

Original Research Article

Gastroprotective and antioxidant effects of curcumin, tahini, and their combination against acute ethanol-induced gastric ulcers in rats

Alaa aldeen Ali irheef, Basim J Hameed*, Qutaiba A Qasim

Department of Clinical Laboratory Sciences, College of Pharmacy, University of Basra, Basra, Iraq

*For correspondence: **Email:** basim.hameed@uobasrah.edu.iq

Sent for review: 7 June 2026

Revised accepted: 19 September 2026

Abstract

Purpose: To investigate the gastroprotective, antioxidant, hepatoprotective, and nephroprotective effects of curcumin and tahini against acute ethanol-induced gastric ulceration and hepato-renal injury in rat model.

Methods: A total of 30 Wistar rats were assigned to 6 groups (n = 5). Group 1 received carboxymethyl cellulose (CMC), group 2 received ethanol, group 3 received esomeprazole (20 mg/kg in 0.5 mL 1 % CMC), group 4 received curcumin (50 mg/kg in 0.5 mL 1 % CMC), group 5 received tahini paste (2.5 mL/kg), group 6 received curcumin in addition to tahini. Biochemical profile was assessed and levels of malondialdehyde (MDA), reduced glutathione (GSH), catalase (CAT), superoxide dismutase (SOD), prostaglandin E₂ (PGE₂), gastric juice pH, and liver/kidney function markers (ALT, AST, CK, urea) and histopathological examination of gastric tissues were evaluated.

Results: Group 2 showed significantly higher MDA, lower GSH, CAT, and SOD, and PGE₂ levels (p < 0.05) compared to group 1. Combination of curcumin and tahini restored levels of GSH, CAT, SOD, MDA, and PGE₂ to normal. Furthermore, Curcumin and Tahini significantly increased gastric pH gastric juice (p < 0.05) and resolved liver and kidney injury.

Conclusion: Curcumin and Tahini significantly reduces levels of inflammatory markers, and restores liver and kidney histoarchitecture.

Keywords: Antioxidants, Curcumin, Ethanol, Gastroprotective, Liver function, Kidney function, Tahini

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided the original work is properly credited.

Tropical Journal of Pharmaceutical Research is indexed by Scopus, Chemical Abstracts, Embase, Index Copernicus, EBSCO, African Index Medicus, JournalSeek, Directory of Open Access Journals (DOAJ), African Journal Online, Bioline International, Open-J-Gate and Pharmacy Abstracts

INTRODUCTION

Gastric ulceration, resulting from erosion of gastric mucosa, is a gastrointestinal disorder associated with considerable morbidity and, in severe cases, mortality [1]. It affects approximately 5 – 10 % of the global population and is associated with multiple etiological factors, including excessive gastric acid secretion, compromised mucosal defense, hematological deficiencies, the use of non-steroidal anti-

inflammatory drugs (NSAIDs), and alcohol consumption. Ethanol is a well-established ulcerogenic agent that induces gastric mucosal injury primarily through oxidative stress, lipid peroxidation, and excessive generation of reactive oxygen species (ROS) [2]. Gastric ulcers are characterized by focal disruption of gastric mucosal barrier, typically resulting from enhanced acid secretion and oxidative damage mediated by free radicals. Experimental ethanol-induced ulcer models are widely used and

considered reliable for evaluating gastroprotective effect of therapeutic candidates [3]. Global prevalence of peptic ulcer disease is estimated at 5 – 10 %, underscoring the need for effective preventive and therapeutic strategies [4]. Major risk factors include *Helicobacter pylori* infection, NSAID use, and alcohol consumption, all of which contribute significantly to ulcer pathogenesis [5]. Additionally, physiological and psychological stress conditions have been implicated in ulceration by inducing mucosal ischemia, erosion, and hemorrhagic damage [6]. Pathogenesis of gastric ulceration is primarily driven by an imbalance between aggressive factors (gastric acid, pepsin, reactive oxygen species, and reduced mucosal blood flow) and protective mechanisms (mucus secretion, bicarbonate production, prostaglandins, nitric oxide, antioxidant activity, and adequate mucosal perfusion) [7]. Disruption of this equilibrium in favor of aggressive factors results in progressive mucosal injury and inflammatory responses [8]. Ethanol, NSAIDs, and *H. pylori* infection exacerbate this imbalance by enhancing ROS production and activating inflammatory pathways [9].

Ethanol-induced gastric injury is characterized by disruption of protective mucus layer, rendering the mucosa vulnerable to acid, pepsin-mediated damage, and oxidative stress. This process is associated with hemorrhagic necrosis, glandular epithelial damage, elevated malondialdehyde (MDA) levels, and depleted endogenous antioxidant enzymes, indicating severe lipid peroxidation and oxidative injury. Furthermore, ethanol promotes gastric acid hypersecretion, inflammatory cytokine release, microvascular dysfunction, and apoptotic cell death, all of which contribute to mucosal degradation [10]. Natural compounds possessing antioxidant and anti-inflammatory properties, such as curcumin and tahini, have attracted increasing attention for potential gastroprotective effect against ethanol-induced gastric injury. The integrity of the gastric mucosa depends on a tightly regulated balance between protective and aggressive factors, which is readily disrupted under oxidative stress conditions. Ethanol exposure disrupts the oxidant–antioxidant balance by increasing lipid peroxidation, and reducing the activity of key antioxidant enzymes (superoxide dismutase (SOD) and catalase), thereby exacerbating gastric mucosal damage [11]. This study therefore investigated the gastroprotective, antioxidant, hepatoprotective, and nephroprotective effects of curcumin, tahini, and their combination against acute ethanol-induced gastric ulceration and hepatorenal injury in a rat model.

METHODS

Animals

A total of 30 Wistar rats were assigned to 6 groups (n = 5). The animals were made to acclimatize for 2 weeks with unrestricted access to standard feed and water under standard laboratory conditions (25 ± 2 °C, 12 h light / dark cycle) [12]. Ethical approval was obtained from the Animal Ethics Committee, College of Pharmacy, University of Basrah (approval no. EC92), and the study was conducted in compliance with the guide for use and care of laboratory animals [13].

Design and treatments

A total of 30 Wistar rats were assigned to 6 groups (n = 5). Group 1 (control) received only the vehicle (0.5 mL 1 % carboxymethyl cellulose (CMC; Chem Cruz / China), group 2 received absolute Ethanol and treated with 0.5 mL 1 % CMC (negative control), group 3 (positive control) received esomeprazole (AstraZeneca / Swedish and British; 20 mg/kg) in 0.5 mL 1 % CMC, group 4 (curcumin group) received curcumin (Iraq; 50 mg/kg in 0.5 mL 1 % CMC), group 5 (tahini) received tahini paste (Iraq; 2.5 mL/kg), and group 6 (curcumin + tahini) received curcumin (Iraq; 50 mg/kg in tahini paste). All treatments were administered orally via gavage to overnight-fasted animals. Absolute ethanol (1 mL at 5 mg/kg) was administered orally to induce gastric gastric ulcers 1 h after treatment. Blood samples were immediately collected via cardiac puncture and transferred into gel-containing tubes. The samples were allowed to clot at room temperature and centrifuged at 3000 × g for 10 min. The obtained serum was carefully separated and stored at –20 °C for further biochemical analysis.

Evaluation of parameters/indices

pH of gastric fluid

The stomach contents were collected into centrifuge tubes and successively centrifuged at 4,000 rpm for 10 min. The pH of the gastric juice was then measured at room temperature using a calibrated digital pH meter [14].

Oxidative stress

Serum levels of malondialdehyde (MDA), reduced glutathione (GSH), catalase (CAT), superoxide dismutase (SOD), and prostaglandin E₂ (PGE₂) were determined using commercially available enzyme-linked immunosorbent assay

(ELISA) and colorimetric assay kits (Elabscience Biotechnology, USA) following manufacturer's instructions [15].

Liver function

Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activity were measured using commercially available colorimetric assay kit (Elabscience Biotechnology, USA) according to the manufacturer's instructions.

Kidney function

Serum creatine kinase (CK) activity was measured using commercial colorimetric assay kit (Elabscience Biotechnology, USA). Serum urea levels were determined using a Urea (BUN) Colorimetric Assay Kit based on urease method (Elabscience Biotechnology, USA), following the manufacturer's protocols. All biochemical parameters were quantified spectrophotometrically in accordance with the kit instructions [16].

Mucosal damage

After blood collection, the abdominal cavity was opened, and the stomach was collected. The tissue was opened along the greater curvature and examined to assess gastric mucosal damage [14].

Histopathology

Gastric tissue samples were collected, gently cleaned, rinsed with normal saline, fixed in 10 % neutral buffered formalin, and processed for routine histological examination. Sections were stained with hematoxylin and eosin (H&E) and examined under a light microscope to assess the extent of gastric mucosal damage and inflammatory changes [17].

Statistical analysis

Data was analyzed using GraphPad Prism 8 (GraphPad Software, San Diego, CA, USA). Values were expressed as mean $\pm$ standard deviation (SD) and compared using one-way analysis of variance (ANOVA). $P < 0.05$ was considered statistically significant.

RESULTS

Gastric antioxidant status and prostaglandin E₂ (PGE₂)

Ethanol significantly decreased GSH, SOD, and CAT and increased MDA levels compared to group 1 ($p < 0.001$), suggesting lipid peroxidation and reduced antioxidant defenses. Treatment with Curcumin and Tahini singly significantly reduced GSH, SOD, CAT, MDA, and PGE₂ from baseline values compared to the combination regimen (Table 1).

Gastric acid secretion and pH

Ethanol significantly increases gastric fluid volume and decreases pH compared to group 1 ($p < 0.05$), suggesting hypersecretion and high acidity. Treatment with Curcumin and Tahini significantly increased gastric volume, with Curcumin significantly increasing pH compared to group 1 ($p < 0.001$). Combination of Curcumin and Tahini significantly reduced gastric fluid and increased pH compared to group 1 ($p < 0.001$; Table 2).

Liver and kidney protective effects

Ethanol administration significantly increased serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), urea, and creatinine levels compared to group 1 ($p < 0.05$). Combined treatment with Curcumin and Tahini significantly reduced Creatinine, ALT, and AST compared to group 1 ($p < 0.05$).

Table 1: Gastric antioxidant status and PGE₂ (mean $\pm$ SD)

Groups	GSH (ng/mL)	SOD (ng/ml)	CAT (ng/ml)	MDA(nmol/ml)	PG-E ₂ (ng/ml)
1	112.24 $\pm$ 5.25	1.85 $\pm$ 0.32	15.22 $\pm$ 1.23	0.57 $\pm$ 0.16	0.69 $\pm$ 0.14
2	68.25 $\pm$ 4.11**	1.34 $\pm$ 0.65*	7.58 $\pm$ 1.12*	1.22 $\pm$ 0.21**	0.26 $\pm$ 0.10**
3	118.52 $\pm$ 4.23**	1.78 $\pm$ 0.24*	14.20 $\pm$ 1.18**	0.79 $\pm$ 0.08**	0.55 $\pm$ 0.11**
4	87.34 $\pm$ 3.65*	1.56 $\pm$ 0.19*	12.54 $\pm$ 2.28*	0.89 $\pm$ 0.11**	0.41 $\pm$ 0.12*
5	82.22 $\pm$ 4.32*	1.48 $\pm$ 0.26*	13.08 $\pm$ 1.10*	0.87 $\pm$ 0.10**	0.46 $\pm$ 0.14**
6	102.32 $\pm$ 5.18**	1.71 $\pm$ 0.22*	17.62 $\pm$ 2.32**	0.70 $\pm$ 0.15**	0.58 $\pm$ 0.09**

Group 1 (0.5 mL 1 % carboxymethyl cellulose (CMC), group 2 (ethanol-induced and treated with 0.5 mL 1 % CMC), group 3 (esomeprazole + CMC), group 4 (curcumin), group 5 (tahini), group 6 (curcumin + tahini). * $P < 0.01$, ** $p < 0.001$ compared to group 1

Table 2: Gastric fluid volume and pH (mean $\pm$ SD)

Group	Gastric Fluid Volume (ml)	pH
1	0.95 $\pm$ 0.28	4.22 $\pm$ 0.41
2	3.5 $\pm$ 0.44**	1.74 $\pm$ 0.82**
3	1.85 $\pm$ 0.34*	3.95 $\pm$ 0.62**
4	1.25 $\pm$ 0.25**	5.15 $\pm$ 0.52**
5	2.10 $\pm$ 0.3**	4.04 $\pm$ 0.42**
6	1.10 $\pm$ 0.2**	5.2 $\pm$ 0.24**

Group 1 (0.5 mL 1 % carboxymethyl cellulose (CMC), group 2 (ethanol-induced and treated with 0.5 mL 1 % CMC), group 3 (esomeprazole + CMC), group 4 (curcumin), group 5 (tahini), group 6 (curcumin + tahini). * $P < 0.01$, ** $p < 0.001$ compared with the normal control

Table 3: Biochemical parameters of liver and renal functions (mean $\pm$ SD)

Groups	Creatinine(mg/dL)	Urea(mg/dL)	ALT(U/mL)	AST(U/mL)
1	0.65 $\pm$ 0.01	21.07 $\pm$ 0.27	41.65 $\pm$ 0.85	125.1 $\pm$ 3.62
2	0.84 $\pm$ 0.04**	41.0 $\pm$ 1.0**	81.75 $\pm$ 0.95**	201.5 $\pm$ 4.24**
3	0.71 $\pm$ 0.01*	33.6 $\pm$ 0.40*	44.6 $\pm$ 0.42**	134.2 $\pm$ 3.4**
4	0.69 $\pm$ 0.09*	25.42 $\pm$ 2.2*	54.45 $\pm$ 2.1**	131.45 $\pm$ 3.0**
5	0.63 $\pm$ 0.04**	24.8 $\pm$ 1.2**	44.21 $\pm$ 1.84**	144.84 $\pm$ 3.81**
6	0.62 $\pm$ 0.06**	22.82 $\pm$ 0.80**	41.32 $\pm$ 1.66**	124.92 $\pm$ 3.22**

Group 1 (0.5 mL 1 % carboxymethyl cellulose (CMC), group 2 (ethanol-induced and treated with 0.5 mL 1 % CMC), group 3 (esomeprazole + CMC), group 4 (curcumin), group 5 (tahini), group 6 (curcumin + tahini). * $P < 0.01$, ** $p < 0.001$ compared to group 1

Gastro histopathology

Group 1 showed completely preserved gastric architecture with intact mucosa, submucosa, and muscularis externa. The surface epithelium consisted of healthy columnar cells with basally located nuclei, normal gastric pits and glands. Also, there was no necrosis, hemorrhage, edema, congestion, or inflammatory infiltration (Figure 1 A). In contrast, absolute ethanol induced severe gastric damage characterized by extensive epithelial sloughing, ulceration, and hemorrhagic erosion, indicating significant disruption of the mucosal barrier. Prominent edema and dense neutrophil infiltration extended into the submucosa. Gastric cells showed pyknosis, vacuolation, and necrotic degeneration (Figure 1 B). Treatment with Esomeprazole significantly preserved gastric histoarchitecture compared to ethanol alone. Only mild superficial erosions were observed, with largely intact epithelial lining and minimal submucosal edema or hemorrhage. Inflammatory infiltration was greatly reduced, and gastric glands remained largely preserved with intact muscular layers (Figure 1 C). Treatment with Curcumin significantly preserved gastric structure, with mostly intact epithelial continuity, mild desquamation, and limited focal hemorrhage. Gastric glands remained well organized, and submucosal edema and congestion were significantly reduced compared to ethanol alone (Figure 1 D). Similarly, treatment with Tahini exhibited well-preserved gastric morphology, with intact epithelial lining, organized glands, and minimal surface erosion. Hemorrhage and edema were rare, and lamina propria and

muscular layers remained structurally stable (Figure 1 E). Combined treatment with Curcumin and Tahini exhibited the most pronounced gastric protection, with near-normal histological architecture comparable to control group. Gastric pits and glands were well organized, with complete preservation of mucosal and muscular layers. No necrosis or inflammation was observed (Figure 1 F).

DISCUSSION

Oxidative stress due to ethanol-induced injury is driven by the activation of proinflammatory signal transduction routes like NF- κ B pathway, leading to oxidative damage, which stimulates ROS formation and release of pro-inflammatory cytokines [18]. However, curcumin treatment reversed these changes. The gastroprotective activity of curcumin is attributed to its ability to directly scavenge reactive oxygen species (ROS) and activate Nrf2-mediated antioxidant response, thereby enhancing the activity of key antioxidant enzymes, including superoxide dismutase (SOD) and catalase (CAT) [19]. Likewise, administration of Tahini showed significant improvements in antioxidant properties, which may have resulted from the presence of bioactive sesame lignans (sesamin and sesamol) [20]. Combination of Curcumin and Tahini scaled up GSH and CAT to levels close to normal (group 1). This synergistic effect was attributed mainly to the lipid-rich medium of Tahini as a natural carrier, which promoted solubility and intestinal uptake of Curcumin [21].

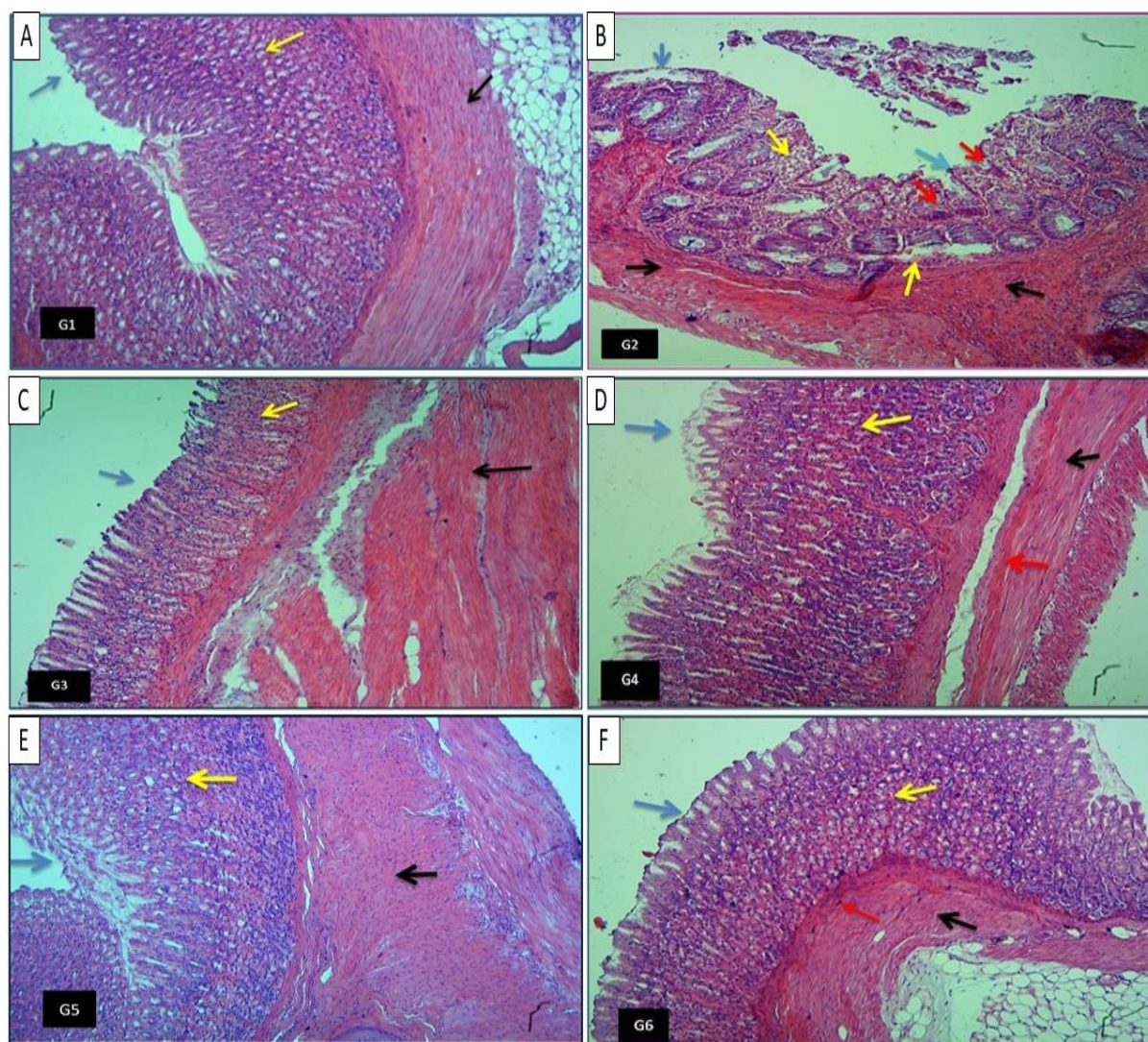


Figure 1: Histopathological changes in rat gastric mucosa of different experimental groups stained with haematoxylin and eosin (H & E). (A) Control, (B) Ethanol-treated, (C) Esomeprazole-treated, (D) Curcumin-treated, (E) Tahini-treated and (F) Tahini + Curcumin-treated groups. Magnifications: 10x and 20x. Arrows indicate the main histopathological findings. Black arrow (muscular layer), red arrow (hemorrhage and congestion), yellow arrow (affected mucosa and submucosa), blue arrow (epithelial layer)

By circumventing the intrinsic limitation of poor bioavailability of curcumin, the Tahini-Curcumin blend provided higher systemic concentrations of polyphenol, maximized the capability to neutralize ROS, and restored PGE₂ levels, consistent with earlier findings [22]. Curcumin and Tahini have been found to exert antisecretory-like effects. Gastric volume was lower and pH increased following administration of Curcumin. Although Curcumin does not target the proton pump directly, its modulating effects on inflammatory mediators and strengthening mucosal barrier indirectly reduce acid-mediated damage. Tahini seemed to play a protective role, improving mucosal coating and anti-inflammatory signaling pathways [23]. Combined therapy had a greater impact on gastric secretion, raising pH, which was also greater than normal values. This highlighted a complete mechanistic synergy of

PGE₂ restoration, leading to secretion of bicarbonate and inhibition of acid formation, with improved bioavailability of curcumin [24].

Curcumin treatment effectively mitigated these systemic markers, demonstrating significant hepatoprotective and nephroprotective effects. This is due to inhibition of pro-inflammatory cytokines and upregulation of Nrf2 antioxidant signaling cascades in a range of organ systems [25]. The curcumin and tahini combination showed the highest absolute protection against ROS. This protection was most effective for the renal system, in which creatinine and urea results were close to normal values. Tahini may have acted as a lipid matrix for curcumin to provide systemic delivery. This makes ROS-based stabilization and other cellular membrane stabilization within the liver and kidney more

effective [26]. Histological evaluation of group 1 showed intact endogenous defense mechanisms such as mucus-bicarbonate secretion, epithelial renewal, and adequate mucosal blood flow that protect against acid and pepsin-mediated injury [27]. In group 2, the injury reflected ethanol-induced reductions in mucosal blood flow and excessive ROS generation, which promoted lipid peroxidation and cellular dysfunction. Microvascular damage with increased permeability was evidenced by hemorrhage and oedema, while inflammatory infiltration indicated activation of pro-inflammatory pathways that amplify tissue injury [28]. This group represented the most severe pathological reference for evaluating therapeutic protection. The protective effect observed in group 3 was mainly attributed to proton pump inhibition, leading to reduced gastric acidity and enhanced mucosal recovery. Increased intragastric pH reduces acid-related injury and supports healing, while additional effects may include improved mucosal blood flow and enhanced mucus/bicarbonate secretion [29]. Overall, this group approached near-normal histology, indicating strong pharmacological gastroprotection. In group 4, muscular layers were largely preserved with rare mild edema. The protective effect of Curcumin is attributed to its antioxidant and anti-inflammatory properties, including ROS scavenging, upregulation of endogenous antioxidant enzymes, and inhibition of pro-inflammatory pathways. These effects contribute to the maintenance of mucosal integrity under oxidative stress [30]. Treatment with Tahini significantly restored tissue architecture (Figure 1 E). The protective effect is attributed to sesame lignans (sesamin, sesamol) and unsaturated fatty acids, which act as antioxidants and inhibit lipid peroxidation. Additionally, its lipid components may support prostaglandin-mediated mucosal defense mechanisms. Overall, tahini demonstrated strong natural cytoprotective potential against ethanol-induced injury consistent with previous studies [31]. A notable finding was mild epithelial vacuolation, associated with healthy nuclear morphology, suggesting enhanced cellular activity rather than degeneration. The submucosa was well vascularized, and tissue integrity was fully maintained, similar to other findings [32]. Combined antioxidant and anti-inflammatory actions of curcumin, together with the lipid-mediated membrane stabilization and prostaglandin support from tahini, produced a synergistic protective effect. This combination demonstrated superior gastroprotection compared with individual treatments and even approached or exceeded standard therapy, supporting reported synergistic interactions

between natural compounds in gastric protection [33].

CONCLUSION

Curcumin and Tahili significantly reduce levels of inflammatory markers and restore liver and kidney histoarchitecture. These findings suggest curcumin and tahini, especially in combination, should be used as a novel and effective therapeutic strategy for the treatment of gastric ulcers. Further research to understand the specific molecular pathways involved, and translating these findings into clinical use should be pursued.

DECLARATIONS

Acknowledgement/Funding

The authors would like to thank the College of Pharmacy, University of Basrah, for their support.

Ethical approval

Ethical approval was obtained from the Animal Ethics Committee, College of Pharmacy, University of Basrah (approval number: EC92).

Use of Artificial intelligence/Large language models

The authors confirm that AI-based tools were used solely for language editing and manuscript polishing purposes (e.g., grammar, phrasing, and clarity). No AI tools were used to generate content, interpret data, or influence study design, results, or conclusions. All intellectual contributions, study analyses, and interpretations reported in the manuscript are the original work of the authors.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Conflict of interest

No conflict of interest is associated with this work.

Contribution of authors

We declare that this work was done by the author(s) named in this article, and all liabilities pertaining to claims relating to the content of this

article will be borne by the authors. B.J.H. and Q. A.Q. designed the study, conducted the analyses and interpreted the data. A.A.I. conducted the methodology and wrote the manuscript. All authors have carefully reviewed and approved the final version of the manuscript.

REFERENCES

- Joshi A, Lehene S, Lasnapure B, Pawar S, Kandipati D, Panchal P. Investigation of antioxidant, anti-ulcer, and analgesic potential of a metal-curcumin complex. *Naunyn Schmiedeberg's Arch Pharmacol* 2023; 396(5): 1043-1052
- Al Moziel MS, Khudhair AA, Qathee MA. Alleviating reproductive dysfunction of lead acetate in male rat: The role of curcumin and metformin. *Bull Pharm Sci Assiut Univ* 2024; 47(2): 1263-1272
- Jabbar AA, Mothana RA, Abdulla MA, Abdullah FO, Ahmed KA-A, Hussien RR, et al. Mechanisms of anti-ulcer actions of *Prangos pabularia* (L.) in ethanol-induced gastric ulcer in rats. *Saudi Pharma J* 2023; 31(12): 101850
- Al Alwan B, Alameri AA, Qasim QA, Vaqqosov S, Jawad KZ, Sivaraman R, et al. Evaluation of structural and thermodynamic parameters of dibenzothiophene desulfurization by carbon nano-filter. *PAC* 2023; 43(9): 8252-8264
- Qasim QA, Alsaad AA, Al-Salman HN. In vivo and in vitro evaluation of the memory-enhancing activity of some isoflavonoids using suitable animal models. *J Chem Health Risks* 2021; 11(3)
- Najm MA, Abd-Alrassol KS, Qasim QA, Hussein HH, Al-Salman HN. Spectrophotometric determination of folic acid using 1, 10-phenanthroline materials with ninhydrin reagent. *Materials Today: Proceedings* 2022; 61: 865-872
- Qasim QA, Fareed YY, Jabir MA, Sahib HB. Evaluation of chemerin and apelin adipokines in obese and non-obese type 2 diabetes mellitus patients. *Int J Pharm Sci Rev Res* 2016; 40(1): 228-233
- Hashim ZR, Qasim QA, AlLabood MH. The association of serum calcium and vitamin D with insulin resistance and beta-cell dysfunction among people with type 2 diabetes. *Arch Razi Inst* 2022; 77(5): 1593
- Parham N, Rahimi K, Ghotbeddin Z, Tabandeh MR. Fish oil ameliorates ethanol-induced gastric injury in rats by modulating genes related to apoptosis. *Sci Rep* 2024; 14(1): 6193
- Jasim SA, Ramirez-Coronel AA, Alameri AA, Sapaev IB, Mustafa YF, Jalil AT, et al. Phenytoin drug detection study through the B24N24 and Al24N24 nano-clusters in gas and solvent phase: DFT, TD-DFT, and thermodynamic study. *Inorganic Commun Chem* 2023; 153: 110887
- Shahzad N, Ibrahim IAA, Alzahrani AR, Al-Ghamdi SS, Alanazi IMM, Ahmad MP, et al. A comprehensive review on phytochemicals as potential therapeutic agents for stress-induced gastric ulcer. *J Umm Al-Qura Univ Appl Sci* 2024; 10(4): 793-808
- Al-Bahadily DC, Alkamil RD, Qasim QA, Shari FH, Al-Salman HN. Analytical and biological activity study of one of the isolated extracts of truffles, ethyl 6-methyl-2-oxo-4-(2-thenyl)-1, 2, 3, 4-tetrahydroperimidined-5-carboxylate (emottc) against three *Candida* species. *Biochem Cell Arch* 2020; 20(2).
- National Research Council. Guide for the care and use of laboratory animals, National Academies Press. Washington, DC; 2010.
- Abd-Alrassol KS, Qasim QA, Ahmed GS, Al-Salman HN. A modified and credible method to estimate nitrofurantoin in the standard of substances and pharmaceutical dosage. *Int J Pharma Res* 2019; 11(4): 1057-1071
- Abd-Alrassol KS, Qasim QA, Al-Salman HN, Hussein HH. The spectrophotometric determination of an antiepileptic drug in standard and pharmaceutical formulations by diazotization coupling reaction and some metal complexes. *Syst Rev Pharm* 2020; 11(3)
- Raish M, Shahid M, Bin Jordan YA, Ansari MA, Alkharfy KM, Ahad A, et al. Gastroprotective effect of sinapic acid on ethanol-induced gastric ulcers in rats: involvement of Nrf2/HO-1 and NF-kappaB signaling and antiapoptotic role. *Front Pharmacol* 2021; 12: 622815
- Ali RS, Shari FH, Hameed BJ. Antioxidant properties of thioctic acid combined with metformin, sitagliptin and rosuvastatin in diabetic rats. *Int J Endocrinol (Ukraine)*. 2025; 21(8): 837-844
- Guo H, Xu Z, Zhu L, Zhu M, Zhang W, Gong M, et al. Standardized aqueous extract of *Abutilon theophrasti* Medic. ameliorates oxidative stress and inflammatory responses against hydrochloric acid/ethanol-induced gastric ulcer in rats. *Front Pharmacol* 2025; 16: 1599810
- Moldoveanu C-A, Tomoaia-Cotisel M, Sevastre-Berghian A, Tomoaia G, Mocanu A, Pal-Racz C, et al. A review on current aspects of curcumin-based effects in relation to neurodegenerative, neuroinflammatory and cerebrovascular diseases. *Molecules* 2024; 30(1): 43
- Ghafoori-Fard S, Shoorei H, Bahroudi Z, Hussien BM, Talebi SF, Taheri M, et al. Nrf2-related therapeutic effects of curcumin in different disorders. *Biomolecules* 2022; 12(1)
- Alshahrani SH, Alameri AA, Kahar F, Ramirez-Coronel AA, Obaid RF, Alsaikhan F, et al. Overview of the role and action mechanism of microRNA-128 in viral infections. *Microb Pathog* 2023; 176: 106020
- Yeerong K, Inpan R, Aisara J, Settakorn K, Takuathung MN, Thinrungraj N, et al. Therapeutic effects of curcumin on upper gastrointestinal diseases: a systematic review and meta-analysis of animal studies. *BMC Complement Med Ther* 2025; 26(1): 54
- Zamanian MY, Golmohammadi M, Nili-Ahmadabadi A, Alameri AA, Al-Hassan M, Alshahrani SH, et al. Targeting autophagy with tamoxifen in breast cancer: From molecular mechanisms to targeted therapy. *Fundam Clin Pharmacol* 2023; 37(6): 1092-1108

24. Atefi M, Vahedi H, Darand M, Entezari MH. Sesame oil improves biomarkers of glycemic control in women with metabolic dysfunction-associated steatotic liver disease: a randomized, double-blind, controlled clinical trial. *BMC Nutr* 2025; 11(1): 134
25. Mohammed K, Basim JH, Nadheerah N. Study the effect of olibanum (*Boswellia carterii*) on hyperuricemia in rat model. *Iraqi J Pharm Sci* 2024; 33(2): 161-169
26. Margiana R, Hamoud AS, Kayumova D, Hussien RAA, Hjazi A, Alsalamy A, et al. Association between maternal exposure to arsenic by drinking water during pregnancy and risk of preterm birth: a systematic review and meta-analysis. *Int J Environ Health Res* 2024; 34(8): 2947-2956
27. Al-Kawaz JMJ, Al-Zubaidy FM, Al-Harbi HJO. Therapeutic effects of esomeprazole, curcumin, chitosan, and curcumin-chitosan mixture on ethanol-induced gastric ulcer in female rats. *J Angiother* 2024; 8(1): 1-12
28. Qasim Q. Antioxidants, lipid profiles, and glucose levels, as well as persistent inflammation, are central to the link between diabetes mellitus type II and oxidative stress. *Georgian Med News* 2024; 1(351): 158-161
29. Yi Z-h, Wu Y, Yu Y, Li F, Zeng T, Zeng Y, et al. Gender differences in the effects of esomeprazole on ethanol-induced acute gastric injury in mice. *Front Biosci (Landmark Ed)* 2026; 31(4)
30. Abu-Elfotuh K, Abbas AN, Najm MA, Qasim QA, Hamdan AM, Abdelrehim AB, et al. Neuroprotective effects of punicalagin and/or micronized zeolite clinoptilolite on manganese-induced Parkinson's disease in a rat model: Involvement of multiple pathways. *CNS Neurosci Ther* 2024; 30(10): e70008
31. Hancı N, Odabaşoğlu F, Dumlupınar FA, Berktaş OA, Kutlu Z, Halıcı M, et al. Sesame oil has gastroprotective and anti-oxidative properties: An experimental study in rats with indomethacin-induced gastric ulcers. *EJFST* 2018; 2(2): 53-68
32. Qian Y, Xu L, Li M, Chen X, Chen J, Cai K, et al. Synergistic co-delivery of curcumin and N-acetylneuraminate via bigel: Enhanced stability, antioxidant performance, and controlled release during in vitro digestion. *Food Chem X* 2025: 103318
33. Darko D, Kwekutsu E, Idoko IP. Synergistic effects of phytochemicals in combating chronic diseases with insights into molecular mechanisms and nutraceutical development. *Int J Innov Sci Res Technol* 2025; 10(3): 1865-1883.