6 in CKD).The flow chart below shows the search findings.

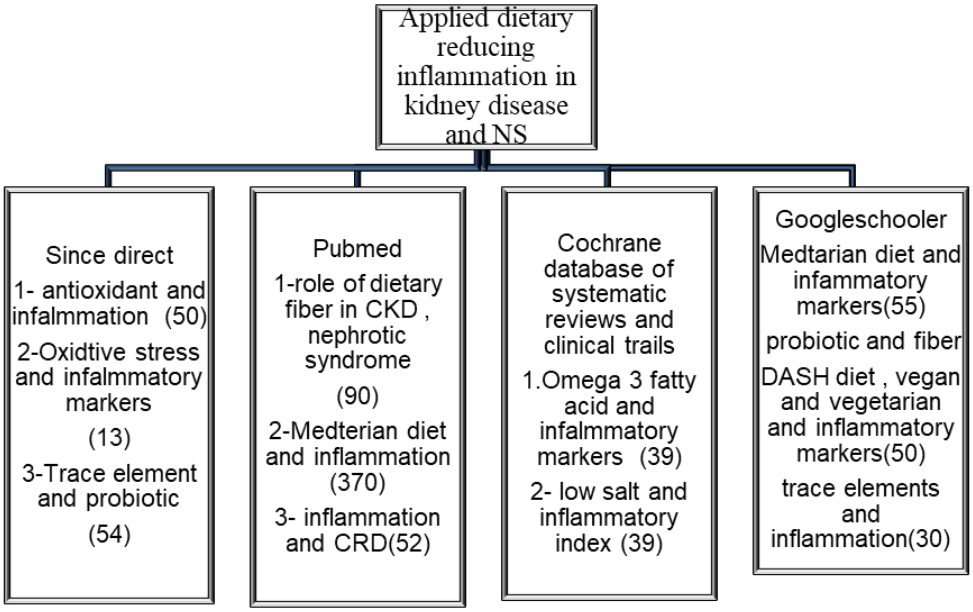

Figure (1): PRISMA Flow Diagram of Study Selection

Dietary strategies to reduce inflammation in kidney disease.

1. Anti-inflammatory Nutrients

a. Omega 3 Polyunsaturated Fatty Acids (PUFAs)

b. Antioxidants (Vitamins, Polyphenols, trace elements)

c. Fiber and Prebiotics

2. Plant-Based Diets and Specific Dietary Patterns

a. Mediterranean Diet

b. Vegetarian/Vegan Diets

c. DASH diet

d. Fasting Mimicking Diet (FMD)

e. Dietary Inflammatory Index (DII) and Sodium Restriction

Omega-3 Polyunsaturated Fatty Acids (PUFAs)

Long-chain omega-3 polyunsaturated fatty acids (PUFAs), particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), exhibit anti-inflammatory, anti-fibrotic, and renoprotective effects in kidney disease. These fatty acids integrate into cell membranes, alter lipid mediator production, and reduce inflammatory signaling pathways. EPA and DHA compete with arachidonic acid for cyclooxygenase (COX) and lipoxygenase (LOX) enzymes, leading to lower

production of pro-inflammatory mediators and increased formation of specialized pro-resolving mediators such as resolvins and protectins. In addition, they inhibit NF-κB activation and reduce cytokine production including TNF-α, IL-6, and IL-1β [5–8].

Clinical evidence suggests that omega-3 supplementation may modestly reduce inflammation and proteinuria in nephrotic syndrome (NS) and chronic kidney disease (CKD), although findings remain heterogeneous. In IgA nephropathy, several randomized controlled trials and meta-analyses demonstrated significant reductions in proteinuria without consistent improvement in glomerular filtration rate (GFR), indicating mainly anti-proteinuric and anti-inflammatory effects [9]. In CKD and hemodialysis patients, omega-3 supplementation has also been associated with lower CRP, IL-6, and TNF-α levels, improved endothelial and cardiovascular function, and possible slowing of CKD progression [10–12]. However, evidence remains limited by small sample sizes, short intervention durations, and variability in CKD stage and disease etiology.

Antioxidants (Vitamins and Polyphenols)

Oxidative stress and inflammation are closely interconnected in CKD and nephrotic syndrome, creating a cycle that accelerates renal injury and fibrosis. Excess reactive oxygen species (ROS) activate inflammatory pathways such as NF-κB, increasing production of pro-inflammatory cytokines including TNF-α, IL-6, and CRP [13,15]. Antioxidant therapies aim to interrupt this cycle by scavenging ROS and suppressing inflammatory signaling. Polyphenols such as curcumin, resveratrol, pomegranate extract, and grape-derived antioxidants have shown anti-inflammatory and antioxidative properties in experimental studies [14]. Animal studies demonstrated that curcumin reduces renal inflammation, fibrosis, and oxidative stress through suppression of TNF-α and NF-κB pathways [15]. However, clinical studies in CKD and hemodialysis patients have produced inconsistent results, with several randomized trials and meta-analyses reporting no significant reductions in CRP, IL-6, or TNF-α after supplementation [14–17]. Similarly, resveratrol supplementation showed limited effects on antioxidant enzymes and inflammatory biomarkers [17].

Vitamins C and E are among the most studied antioxidant vitamins in CKD. Vitamin E supplementation has been associated with improved antioxidant capacity, reduced lipid peroxidation, and lower CRP levels in dialysis patients [17]. Vitamin C may also reduce CRP, although findings regarding oxidative stress markers remain inconsistent [17–19]. Vitamin D additionally exhibits immunomodulatory effects by suppressing pro-inflammatory cytokines and enhancing regulatory T-cell activity, with observational studies linking adequate vitamin D status to lower proteinuria and inflammation [19].

Trace elements such as selenium and zinc also contribute to antioxidant defense. Selenium functions through selenoproteins such as glutathione peroxidase, while zinc supports superoxide dismutase activity and inhibits NF-κB signaling. Supplementation with selenium and zinc in CKD

and hemodialysis patients has been associated with reductions in oxidative stress markers and inflammatory mediators, as well as improvements in nutritional and immune status [20,21]. Overall, antioxidants may provide supportive benefits in reducing oxidative stress and inflammation in CKD and NS, but larger and longer-term clinical trials are needed to confirm their effects on renal outcomes.

Dietary Fiber and Prebiotics

Gut microbiota dysbiosis is common in chronic kidney disease (CKD) and likely contributes to nephrotic syndrome (NS) progression. CKD is characterized by reduced beneficial short-chain fatty acid (SCFA)-producing bacteria and increased proteolytic bacteria that generate uremic toxins such as indoxyl sulfate (IS) and p-cresyl sulfate (PCS). These toxins promote oxidative stress, NF-κB activation, inflammation, fibrosis, and cardiovascular complications [22,23]. Dietary fiber and prebiotics, including inulin and fructo-oligosaccharides, help restore gut microbial balance by promoting saccharolytic fermentation and increasing beneficial bacteria such as Bifidobacterium. Fermentation of fiber produces SCFAs (acetate, propionate, and butyrate), which strengthen intestinal barrier integrity, reduce gut permeability, lower endotoxemia, and exert systemic anti-inflammatory effects [24]. Higher dietary fiber intake has also been associated with improved bowel function, reduced metabolic acidosis, lower inflammation, and decreased mortality risk in CKD patients [25].

Clinical studies suggest that prebiotics, probiotics, and synbiotics may reduce circulating uremic toxins, particularly PCS, while modestly improving inflammatory and renal parameters. Inulin and resistant starch supplementation have shown beneficial effects on gut microbiota composition, SCFA production, oxidative stress, and inflammatory markers, although findings for IS reduction remain inconsistent [27–32]. Overall, microbiota-targeted nutritional interventions appear promising adjunct therapies in CKD and potentially NS, but larger long-term trials are still required.

Plant-Based Diets and Specific Dietary Patterns

Mediterranean Diet

The Mediterranean diet is a plant-based dietary pattern rich in fruits, vegetables, legumes, whole grains, nuts, olive oil, and fish, with low intake of red meat and processed foods. Its anti-inflammatory properties are attributed to high levels of fiber, polyphenols, antioxidants, monounsaturated fats, and omega-3 fatty acids, which collectively reduce oxidative stress and inflammatory signaling [33]. Evidence from clinical trials and cohort studies indicates that the Mediterranean diet lowers inflammatory biomarkers such as CRP and IL-6, improves endothelial and cardiovascular function, and promotes a healthier SCFA-producing gut microbiota [33,34]. In CKD, higher adherence to Mediterranean-style diets has been associated with better kidney function, lower creatinine and urea levels, reduced CKD incidence, and lower mortality risk

[35,36]. Despite concerns regarding potassium and phosphate intake from plant foods, appropriately modified Mediterranean-style diets may still be suitable for CKD patients when individualized by dietitians. Plant-based foods also provide alkali and fiber, helping reduce metabolic acidosis, constipation, inflammation, and gut dysbiosis [33,35].

DASH Diet

The Dietary Approaches to Stop Hypertension (DASH) diet emphasizes fruits, vegetables, whole grains, low-fat dairy products, nuts, and lean proteins while restricting sodium, saturated fat, and processed foods. The diet is rich in potassium, magnesium, calcium, antioxidants, and fiber, contributing to improved endothelial function, blood pressure reduction, and lower oxidative stress and inflammation [37]. In CKD and nephrotic syndrome, DASH-based dietary patterns may help control hypertension and reduce proteinuria, thereby slowing renal progression. However, modifications are often necessary in advanced CKD to limit potassium, phosphate, and sodium intake while maintaining adequate nutritional balance [37].

Vegetarian and vegan diets emphasize plant-based foods with lower intake of animal protein and higher intake of fiber, antioxidants, and phytochemicals, offering potential renoprotective effects in nephrotic syndrome (NS) and chronic kidney disease (CKD). These dietary patterns may reduce metabolic acidosis, decrease glomerular hyperfiltration and proteinuria, and improve inflammation through modulation of gut microbiota and suppression of NF-κB signaling [38]. Evidence from observational studies and clinical trials suggests that plant-based diets are associated with slower CKD progression, preservation of estimated glomerular filtration rate (eGFR), and reduced albuminuria compared with diets rich in animal protein [38]. Plant proteins also appear to produce less phosphorus retention and renal stress than animal-derived proteins, which may benefit patients with advanced CKD. Increased fruit and vegetable intake has additionally been shown to reduce dietary acid load, lower blood pressure, and decrease proteinuria, with effects comparable to sodium bicarbonate therapy in some CKD patients [38]. Vegetarian diets may also improve insulin sensitivity and glucose metabolism, which are important factors in CKD progression [38]. Despite these benefits, strict vegetarian or vegan diets in NS and CKD require careful monitoring to avoid deficiencies in vitamin B12, iron, zinc, omega-3 fatty acids, and protein inadequacy. Therefore, individualized dietitian-guided plant-forward approaches are generally preferred over unrestricted vegan diets, particularly in dialysis or severe proteinuric states [38].

Fasting-Mimicking Diet (FMD)

The fasting-mimicking diet (FMD) is a periodic low-calorie, low-protein, plant-based dietary regimen designed to reproduce the metabolic effects of fasting while maintaining nutrient intake. A low-salt fasting-mimicking diet (LS-FMD) has been adapted for kidney disease patients to reduce glomerular stress and inflammation [39]. Preclinical studies in CKD and glomerulopathy models demonstrated that repeated LS-FMD cycles improved podocyte function, restored

glomerular filtration barrier integrity, reduced albuminuria and tubular injury, and promoted renal regenerative pathways [39,40]. Proposed mechanisms include reduced oxidative stress and inflammation, activation of autophagy, improved insulin sensitivity, and stimulation of nephrogenic and repair-related gene pathways [40,41]. Early human pilot studies also suggest that LS-FMD may reduce proteinuria, improve endothelial function, and lower inflammatory markers in CKD patients, with some benefits persisting after one year of follow-up [39]. Although these findings are promising, current evidence remains limited to small studies, and larger randomized clinical trials are needed before routine clinical application can be recommended.

Dietary Inflammatory Index (DII) and Sodium Restriction

The Dietary Inflammatory Index (DII) is a tool used to assess the inflammatory potential of dietary patterns. Higher DII scores reflect pro-inflammatory diets rich in saturated fats, refined carbohydrates, red meat, and sodium, whereas lower scores indicate anti-inflammatory diets rich in fiber, omega-3 fatty acids, fruits, vegetables, and polyphenols [42,43]. Pro-inflammatory diets promote oxidative stress, NF-κB activation, endothelial dysfunction, and cytokine production, thereby contributing to CKD progression and renal inflammation [44]. Excess sodium intake is particularly important in nephrotic syndrome and CKD because it worsens hypertension, proteinuria, edema, and glomerular injury. Sodium restriction (<2 g/day) has been associated with improved blood pressure control, reduced proteinuria, preservation of kidney function, and lower inflammatory burden [43]. Epidemiological studies consistently demonstrate that higher DII scores are associated with increased CKD risk and poorer renal function. Meta-analyses reported that individuals consuming highly pro-inflammatory diets had significantly greater odds of CKD compared with those consuming anti-inflammatory diets [45]. Similarly, Western dietary patterns characterized by processed foods, refined grains, red meat, and sodium have been linked to accelerated kidney function decline and increased microalbuminuria [46,47]. These findings support the importance of adopting anti-inflammatory dietary patterns as part of CKD and NS management strategies.

Dietary Components that Promote Inflammation

1. **Western Diet, Inflammaging, and CKD**

The Western diet (WD), characterized by high intake of processed foods, red meat, saturated fats, refined sugars, and phosphate additives, contributes significantly to chronic low-grade inflammation (“inflammaging”), oxidative stress, gut dysbiosis, and progression of chronic diseases including nephrotic syndrome (NS) and chronic kidney disease (CKD) [48,49]. These dietary patterns promote microbiota imbalance, increased production of uremic toxins, endothelial dysfunction, and activation of inflammatory pathways such as NF-κB, while suppressing protective antioxidant pathways including Nrf2 [48,51]. In CKD and renal replacement therapy (RRT) patients, processed foods rich in inorganic phosphate additives further worsen

hyperphosphatemia, vascular calcification, and renal inflammation because inorganic phosphate is highly absorbable compared with organic phosphate sources [50]. Westernized dietary habits also accelerate obesity, metabolic dysfunction, cardiovascular disease, and premature vascular aging. Conversely, plant-based dietary patterns rich in fiber, antioxidants, omega-3 fatty acids, and polyphenols may help counteract these inflammatory effects and improve gut microbiota homeostasis [50,51].

2. High Sodium Intake

Excess sodium intake contributes to hypertension, endothelial dysfunction, vascular inflammation, and kidney injury through mechanisms extending beyond blood pressure elevation [52]. High sodium intake activates oxidative stress pathways, increases reactive oxygen species (ROS) generation, stimulates NF-κB and NLRP3 inflammasome signaling, and alters gut microbiota composition, promoting systemic inflammation and vascular dysfunction [52–55]. In nephrotic syndrome, excessive sodium intake worsens edema, proteinuria, glomerular hyperfiltration, and salt-sensitive hypertension through enhanced sodium reabsorption in the collecting ducts [56]. In CKD, sodium intake above 2 g/day is associated with increased proteinuria, accelerated renal decline, and higher cardiovascular risk [56]. High sodium exposure also activates immune cells including macrophages, dendritic cells, and T lymphocytes, further amplifying inflammatory cytokine production and endothelial injury [57,58]. Therefore, sodium restriction remains a cornerstone of dietary management in NS and CKD.

3**. High Phosphate Intake**

Elevated phosphate intake, particularly from processed foods and phosphate additives, promotes inflammation, oxidative stress, vascular calcification, and CKD progression [59]. Inorganic phosphate acts as a pro-inflammatory stimulus by activating the NLRP3 inflammasome and increasing production of inflammatory cytokines such as IL-1β [61]. Hyperphosphatemia additionally induces oxidative stress and ROS generation, contributing to endothelial dysfunction, fibrosis, vascular calcification, and accelerated aging [62].

Experimental studies have demonstrated that high phosphate diets worsen glomerulosclerosis, tubular injury, interstitial fibrosis, and renal inflammation, whereas phosphate restriction slows CKD progression and attenuates inflammatory damage [63,64]. Elevated urinary phosphate may also promote formation of calcium-phosphate particles that activate toll-like receptor 4 (TLR4)-mediated tubulointerstitial inflammation [65]. Clinical studies further suggest that better phosphate control is associated with slower CKD progression and reduced cardiovascular complications [66].

4. Advanced Glycation End Products (AGEs)

Advanced glycation end products (AGEs) are formed endogenously through oxidative stress and hyperglycemia and exogenously during high-temperature cooking methods such as frying and

grilling [67]. In CKD and NS, impaired renal clearance combined with increased oxidative stress leads to AGE accumulation, which contributes to chronic inflammation, endothelial dysfunction, vascular stiffness, and progressive renal injury [67,68].

AGEs exert their pathogenic effects primarily through binding to the receptor for AGEs (RAGE), activating NF-κB signaling and increasing inflammatory cytokine release [67]. AGE accumulation has been linked to podocyte injury, oxidative stress, cardiovascular disease, and increased mortality in CKD patients [68]. Soluble RAGE (sRAGE) may function as a protective decoy receptor, and the AGE/sRAGE ratio has been proposed as a potential biomarker for CKD progression and cardiovascular risk [68]. Reducing dietary AGE exposure through lower-temperature cooking methods may therefore represent a useful anti-inflammatory strategy.

5. Saturated and Trans Fats

High intake of saturated and trans fats promotes systemic inflammation, oxidative stress, dyslipidemia, and renal injury in CKD and NS [69]. High-fat diets (HFDs) induce lipid accumulation in proximal tubular epithelial cells, mitochondrial dysfunction, ROS production, and activation of inflammatory pathways including NF-κB, leading to tubular injury and fibrosis [69,70]. Experimental studies show that HFDs increase serum creatinine, blood urea nitrogen (BUN), inflammatory cytokines, and renal injury biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL) [70]. Structural damage including tubular vacuolization, epithelial shedding, and mitochondrial dysfunction has also been observed in renal tissues exposed to HFDs [70]. In CKD populations, diets rich in saturated and trans fats are associated with dyslipidemia, accelerated eGFR decline, proteinuria, endothelial dysfunction, and increased cardiovascular risk [70].

Discussion

Anti-inflammatory dietary strategies represent promising adjunctive approaches for reducing chronic low-grade inflammation in nephrotic syndrome (NS) and chronic kidney disease (CKD). Among available interventions, omega-3 polyunsaturated fatty acids (PUFAs), dietary fiber/prebiotics, and Mediterranean-style dietary patterns demonstrate the most consistent reductions in inflammatory biomarkers such as CRP and IL-6 across clinical studies [6,27,33]. These interventions target interconnected mechanisms including NF-κB activation, oxidative stress, gut dysbiosis, and uremic toxin generation.

Omega-3 PUFAs have shown beneficial effects on inflammation and proteinuria, particularly in IgA nephropathy and early CKD, likely through production of resolvins and inhibition of pro-inflammatory cytokines [6,13]. Fiber and prebiotic supplementation may reduce gut-derived uremic toxins, especially p-cresyl sulfate (PCS), while enhancing short-chain fatty acid (SCFA) production and improving gut barrier integrity [27,32]. Mediterranean, DASH, and plant-based diets are associated with lower inflammatory burden, improved cardiometabolic profiles, and

slower CKD progression [33–36]. Emerging interventions such as fasting-mimicking diets (FMDs) and synbiotic therapies also show potential benefits through modulation of oxidative stress, podocyte repair, and gut–kidney axis signaling, although current evidence remains limited [39–41]. Restriction of sodium, phosphate, saturated fats, and advanced glycation end products (AGEs) further contributes to lowering inflammation and reducing vascular and renal injury [43,66–68]. Despite encouraging findings, substantial limitations remain. Many studies involve small sample sizes, short intervention periods, heterogeneous CKD populations, and inconsistent dietary protocols, limiting generalizability. Most evidence derives from observational studies or small randomized trials lacking hard renal endpoints such as ESRD progression, cardiovascular events, or mortality. Additional confounding factors including medication use, lifestyle habits, and microbiome variability complicate interpretation of results. Future research should prioritize large multicenter randomized controlled trials using standardized dietary interventions and long-term follow-up. Personalized nutrition approaches integrating genetics, microbiome profiling, and metabolic phenotyping may help optimize anti-inflammatory dietary therapies in NS and CKD. Combined dietary strategies targeting multiple inflammatory pathways simultaneously may ultimately provide the greatest clinical benefit.

Challenges and Future Directions

Current evidence regarding dietary anti-inflammatory interventions in NS and CKD is limited by methodological heterogeneity, small study populations, short durations, and variability in disease stage and etiology. Many studies rely on observational data or self-reported dietary assessments, which introduce recall bias and limit causal inference. Randomized controlled trials are often underpowered and focus primarily on surrogate biomarkers rather than clinically meaningful renal outcomes. Variability in patient populations—including differences in CKD stage, nephrotic syndrome subtype, age, comorbidities, and concurrent therapies—further complicates interpretation of results. Additionally, lack of standardized dietary protocols, nutrient formulations, and adherence monitoring contributes to inconsistent findings across studies. Future investigations should focus on adequately powered, long-term multicenter randomized trials evaluating hard outcomes such as eGFR decline, ESRD progression, cardiovascular events, hospitalization, and mortality. Standardized anti-inflammatory dietary models, including modified Mediterranean or plant-forward renal diets, should be evaluated alongside biomarkers of inflammation, oxidative stress, and gut microbiota composition. Integration of multidisciplinary teams involving nephrologists, dietitians, and researchers will also be essential for translating nutritional interventions into clinical practice.

Conclusion

Inflammation plays a central role in the progression of nephrotic syndrome (NS) and chronic kidney disease (CKD) through mechanisms involving oxidative stress, proteinuria, endothelial dysfunction, gut dysbiosis, and uremic toxin accumulation. Dietary strategies targeting these

inflammatory pathways may provide important adjunctive benefits alongside conventional pharmacological therapies. Anti-inflammatory nutritional approaches including omega-3 PUFAs, dietary fiber and prebiotics, Mediterranean and plant-based dietary patterns, sodium and phosphate restriction, low-AGE cooking methods, and selected micronutrient supplementation have demonstrated modest but clinically relevant improvements in inflammatory biomarkers, proteinuria, oxidative stress, and renal function parameters. These interventions may reduce uremic toxin production, improve gut barrier integrity, attenuate systemic inflammation, and slow CKD progression. However, current evidence remains limited by study heterogeneity and lack of large long-term randomized controlled trials. Future precision nutrition approaches integrating dietary patterns, microbiome modulation, and individualized metabolic profiling may improve the effectiveness of anti-inflammatory nutritional therapy in NS and CKD. Overall, dietary management based on whole anti-inflammatory dietary patterns rather than isolated nutrients appears to offer the most promising strategy for improving renal and cardiovascular outcomes in these populations.

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