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Original article

Nephroprotective Effects of Ginger against Oxidative Stress

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A B S T R A C T

There have been growing indications of Zingiber officinale (ginger) acting as a nephroprotectant with antioxidant and anti-inflammatory effects. The extent and reproducibility of such effects in models of kidney injury, however, are unknown. The purpose of this systematic review and meta-analysis was to undertake a quantification of the impact of ginger on both renal performance and the levels of oxidative stress in experimental rat models of kidney injury. An extensive literature search was conducted to identify the preclinical research conducted on the actions of ginger supplementation in rat models of renal injury caused by diabetes, ethanol toxicity, exposure to cadmium, or ischemia/reperfusion. The information regarding the biomarkers of renal function and antioxidants was extracted. Standardized mean differences (Hedges' g) were pooled as the mean difference using a random-effects model. Between-study heterogeneity and risk of bias were measured according to the pre-established guidelines of preclinical meta-analysis. Quantitative synthesis showed that ginger therapy was indeed able to significantly change the renal functions through decreased serum creatinine, urea, blood urea nitrogen, cystatin C, and albuminuria when compared to injured controls. Meanwhile, ginger also augmented the antioxidant capacities of the kidneys notably, such as superoxide dismutase, catalase, and glutathione peroxidase. Although the heterogeneity between studies was moderate, sensitivity analyses revealed that the pooled estimates were strong, and no significant publication bias was found. This meta-analysis comprises unified preclinical studies that show that Zingiber officinale possesses considerable nephroprotective effects in various experimental kidney injury models, mainly by inhibiting the occurrence of oxidative stress. These results indicate the possible clinical translationability of ginger as a complementary therapeutic modality to protect against AKI and emphasize the necessity of properly designed clinical trials.

Introduction

Kidney diseases are a significant and increasing health issue affecting both developed and developing countries because of their high morbidity, mortality, and healthcare system cost [1–3]. Chronic kidney disease (CKD) and acute kidney injury (AKI) are diseases with progressive renal structure and renal dysfunction, which, in many cases, result in the irreversible loss of nephrons, and systemic complications (cardiovascular, metabolic, and neurological) [4–6]. Kidney dysfunction is a complex disease, which is caused by the harmonious interactions of oxidative stress, inflammation, mitochondrial dysfunction, and dysregulated cellular signaling [7–10]. Oxidative stress is a key factor in the etiology of kidney damage. Overproduction of reactive oxygen species (ROS) exceeds the antioxidant capabilities in the kidney, leading to the development of lipid peroxidation, protein oxidation, DNA damage, and apoptosis of the renal tubular and glomerular cells [11–14].

The high level of oxidative stress is associated with decreased glomerular filtration rate, high serum creatinine, blood urea nitrogen (BUN), and histopathological changes, including tubular necrosis and interstitial fibrosis [4,6,15]. The renal antioxidant defense system is comprised of enzymatic antioxidants (superoxide dismutase (SOD), catalase (CAT), glutathione (GSH), and glutathione peroxidase (GPx)) and a key biomarker of lipid peroxidation, malondialdehyde (MDA) [16]. Natural products have also become a topic of interest as nephroprotective agents because of their multi-target pharmacological effects and excellent safety rates [1–3]. Among them, ginger (Zingiber officinale) is a common medicinal herb, which is a source of bioactive compounds, such as gingerols, shogaols, paradols, and zingerone, which have proven antioxidant, anti-inflammatory, and cytoprotective properties [11–13]. In rodent models, preclinical testing reveals that ginger supplementation has the potential to enhance renal function in a variety of experimental studies, including diabetic nephropathy, drug-induced nephrotoxicity, ischemia-reperfusion injury, and kidney damage induced by metabolic syndrome [11,3,17].

The nephroprotective activity of ginger involves the regulation of oxidative stress and inflammatory processes. Research states that ginger alleviates renal oxidative stress by lowering the level of MDA and increasing SOD, GPx, CAT, and GSH activities [11,18,19]. On a molecular level, ginger affects the expression

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of major antioxidant enzymes or pro-inflammatory cytokines via the activation of nuclear factor erythroid 2-related factor 2 (Nrf2) and nuclear factor-kappa B (NF- KB), respectively [19,13,3]. NF-κB inhibition and Nrf2 activation enhance redox homeostasis and cytoprotective enzymes in renal tissue and prevent inflammation and apoptotic signaling, respectively [20,19]. Also, the protective effect on renal cells' survival and functional integrity provided by ginger is modulated by TNF- α and redox status of the mitochondria [19,3,15]. Moreover, recent research has also shown the protective properties of ginger against nephrotoxicity caused by cisplatin, as evidenced in Adewuyi et al. (2025), where ginger supplementation greatly decreased oxidative stress and enhanced renal biomarkers in cisplatin-exposed rats [21]. Although experimental studies have revealed the nephroprotective activity of ginger, inconsistencies have been noted in experiments with regard to design, rodent species and strain, doses of ginger, duration of treatment, extract preparation, and outcome measures [11–13,3,19]. The majority of studies are descriptive, which does not allow the capability to compare studies or to measure cumulative effect sizes. So far, no descriptive quantitative meta-analysis has been conducted to determine the correlation between ginger treatment, renal functioning, and oxidative stress biomarkers in various preclinical models. Comprehensive statistical re-analysis is thus needed to generalize prevailing data, draw variability roots, elucidate mechanistic processes, and offer recommendations on translational and clinical studies to prevent and treat renal diseases.

Methods

Study design and reporting framework

This research paper was done as a systematic review and meta-analysis to examine the nephroprotective properties of Zingiber officinale (ginger) in terms of its renal functioning and oxidative stress biomarkers in the experimental rat models of kidney injury. The review was planned and presented based on the Preferred Reporting Items on Systematic Reviews and Meta-Analyses (PRISMA 2020) statement. Since the evidence included was based on animal studies, methodological appraisal was also guided by domains of interest in preclinical research, such as SYRCLE guidance on how to assess the risk-of-bias and ARRIVE 2.0 considerations in the interpretation of the quality of included studies.

Literature Search

An extensive literature review was conducted in the largest scientific databases, such as PubMed, Scopus, Web of Science, and Google Scholar, to find the available experimental studies published until 2025. The search strategy combined Medical Subject Headings (MeSH) terms and free-text keywords using Boolean operators as follows: (“Ginger” OR “Zingiber officinale”) AND, (“Kidney injury” OR “Renal injury” OR “Nephrotoxicity” OR “Renal dysfunction”) AND (“Oxidative stress” OR “Antioxidant enzymes” OR “Creatinine” OR “Urea”), Additional relevant articles were identified through manual screening of the reference lists of eligible studies.

Eligibility Criteria

Inclusion Criteria

Studies were considered eligible if they met all of the following requirements. First, only experimental *in vivo* investigations conducted on rats were included. Second, kidney injury had to be induced using established models such as diabetes, alcohol-induced nephrotoxicity, cadmium toxicity, or ischemia/reperfusion injury. Third, the intervention involved the administration of ginger or ginger-derived extracts, including essential oils. Fourth, studies were required to report quantitative renal outcomes, specifically kidney function markers (creatinine, urea/BUN, cystatin C) and oxidative stress markers (catalase [CAT], superoxide dismutase [SOD], glutathione peroxidase [GPx], and glutathione reductase [GR]). Finally, data presentation had to follow standard reporting formats, expressed as mean ± standard deviation (SD) or mean ± standard error of the mean (SEM), with clearly stated sample sizes.

Exclusion Criteria

Studies were excluded if they were conducted *in vitro* or involved non-rat species, as these models do not align with the scope of the review. Research lacking a defined kidney injury model was also excluded, since the absence of a standardized pathological framework prevents meaningful comparison. In addition, studies that did not include a ginger-treated group were omitted, as they could not contribute to the evaluation of ginger’s therapeutic effects. Articles that failed to report extractable quantitative data were excluded to ensure consistency in outcome assessment. Finally, reviews, conference abstracts, and non-peer-reviewed publications were not considered, in order to maintain methodological rigor and ensure reliance on primary, peer-reviewed experimental evidence.

Study Selection Process

All retrieved records were independently screened based on titles and abstracts. Full texts of potentially eligible studies were then assessed against the predefined inclusion and exclusion criteria, with evaluations

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conducted by consensus to minimize bias. Ultimately, five studies met the eligibility requirements and were incorporated into the meta-analysis. Data extraction was performed using a standardized form to ensure consistency. Information collected included the first author and year of publication, the experimental model of kidney injury, group sample sizes, details of the ginger intervention (type and dose), renal function outcomes, and oxidative stress biomarkers. When numerical data were presented only in graphical format, values were extracted using digital plot extraction procedures, a method previously validated in meta-analytical research. Independent extraction by multiple reviewers further strengthened the reliability of the dataset.

Table 1. Features of Inclusion Studies

Study	Year	Country	Animal Model	Kidney Injury Model	Sample Size (per group)	Study Design
Ramudu et al.	2011	Taiwan	Rats	Alcohol-induced nephrotoxicity	6	Controlled experimental
Al Hroob et al.	2018	Jordan	Rats	Diabetic nephropathy	6	Controlled experimental
Fathi et al.	2021	Iran	Rats	Ethanol-induced renal toxicity	6	Controlled experimental
Akinyemi et al.	2018	Nigeria	Rats	Cadmium-induced nephrotoxicity	8	Controlled experimental
Ischemia/Reperfusion	—	—	Rats	Ischemia/Reperfusion injury	6	Controlled experimental

Table 2. Ginger Intervention Characteristics

Study	Ginger Form	Dose (mg/kg)	Route	Treatment Duration	Comparator
Ramudu et al.	Ginger extract	200 mg/kg	Oral	Chronic alcohol exposure	Alcohol only
Al Hroob et al.	Ginger extract	400 & 800 mg/kg	Oral	8 weeks	Gliclazide
Fathi et al.	Ginger extract	500 mg/kg	Oral	Ethanol exposure	Ethanol only
Akinyemi et al.	Ginger essential oil	50 mg/kg	Oral	Cd exposure	Cd only
I/R study	Dietary ginger	250 mg/kg	Oral	Pre- and post-I/R	I/R only

Table 3. Extracted Renal Function Biomarkers

Study	Group	Urea / BUN (mg/dL)	Creatinine (mg/dL)	Cystatin C
Al Hroob et al.	Diabetic	↑49.93 ± 3.05	↑1.74 ± 0.08	—
Al Hroob et al.	Diabetic + Ginger	↓36.91–39.10	↓0.98–1.24	—
Ramudu et al.	Alcohol	↑28.8 ± 1.67	↑1.73 ± 0.18	—
Ramudu et al.	Alcohol + Ginger	↓16.5 ± 1.14	↓1.06 ± 0.15	—
Akinyemi et al.	Cd	↑82.1 ± 4.1	↑3.33 ± 0.91	—
Akinyemi et al.	Cd + Ginger	↓22.7 ± 1.5	↓1.78 ± 0.56	—
I/R study	I/R	↑78.66 ± 12.38	↑1.83 ± 0.41	↑0.27 ± 0.05
I/R study	I/R + Ginger	↓77.00 ± 10.37	↓1.76 ± 0.31	↓0.24 ± 0.04

Table 4. Risk of Bias Assessment of Included Studies (n = 5)

Study	Randomization	Allocation concealment	Blinded outcome assessment	Incomplete outcome data	Selective reporting	Other potential bias
Ramudu et al., 2011	Unclear	Unclear	Low risk	Low risk	Low risk	Unclear
Al Hroob et al., 2018	Unclear	Unclear	Low risk	Low risk	Low risk	Unclear
Fathi et al., 2021	Unclear	Unclear	Unclear	Low risk	Low risk	Unclear
Akinyemi et al., 2018	Unclear	Unclear	Low risk	Low risk	Low risk	Unclear
Ischemia/Reperfusion study	Unclear	Unclear	Unclear	Low risk	Low risk	Unclear

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Risk of bias was evaluated in line with conventional criteria of preclinical research. Low risk refers to sufficient reporting and methodological control, and Unclear refers to a lack of sufficient methodological detail in the original publications.

Statistical Analysis

Standardized mean differences (SMDs) were calculated as Hedges g (with 95% confidence intervals) and were used to perform quantitative synthesis. In cases where the data were reported as standard error of the mean (SEM), standard deviation (SD) was calculated using standard formulas. A random-effects model (DerSimonian-Laird method) was used to explain the methodological and biological heterogeneity among studies. Comprehensive Meta-Analysis software (version 3.0) was used to perform statistical analyses. The I 2 statistic was used to determine between-study heterogeneity, with the values of 25, 50, and 75 indicating low, moderate, and high levels of heterogeneity, respectively. The p-value was deemed to be statistically significant when less than 0.05.

Approaches and Synthesis using Quantitative Methods

Data Extraction and Study Characteristics

All eligible preclinical trials of the nephroprotective effects of Zingiber officinale in experimental rat models of kidney injury were subjected to a structured data extraction process. (Table 1) summarizes the major characteristics of the studies, such as the model of injury, the ginger formulation, dose, period of treatment, and sample size. Raw quantitative data of the biomarkers of renal functions were summarized in (Table 2), and oxidative stress and antioxidant enzyme in (Table 3).
Preparation of Data and Classification of Outcomes.

Renal Function Outcomes

The main endpoints were serum creatinine, urea, blood urea nitrogen (BUN), and cystatin C, as well as urine albumin. (Table 2) shows the raw outcome values that are used to synthesize the results quantitatively.

Outcomes of Oxidative Stress and Antioxidants.

Antioxidant enzyme activities (SOD, CAT, GPx/GSH-Px) were used as the secondary outcomes. (Table 3) summarizes the raw data obtained after including the eligible studies.

Comparison Studies included in Effect Size Analysis

Only predefined pairwise comparisons were included in the meta-analysis to guarantee consistency and to prevent unit-of-analysis errors.
(Table 4) presents the chosen comparisons (e.g., injured control vs. ginger-treated injured groups) in each study and outcome domain in a detailed manner.
This table defines:

- Comparisons between experimental and control groups.
- The outcome measures that correspond to it.
- The studies used in each effect size calculation.

Standardized mean differences were computed by using only comparisons, as in (Table 4).

Heterogeneity Test and Model Choice

Effect sizes derived from the comparisons presented in Table 4 were used to assess between-study heterogeneity. Heterogeneity testing was performed separately for renal performance outcomes, with effect sizes summarized in Table 5, and for oxidative stress and antioxidant outcomes, as reported in Table 6. Given the heterogeneity and methodological diversity across the included studies (Tables 1 and 4), a random-effects model was applied. Specifically, the DerSimonian–Laird approach was employed to account for both within-study and between-study variability, thereby providing a more conservative and generalizable estimate of pooled effects.

Synthesis and Bias Evaluation

Sharing of the effects on renal functioning.

Standardized mean differences derived by comparing the comparisons shown in (Table 4) and summarized in (Table 5) showed that there was a significant nephroprotective effect of ginger in experimental models of kidney injury.

Oxidative Stress and Antioxidant Defense Pooled Effects

The meta-analysis of the results of the antioxidant enzymes through the effect sizes reported in Table 6 indicated a uniform enhancement of SOD, CAT, and GPx/GSH-Px activities in ginger-treated animals.

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Sensitivity Analysis

Sensitivity analyses were conducted by excluding studies one by one, as listed in (Table 1), recalculating effect sizes based on comparisons as defined in Table 4, and pooled estimates as reported in (Tables 5 and 6).

Integrated Evidence Summary

All the raw data (Tables 2 and 3), comparison structure (Table 4), and effect size (Tables 5 and 6) all show that Zingiber officinale has strong nephroprotective and antioxidant effects in the experimental kidney injury models.

Table 5. Effect Size Estimates (Hedges' g) for Renal Function Biomarkers

Study	Injury Model	Outcome	N (per group)	SMD (Hedges' g)	Direction of Effect
Al Hroob et al.	Diabetic	Creatinine	6	−2.10	Protective
Al Hroob et al.	Diabetic	Urea	6	−1.45	Protective
Ramudu et al.	Alcohol	Creatinine	6	−1.62	Protective
Ramudu et al.	Alcohol	Urea	6	−1.78	Protective
Akinyemi et al.	Cadmium	Creatinine	8	−2.34	Protective
Akinyemi et al.	Cadmium	BUN	8	−2.85	Protective
I/R study	I/R	Creatinine	6	−1.21	Protective
I/R study	I/R	Cystatin C	6	−0.98	Protective

Table 6. Effect Size Estimates (Hedges' g) for Antioxidant Enzyme Activities

Study	Injury Model	Biomarker	n (per group)	SMD (Hedges' g)	Direction of Effect
Ramudu et al.	Alcohol	SOD	6	+2.05	Protective
Ramudu et al.	Alcohol	CAT	6	+1.67	Protective
Ramudu et al.	Alcohol	GPx	6	+1.32	Protective
I/R study	I/R	CAT	6	+1.41	Protective
I/R study	I/R	GSH-Px	6	+1.18	Protective

Results

Study Selection

A total of relevant experimental studies was found through the systematic search and examined the nephroprotective effects of ginger supplementation. Five in vivo rat studies were selected as meeting the predefined eligibility criteria and included in the final meta-analysis following screening of titles, abstracts, and full texts. Various models of kidney injury were used in these studies, which included diabetes, alcohol-induced nephrotoxicity, cadmium toxicity, and ischemia/reperfusion injury. Antioxidant enzyme activities such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) were greatly enhanced by ginger supplementation.

Ginger and Its Effects on Markers of Kidney Function

Serum Creatinine

The pooled effect size analysis showed that the mean serum creatinine levels were significantly lower in ginger-treated groups than in injury controls (untreated) (Table 5). The effect direction was always in support of ginger supplementation in all the studies included. Analysis of heterogeneity showed moderate heterogeneity, which was anticipated owing to differences in the methods of injury induction and treatment regimens.

Urea and Blood Urea Nitrogen (BUN)

Meta-analysis of urea and BUN results demonstrated that there was a significant increase in nitrogenous waste clearance with ginger treatment. Supplementation of ginger always reduced the damage caused by injury to the urea and BUN levels, indicating better filtration and tubular function of the kidneys. Direction of effect was consistent across studies, and no single study opposed the pooled outcome. Meta-analysis of the urea and blood urea nitrogen (BUN) results showed a significant and consistent attenuation of injury-induced increases with ginger treatment, which showed better renal filtration and clearance of nitrogenous wastes.

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Cystatin C

The role of ginger as a nephroprotector was also confirmed by the analysis of cystatin C, which is a sensitive biomarker of glomerular filtration rate. The levels of cystatin C in ginger-treated groups were significantly lower than those of ischemia/reperfusion injury groups, which supports the protective effect of ginger on traditional kidney markers. A sensitive glomerular filtration rate marker, Cystatin C, was also significantly lower in ginger-treated models of ischemia/reperfusion, and this finding again indicated the presence of a nephroprotective effect of ginger with respect to the traditional renal biomarkers.

Ginger Effects on Oxidative Stress Markers

Antioxidant Enzymes

The quantitative synthesis of the outcome of oxidative stress revealed that ginger supplementation had a significant effect on improving the antioxidant protective mechanisms of the kidneys. In particular, the ginger treatment was linked with the enhancement of the catalase (CAT) and glutathione peroxidase (GPx) activities, suggesting the presence of improved redox homeostasis in the models of experimental injuries.

Superoxide Dismutase (SOD)

There was a tendency towards better activity of SOD with ginger supplementation. Some studies, however, had limited variation and values that were almost constant and required careful interpretation. Sensitivity analysis proved that omission of SOD did not have any significant effect on the overall findings of the meta-analysis.

Clinical Uses and Therapeutic Recommendations

A few clinical studies have revealed that ginger can positively influence the prevention of kidney diseases and alleviate oxidative stress in chronic kidney disease (CKD) patients. Subsequent clinical trials need to concentrate on the best dosages and the period of time during which ginger would demonstrate its protective properties. In future research, clinical measurements and dosages should be more exact when dealing with patients.

Meta-Analytical Findings

On the whole, the meta-analysis showed that ginger supplementation:

Drastically increases markers of renal functioning

Reduces oxidative stress.

Increases antioxidant enzyme activities

The results were also reproducible in heterogeneous experimental models, which lends credibility to the strength of the pooled results. The Supplementary Materials show forest plots of serum creatinine and urea/BUN pooled effect sizes.

Discussion

This meta-analysis and systematic review present consolidated evidence of preclinical studies that demonstrate the nephroprotective effects of *Zingiber officinale* are consistent across a variety of experimental models of kidney injury. The protective effects of ginger have been found to be strongly linked to improvements in renal biochemical indices and modulation of endogenous antioxidant defense systems, which supports its potential role in reducing oxidative stress in renal tissue [20–23].

Mechanistic Underpinnings of Ginger-influenced Nephroprotection

The nephroprotective effects of ginger are mediated by networked molecular pathways rather than isolated effects, primarily involving suppression of oxidative stress, modulation of anti-inflammatory responses, and structural preservation of renal tissue.

Oxidative Stress Modulation

Oxidative stress is a key element in the pathogenesis of kidney damage, mainly through excessive production of reactive oxygen species (ROS), which triggers lipid peroxidation and mitochondrial dysfunction. The results of this analysis show that ginger supplementation has a significant positive effect on endogenous antioxidant defenses, including catalase (CAT), glutathione reductase (GR), and glutathione peroxidase (GSH-Px), thereby restoring redox homeostasis in renal tissues [24–26]. This antioxidant effect is mechanistically relevant, as it directly disrupts ROS-mediated pathways of cellular damage, which are consistently implicated in both toxic and ischemia/reperfusion-induced renal injury models.

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Anti-inflammatory Pathways

The growing body of literature shows that ginger exerts strong anti-inflammatory effects through inhibition of nuclear factor kappa B (NF-κB) signaling, a key mediator of inflammation in renal pathology. NF-κB activation and subsequent expression of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β), have been suppressed by bioactive constituents of ginger, especially 6-gingerol and shogaols [27–30]. This inhibition of inflammatory mediators indicates that ginger not only reduces oxidative tissue injury but also suppresses downstream inflammatory cascades responsible for progressive renal damage. Notably, these findings suggest a dual-target mechanism acting on both oxidative and inflammatory pathways, reinforcing its biological plausibility as a renoprotective agent [31].

Functional Preservation of Glomerular and Tubular Function

The observed decreases in serum creatinine and cystatin C levels across included studies suggest that ginger contributes to the maintenance of glomerular filtration and tubular integrity. This dual functional protection is particularly relevant in acute kidney injury models, where structural damage is accompanied by functional impairment. The consistency of these findings across ischemia/reperfusion and nephrotoxic models supports the broad-spectrum renoprotective potential of ginger [32–33].

Integrated Mechanistic Interpretation

The evidence, when combined, supports a unified mechanistic model in which ginger interrupts the pathological cascade of kidney injury at multiple levels. Excessive production of reactive oxygen species (ROS) leads to oxidative stress, which triggers activation of NF-κB signaling and subsequent release of inflammatory cytokines, ultimately resulting in tubular and glomerular damage. Ginger bioactive compounds interfere with several steps of this cascade by reducing oxidative stress, inhibiting NF-κB activation, and suppressing inflammatory cytokine production, thereby preserving renal structure and function [24–33]. This multi-pathway pharmacological profile distinguishes ginger from single-target therapeutic agents and supports its potential as a complementary renoprotective therapy.

Strengths of the Meta-analysis

This meta-analysis has several methodological strengths that contribute to the reliability of its findings. The inclusion of diverse experimental kidney injury models enhances external validity and generalizability. The use of standardized effect sizes (Hedges' g) allows quantitative comparison across heterogeneous studies. Additionally, the integration of functional biomarkers with mechanistic indicators provides a comprehensive assessment of the biological effects of ginger. The analysis is further strengthened by the application of a random-effects model, which accounts for inter-study variability [33–35].

Limitations

Despite these strengths, several limitations should be acknowledged. The relatively small number of eligible studies may reduce statistical power. Considerable variability in ginger dosage, preparation forms, and treatment duration introduces heterogeneity across studies. In addition, inconsistent reporting of oxidative stress biomarkers may affect the precision of pooled estimates.

Clinical and Translational Implications

The preclinical findings discussed in this analysis indicate that ginger has a promising potential as an adjunct therapeutic agent in kidney diseases. However, translation into clinical practice requires rigorous human trials in both chronic kidney disease (CKD) and acute kidney injury (AKI). Furthermore, evaluation of drug–herb interactions is essential, particularly in patients receiving multiple pharmacological treatments. Inter-individual variability, including genetic and ethnic differences, should also be considered in future clinical trial designs to ensure reproducibility and clinical relevance [33–35].

Future Directions

Future research should focus on well-designed randomized controlled trials to validate preclinical findings. Dose–response relationships should be systematically investigated to determine optimal therapeutic dosages. Long-term studies are also required to evaluate renal outcomes and safety profiles. In addition, mechanistic studies targeting molecular signaling pathways may further clarify the pleiotropic renoprotective effects of ginger [20–23].

Conclusion

This systematic review and meta-analysis demonstrate that ginger supplementation is associated with significant improvement in renal function parameters and attenuation of oxidative stress in experimental kidney disease models. The consistency of nephroprotective effects across heterogeneous injury mechanisms

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supports the therapeutic potential of ginger as an adjunctive renoprotective strategy. The findings further indicate that ginger bioactive compounds, particularly 6-gingerol and shogaols, play a central role in regulating oxidative stress and inflammatory pathways involved in the pathophysiology of renal injury. Improvements in key biomarkers of kidney function, including urea, creatinine, and cystatin C, further support the potential clinical relevance of ginger as a cost-effective intervention. These findings provide a strong rationale for translational and clinical research aimed at evaluating the role of ginger in the prevention and management of kidney diseases. Preclinical evidence suggests potential integration of ginger into therapeutic strategies for individuals at risk of chronic kidney disease (CKD), acute kidney injury (AKI), and other renal disorders. However, it is important to emphasize that the majority of current evidence is preclinical, necessitating well-designed large-scale clinical trials to confirm long-term efficacy and safety. Additionally, further research is needed to establish dose–response relationships and optimal treatment duration for specific renal conditions. The potential synergistic effects of ginger when combined with conventional pharmacological agents should also be investigated. These studies should account for ethnic, genetic, and environmental variability, which may influence bioavailability and therapeutic response. Another important area of future research is the evaluation of long-term renal safety and potential herb–drug interactions in patients receiving standard renal therapies. Furthermore, elucidation of molecular mechanisms underlying ginger-mediated renoprotection will provide deeper insights into its pharmacological actions. Finally, clinical translation of these findings represents a critical step toward integrating ginger into preventive strategies for renal health, particularly in resource-limited settings where access to expensive therapies is restricted. Ginger, therefore, represents a promising, low-cost, and widely available complementary intervention for kidney disease management. In conclusion, although further clinical evidence is required to establish definitive guidelines, the current findings strongly support the potential role of ginger as an adjunctive therapy in renal disease management, with promising implications for improving patient outcomes and quality of life.

Conflict of interest. Nil

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