Research Article

Effect of *Eruca sativa* Leaf Extract on Serum Cystatin C Levels and Renal Function Markers in Experimental Rats

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Abstract | Nephrotoxicity is a significant health concern, primarily driven by oxidative stress and inflammation, which disrupt renal structure and impair kidney function. Cystatin C has emerged as a superior biomarker for early detection of renal dysfunction compared to traditional markers like urea and creatinine. *Eruca sativa* (commonly known as rocket) is a medicinal-plant known for its anti-oxidant and anti-inflammatory properties. This study aimed to evaluate the effect of *E. sativa* leaf extract on serum cystatin C and conventional renal function markers in a rat model of ethanol-induced nephrotoxicity. Thirty male Wistar rats were divided into five groups: normal-control, ethanol-only, and three treatment groups receiving ethanol plus *E. sativa* extract at 250, 375, and 500 mg/kg/day, respectively, for 30 days. Serum levels of cystatin C, urea, and creatinine were measured and statistical-analysis was conducted using ANOVA followed by Tukey's test. Ethanol administration significantly elevated serum cystatin C (1.25 ± 0.12 mg/L), urea (12.8 ± 1.2 mmol/L) and creatinine (68.7 ± 5.4 µmol/L) compared to controls ($p \leq 0.05$). Treatment with *E. sativa* extract produced dose-dependent reductions in all biomarkers, with near-normalization at 500 mg/kg: cystatin C (0.58 ± 0.06 mg/L), urea (6.1 ± 0.5 mmol/L), and creatinine (37.1 ± 3.4 µmol/L) ($p \leq 0.05$ vs. ethanol group). The protective effects are likely attributed to the plant's phytochemicals that mitigate oxidative-stress and inflammatory-responses. *E. sativa* leaf extract significantly ameliorates ethanol-induced renal injury in rats, as evidenced by improved renal biomarkers particularly cystatin C. These results show its potential role as a natural nephroprotective agent by decreasing level of serum cystatin C, urea and creatinine. This effect may also be relevant to other animal species, such as companion animals, that develop nephrotoxicity due to drugs, environmental toxins, heavy metals, and other factors.

Keywords | Cystatin C, *E. sativa*, Phytotherapy, Nephrotoxicity, Renal injury

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INTRODUCTION

The kidneys serve as essential regulators of systemic homeostasis which performing critical roles in fluid and electrolyte balance, waste product excretion, acid–base regulation and endocrine-signaling (Crintea *et al*., 2025). These vital organs are particularly susceptible to damage from a range of toxic-agents including xenobiotics, pharmaceuticals and environmental compounds (Iovdijová and Bencko, 2010). In companion animals, kidney toxicity can result from various causes such as drugs, environmental toxins, and other harmful substances, and may ultimately lead to death. In humans, ethanol is a widely consumed substance known to induce systemic toxicity when ingested in high quantities (Le Dare *et al*., 2019). Excessive ethanol exposure can lead to both acute and chronic renal impairment primarily through mechanisms involving oxidative-stress, inflammation and direct cytotoxic effects on renal tissues (Rodrigo and Rivera, 2002). Ethanol metabolism generates excessive reactive oxygen species

(ROS) which disrupt the cellular redox state and overwhelm intrinsic antioxidant defenses (Contreras-Zentella *et al.*, 2022). This oxidative burden coupled with ethanol-induced upregulation of pro-inflammatory cytokines contributes to functional deterioration and structural damage in nephron units particularly in glomerular and tubular-components (Yang *et al.*, 2022).

Early diagnosis of renal-dysfunction is essential for preventing the progression of irreversible kidney damage (Locatelli *et al.*, 2002). Traditionally, serum creatinine and urea are employed to assess renal-function. However, these conventional markers are influenced by numerous confounding variables such as age, body mass, dietary protein intake and hydration-status (Al-Ziaydi *et al.*, 2020). These limitations hinder their sensitivity, especially in detecting mild or subclinical renal impairment. In recent years, cystatin C has emerged as a more accurate and sensitive biomarker for assessing glomerular-filtration-rate (GFR). Cystatin C is a low-molecular-weight cysteine protease inhibitor produced consistently by all nucleated cells. It is freely filtered by the glomeruli and almost completely reabsorbed and degraded in the renal tubules without significant-secretion (Onopiuk *et al.*, 2015). Significantly, its serum concentration is unaffected by age, sex, muscle mass or diet making it a more reliable index of GFR than creatinine (Rajagopalan *et al.*, 2016). Elevated serum cystatin C levels are indicative of early-stage renal-dysfunction and have also been linked to systemic-inflammatory-responses, cardiovascular-pathology and metabolic abnormalities (Tanwar *et al.*, 2021). Therefore, cystatin C holds promise not only as a renal function biomarker but also as a broader indicator of systemic stress and disease.

Numerous medicinal plants are known to possess potent antioxidant and anti-inflammatory properties which may contribute to cellular protection in organ systems vulnerable to oxidative and inflammatory-damage (Ondua *et al.*, 2019). *E. sativa*v generally referred to as (rocket or arugula) is one such plant that has attracted considerable attention. It belongs to the *Brassicaceae* family and has long been consumed for both culinary and traditional medicinal purposes (Barazani and Ziffer-Berger, 2014). Rich in a diverse array of phytochemicals including glucosinolates, isothiocyanates, flavonoids, phenolic acids, vitamins and minerals. *E. sativa* exhibits a wide spectrum of pharmacological activities (Jin *et al.*, 2009). These include antioxidative, anti-inflammatory, antimicrobial, hepatoprotective, cardioprotective and anticancer effects (Duru *et al.*, 2022).

The antioxidative properties of *E. sativa* are primarily attributed to its capacity to scavenge ROS enhance endogenous-antioxidant enzymes such as superoxide dismutase and glutathione-peroxidase and reduce lipid peroxidation (Piragine, 2020). Additionally, the plant exerts anti-inflammatory effects through downregulation of key proinflammatory mediators, including tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6) (Ochar *et al.*, 2024). Its isothiocyanate content particularly erucin derived from glucoerucin which is believed to modulate nuclear factor kappa B (NF-κB) signaling and other molecular pathways involved in inflammation (Genah *et al.*, 2024). These pharmacodynamic properties suggest a promising role for *E. sativa* in counteracting ethanol-induced toxicity which is known to be mediated by similar oxidative and inflammatory mechanisms (Vicidomini *et al.*, 2024).

Ethanol-induced kidney injury is typically characterized by structural alterations in the renal cortex and medulla including glomerular atrophy, tubular epithelial degeneration and interstitial infiltration by inflammatory cells (Rikalo and Romanenko, 2018). These histopathological features are accompanied by biochemical derangements, including elevated serum urea and creatinine, as well as increased levels of oxidative stress biomarkers such as malondialdehyde (Dinu *et al.*, 2006). The depletion of intracellular antioxidants further exacerbates renal damage (Dennis and Witting, 2017). In this context, therapeutic agents capable of restoring antioxidant balance and attenuating inflammation may preserve renal architecture and function (Lee *et al.*, 2024). *E. sativa* due to its phytochemical constituents, is hypothesized to confer such benefits. This study aims to evaluate the effects of *E. sativa* leaf extract on serum cystatin C levels and overall renal function in a rat model of ethanol-induced nephrotoxicity.

MATERIALS AND METHODS

Plant material and extract preparation

Fresh *E. sativa* leaves were procured from a local source and authenticated by a qualified botanist. The leaves were thoroughly rinsed with distilled water then air-dried under shade at "ambient room temperature" for seven days. Subsequently, the dried leaves were pulverized into a fine powder using an electric-grinder. A quantity of 100 grams of the powdered material was subjected to maceration in 70% ethanol at a ratio of 1:10 (w/v) for 72 hours with intermittent agitation. The resulting extract was filtered through muslin cloth followed by Whatman No.1 filter paper (Abed, 2015; Ali *et al.*, 2024). The filtrate was concentrated under reduced pressure at 40°C using a rotary evaporator to yield a semi-solid-extract, which was stored at 4°C in airtight containers until further use.

Experimental animals and design

Thirty male Wistar rats, each weighing between 210 and 275 grams, were obtained and allowed to acclimate for one week under controlled laboratory conditions temperature maintained at 22 ± 2°C, relative humidity at 55 ± 5%, and a 12-hour light/dark cycle. Animals had unrestricted access to standard laboratory chow and water throughout the study. The experimental protocol received prior approval from The experimental protocol received prior approval from the Animal Ethical Research committee, college of dentistry, Al-Iraqia university (Ethical approval Number: ESA and HER- 10-10-07-2025), ensuring compliance with accepted guidelines for the care and use of laboratory animals (Kiani *et al*., 2022).

The ethanol (95% ethyl alcohol, BDH England) used in the current was purchased from local medical laboratory supplies sales office, Baghdad, Iraq. Ethanol was used at concentration of 60% v/v diluted by distilled water. A 5 g/kg/day was the dose of ethanol at estimated volume between 2.3 to 2.9 ml for each rat.

The animals were randomly allocated into five groups of six rats each (n = 6) when group I (Normal-Control) received distilled water orally and served as the untreated control group, group II (Ethanol Control) administered ethanol orally at a dose of 5 g/kg/day to induce renal toxicity according to previously published study (Pruett *et al*., 2020). Group III (Ethanol + *E. sativa* 250 mg/kg) treated with ethanol plus *E. sativa* extract at 250 mg/kg/day. Group IV (Ethanol + *E. sativa* 375 mg/kg) treated with ethanol plus *E. sativa* extract at 375 mg/kg/day. Finally, group V (Ethanol + *E. sativa* 500 mg/kg) was treated with ethanol plus *E. sativa* extract at 500 mg/kg/day.

All interventions were administered orally via gavage once daily for a total duration of 30 days. In the treatment groups, *E. sativa* extract was administered one hour prior to ethanol to assess its potential nephroprotective effects.

Sample collection and biochemical measurements

At the end of the experimental period, animals were fasted overnight and anesthetized with intraperitoneal injections of ketamine (80 mg/kg) and xylazine (10 mg/kg). Blood samples were collected by cardiac-puncture, allowed to clot at room temperature, and then centrifuged at 3000 revolutions per minute for 15 minutes (Abed *et al*., 2023). Serum was carefully separated and stored at –20°C pending biochemical analysis.

Serum cystatin C concentrations were quantified using a species-specific sandwich enzyme-linked immunosorbent assay (ELISA) kit (Shanghai DeBo Biochemistry Laboratory Inc). Then, the absorbance of the samples was measured at 450 nm with ELISA Lab immunoassay reader (Cobrin *et al*., 2013). The results were displayed as µg/ mL Cystain C in the blood. The creatinine serum detection kit was used to estimate the level of creatinine using colorimetric measurement method and according to instruction of the manufacture (StressXpress®). Additionally, the urea was also estimated by colorimetric assay kit (Urease method) (E-BC-K183-S) and measured by spectrophotometer (at wave length of 580 nm) according to the instruction of the manufacture.

Statistical analysis

Data were expressed as mean values with corresponding standard-deviations (mean ± SD). Statistical comparisons among groups were performed using one-way analysis of variance (ANOVA), followed by "Tukey's post hoc test" for multiple comparisons. Differences were considered statistically-significant when *p*-values were less than 0.05 (Abed *et al*., 2022). Statistical-analyses and graphical representations were carried out using GraphPad-Prism software (version 6).

RESULTS AND DISCUSSION

The present results assessed the renoprotective potential of *E. sativa* leaf extract against ethanol-induced nephrotoxicity by evaluating serum concentrations of cystatin C, urea and creatinine. Chronic ethanol administration (5 g/kg/day for 30 days) elicited significant renal-impairment, evidenced by marked elevations in all measured biomarkers compared to the normal control group ($p \leq 0.05$), the results are illustrated in Figures 1-3.

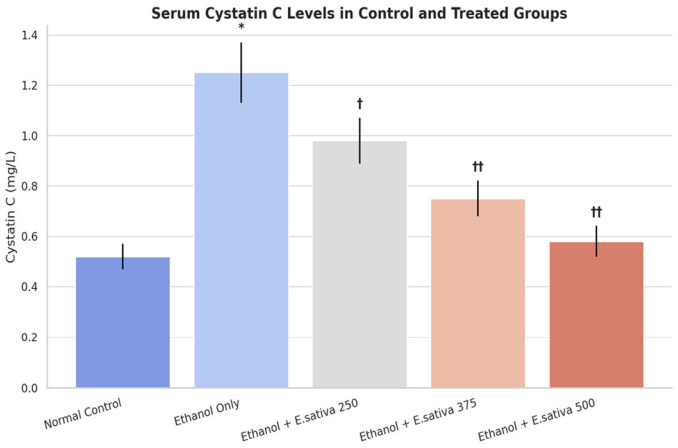

Figure 1: Effect of *E. sativa* extract on serum cystatin C levels in ethanol-treated rats. Values are mean ± standard deviation (SD), n = 6 per group. *Significantly different from Normal Control ($p \leq 0.05$). † Significantly different from Ethanol Only group ($p \leq 0.05$). †† Significantly different from Ethanol Only group with stronger effect ($p \leq 0.05$).

Serum cystatin C, a highly sensitive marker for early renal dysfunction and decline in glomerular filtration rate, increased significantly from 0.52 ± 0.05 mg/L in the control group to 1.25 ± 0.12 mg/L in ethanol-treated rats ($p \leq 0.05$). This substantial increase indicates significant glomerular injury induced by ethanol toxicity, confirming the utility of cystatin C as a reliable early biomarker in nephrotoxicity models. Importantly, treatment with *E. sativa* extract elicited a statistically-significant, dose-dependent reduction in cystatin C levels: 0.98 ± 0.09 mg/L, 0.75 ± 0.07 mg/L, and 0.58 ± 0.06 mg/L at 250, 375, and 500 mg/kg doses, respectively ($p \leq 0.05$ versus ethanol group). The near normalization of cystatin C at the highest dose underscores the extract's potent capacity to mitigate early glomerular damage as in Figure 1. Cystatin C is a low molecular weight protein that produced consistently by nucleated cells. It is filtrated freely at the glomerulus and reabsorbed and catabolized by cell of the proximal tubule (Abrahamson *et al*., 1990). The result of the current study showed that, the obvious decreasing of cystatin C level in groups treated with *E. sative* suggests that the plants components including the flavonoids, glucosinolates, and isothiocyanates have a protection effects and restore the glomerular filtration capacity by ameliorative oxidative and inflammatory insults caused by ethanol metabolism. These results agree with previously published study (Sarwar *et al*., 2007), who approved the antioxidant and protective activities of *E. sative* seeds on the mercuric chloride induced renal toxicity.

Serum urea is an indicator of renal nitrogenous waste clearance, rose significantly from 5.3 ± 0.6 mmol/L in the control group to 12.8 ± 1.2 mmol/L following ethanol exposure ($p \leq 0.05$). Administration of *E. sativa* resulted in significant, dose-dependent decreases in serum urea: 9.5 ± 0.9 mmol/L, 7.2 ± 0.7 mmol/L, and 6.1 ± 0.5 mmol/L at the respective doses ($p \leq 0.05$ vs. ethanol group). These reductions suggest restoration of renal excretory function and amelioration of tubular impairment caused by ethanol (Figure 2). A treatment with *E. sativa* extract led to progressive and dose dependent decreases in urea concentration that approach the normal values at higher doses. The decrease in urea levels suggests an improvement in nitrogen clearance, likely due to the regeneration of tubular structures and enhanced renal perfusion. This finding is consistent with previously published studies (Hannan et al., 2021), which demonstrated the pharmacological activity of *Nigella sativa* and its bioactive constituent, thymoquinone, in protecting against kidney injury.

Serum creatinine, a conventional marker of glomerular filtration, was also significantly elevated by ethanol treatment, increasing from 35.4 ± 3.1 µmol/L in the control group to 68.7 ± 5.4 µmol/L ($p \leq 0.05$). Treatment with *E. sativa* extract yielded significant dose-dependent decreases in creatinine concentration: 52.3 ± 4.7 µmol/L, 42.8 ± 3.9 µmol/L, and 37.1 ± 3.4 µmol/L for 250, 375, and 500 mg/kg doses, respectively ($p \leq 0.05$ compared to ethanol-only group). These results indicate improved glomerular filtration capacity and reduced renal dysfunction (Figure 3). The level of serum creatinine is extensively used in clinical laboratory investigation. However, it is less sensitive to early kidney dysfunction because it elevated only after substantial nephron loss (Ávila *et al*., 2025). These results are compatible with previously published study by (Boukarine *et al*., 2023), who approved the antioxidant therapeutic effects of *E. sativa* against hepato-renal toxicity caused by xylene in Wistar rats.

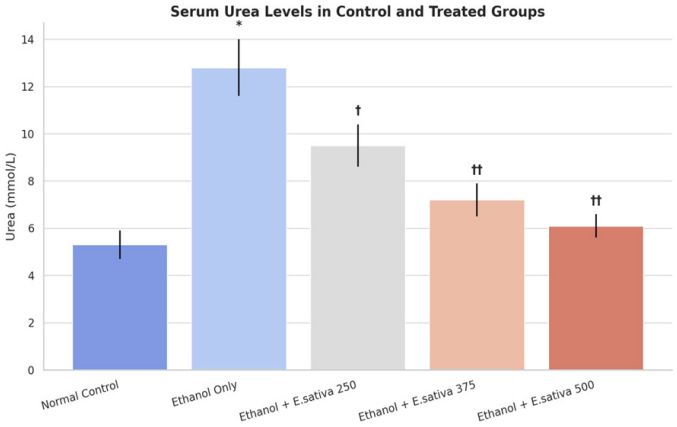

Figure 2: Effect of *E. sativa* extract on serum urea levels in ethanol-treated rats. Values are mean ± standard deviation (SD), n = 6 per group. *Significantly different from Normal Control ($p \leq 0.05$). † Significantly different from Ethanol Only group ($p \leq 0.05$). †† Significantly different from Ethanol Only group with stronger effect ($p \leq 0.05$).

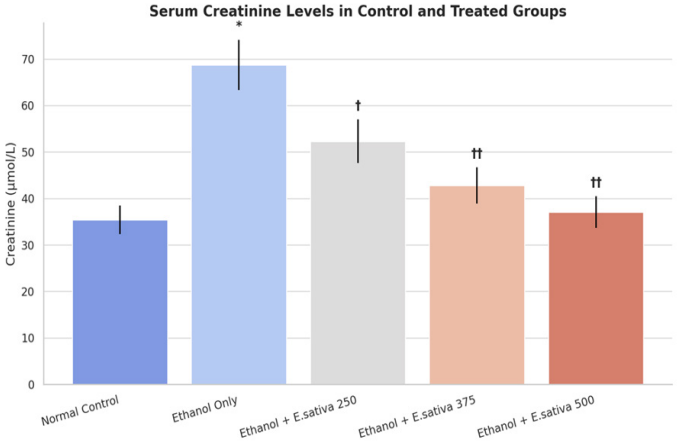

Figure 3: Effect of *E. sativa* extract on serum creatinine levels in ethanol-treated rats. Values are mean ± standard deviation (SD), n = 6 per group. *Significantly different from Normal Control ($p \leq 0.05$). † Significantly different from Ethanol Only group ($p \leq 0.05$). †† Significantly different from Ethanol Only group with stronger effect ($p \leq 0.05$).

The nephroprotective effects observed are likely attributable to the phytochemical constituents of *E. sativa*, which include flavonoids, glucosinolates, and phenolic compounds with well-documented antioxidant and anti-inflammatory properties (Raquel and Fernando, 2016). These bioactive molecules mitigate oxidative stress by scavenging reactive oxygen species generated during ethanol metabolism, thereby preventing lipid peroxidation and cellular damage in renal tissues (Sharma *et al*., 2023). Furthermore, the extract may inhibit proinflammatory cytokine release (e.g., TNF-α, IL-6), thereby attenuating inflammation-mediated renal injury (Thiruvengadam *et al*., 2024). The cumulative antioxidant and anti-inflammatory activities of *E. sativa* plausibly underpin the preservation of renal structural and functional integrity, as reflected by normalized serum biomarkers (Fuentes *et al*., 2014).

The dose-dependent improvements across cystatin C, urea, and creatinine not only confirm the extract's efficacy but also emphasize the importance of appropriate dosing to maximize therapeutic benefits. Of particular note is the significant reduction in cystatin C, which highlights the extract's capability to prevent early glomerular filtration rate decline a critical advantage for early intervention strategies (Seronie-Vivien *et al*., 2008). These results are consistent with existing studies on *E. sativa's* protective effects in models of organ toxicity and extend its application to renal protection in ethanol-induced nephrotoxicity (Alam *et al*., 2007; Boukarine *et al*., 2023).

CONCLUSION

E. sativa leaf extract significantly attenuates ethanol-induced renal dysfunction in rats, as demonstrated by the dose-dependent reduction in serum cystatin C, urea, and creatinine levels. These results suggest that *E. sativa* possesses potent antioxidant and anti-inflammatory properties capable of preserving glomerular and tubular function. This study provides a scientific rationale of *E. sativa* as a natural therapeutic agent for protecting renal health against alcohol-related toxicity.

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NOVELTY STATEMENT

Contrasting previous published studies, that focused on antioxidant and hepatoprotective effect of *E. sativa,* this study estimates the nephroprotective protentional using a sensitive biomarker (Cystain C) accompanying the traditional renal function markers. Moreover, this study gives a new vision into dose-response interactions and the possible therapeutic relevance of *E. sativa* in ameliorative renal dysfunction.

AUTHOR'S CONTRIBUTION

All authors contribute and collaborate equally in this article.

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This is a self-funded research, no fund received from any authorities.

Ethical approval

This study has been approved by Ethical and research committee approval number: ESA and HER- 10-10-07-2025.

Data availability

All data generated or analyzed during the current study are available upon request.

Generative AI and AI-assisted technology statement

The authors declare that no Genrative AI was used in the creation of this manuscript.

Conflict of interest

All authors declared no conflict if interest.

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