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Exploring the Multifaceted Healing Powers of Turnip Leaves from Saudi Arabia: Chemical Composition, Anti-ulcer, Antibacterial, and Apoptosis Regulatory Properties

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ABSTRACT

This study investigates the therapeutic potential of turnip leaves powder (TLP) for treating peptic ulcers and bacterial infections, focusing on its phytochemical composition and bioactivity. GC-MS analysis showed that the major bioactive components identified in TLP were 9,12,15-octadecatrienoic acid methyl ester, hexadecanoic acid methyl ester, ageratriol, 2-hexadecanol, 7,10,13-hexadecatrienoic acid methyl ester, methyl tetradecanoate, 2-myristynoyl pantetheine, linalool and 9-hexadecenoic acid. TLP significantly reduced the size of ethanol-induced gastric ulcers in Wistar rats by 79.7%, which was comparable to that caused by omeprazole (85%). In addition, TLP treatment caused a significant decrease in proinflammatory cytokines TNF-α, IL-1β and IL-6, with the most significant decrease observed in TNF-α (89.6 %) and IL-1β (87.2 %). Conversely, treatment with TLP increased anti- inflammatory cytokines IL-10 and IFN-γ, demonstrating a balanced immunological response that favours ulcer healing. Finally, treatment with TLP decreased apoptosis indicators; caspase-3 and caspase-9 and showed significant antibacterial activity against several bacterial strains.

Keywords: Turnip, Anti-ulcer, Cytokines, Anti-apoptosis, Antibacterial.

INTRODUCTION

Gastric and duodenal ulcers are prevalent global health issues, exerting substantial influence on the overall health of individuals and the functioning of healthcare systems. Gastro esophageal reflux

disease (GERD) is a condition related to peptic ulcers that occurs when there is an imbalance between the stomach lining's protective factors and harmful substances, especially acidic secretions. Various factors, such as viral infections, smoking, stress, certain drugs, and high alcohol consumption, can exacerbate this imbalance.[1-4] Bacterial infections are another health concern that is becoming more challenging due to the rise in bacterial strains that are resistant to a wide range of antibiotics. Emphasizing disease prevention is frequently regarded as more advantageous than depending on many therapies. The significance of diet in general health is paramount since it serves as the primary provider of both mental and physical energy. The functional characteristics of food play a crucial role in supplying needed nutrients and promoting and enhancing overall health.

The biennial herbaceous plant, called turnip, is formally classified as *Brassica rapa* L (BR). It is a member of the Cruciferae Brassica family. This adaptable plant often reaches maturity over two months and can be cultivated during various seasons, such as spring, late summer, and autumn. The flexibility of turnip to adjust to multiple growing seasons and its harvestable components can be used for both culinary and dietary purposes, making it a practical and readily available plant.[5] Turnips offer substantial nutritional benefits, rich in carbohydrates, vitamins, and minerals, and have long been used in traditional medicine for treating various ailments.[5,6,7,8,9] Research has demonstrated that it possess several beneficial biological features, including anti-inflammatory and antioxidant characteristics and antibacterial and possibly anticancer benefits.[5,10] Numerous studies have also investigated the anti-diabetic and analgesic properties of turnips and their efficacy in reducing cholesterol levels and enhancing cardiovascular, renal, and hepatic well-being.[11] The potential of turnips in preventing osteoporosis and their efficacy in treating pulmonary edema and cerebral hypoxia highlight the wide-ranging and substantial health advantages linked to turnips.[5,12-14] The leaves, florets and various parts of medicinal plants play a crucial role in both traditional and modern medicine and offer a wealth of therapeutic benefits[15]. These leaves contain various bioactive compounds that are utilized for their healing properties.[15-17] Therefore, based on the traditional uses of turnip in disease treatment and on the pharmacological studies that revealed the high bioactivity of turnip, it has been decided to further investigate the therapeutic efficacy of turnip leaves from Saudi Arabia. Herein, we investigate the potential of turnip as an effective treatment for gastric ulcers and bacterial infection.

MATERIALS AND METHODS

The ELISA kits employed in this study were procured from My BioSource and Abcam, USA whereas the remaining chemicals were obtained from Sigma Aldrich, USA.

Plant collection, identification, and Cold maceration extraction

Samples of turnip leaves were obtained from a local market in Ha'il Saudi Arabia (27°29'18.8"N 41°41'09.6"E) and carefully rinsed with Millipore water to remove any impurities. Then, they were air-dried for 10 days. The turnip plant was identified by Dr. Naila Alkefai (University of Hafr Al-Batin, Saudi Arabia) and a specimen of the stem and leaves were deposited at the Pharmacy College, University of Hail with the number UOHCOP014. The bioactive components of TLP were extracted by cold maceration with methanol by method followed by Syed, R. U *et al.,* 2022.[17]

GC-MS analysis

TLP's methanolic extract was analyzed using the Thermo Scientific GC–MS-AS 3000 autosampler with an IQS detector and equipped with a TR 5MS Capillary Standard non-polar column that has dimensions of 30 mm in length, 0.25 mm in internal diameter, and a 0.25 µm film coating. The identity of components was determined by utilizing the NIST and MAINLIB libraries, as well as by comparing their retention indices.[17]

In vivo experiments

Experimental animals

Male Wistar rats (150 g–300 g) were used for the study. Each rat was housed individually in its specific pathogen free cage at temperatures between 22 and 28°C and relative humidity between 50 and 60% at a controlled atmosphere with equal periods of light and darkness and unrestricted access to clean water and food. The experiment protocol was approved by the Research Ethics Committee (REC) at the University of Ha'il, Saudi Arabia, with the reference number H-2024-102.

Gastric ulcer models

20 male Wistar rats took part in the study and were divided into 04 groups (G1to G4) of 05 rats each. The experimental strategy included creating ulcers using a slightly modified approach based on an already established procedure.[17-19] G 1: Normal control-These rats were not subjected to any intervention or therapy. G 2: Disease control (ulcer group)-ulcers were induced by administration orally of 95% ethanol at a dose of 5 mL/kg body weight. G3: Standard drug treatment-The rats in this group received oral administration of omeprazole (OMZ) (20 mg/kg body weight, diluted in distilled water (DW)) 2 h before ethanol exposure. G 4: Treatment with the test drug. The rats in this group received 1 mL of turnip leaves powder (TLP) solution at a 400 mg/kg body weight, dissolved in DW, and administered orally 2 h before exposure to ethanol. Before the rats were euthanized for the purpose of blood sampling, diethyl ether was administered as an anesthetic. Euthanasia was performed by surgically severing the jugular veins in the neck with a sterilized scalpel.

A dose of 400 mg/kg TLP was used based on previous studies that administered the same dose of the same plant species (Brassica rapa) to groups of mice and rats.[20,21]

Determination of ulcer index and %inhibition of ulcer

The ulcer index (UI) was determined by a previously used method,[22] through the following equation:

$$U.I. = \frac{Ulcerated\ area}{Total\ stomach\ area} \times 100$$

The ulcerated area was determined using a dissecting microscope, whereby 2 mm × 2 mm squares were counted throughout the ulcer's length and breadth, and the sum of these squares represented the ulcerated region's area. The following was used for calculating inhibition:

Macroscopic and biochemical gastric evaluations

The size of the ulcerated region was estimated according to a standard procedure. In addition, the calculation of percent inhibition was done by slightly modified method described in a previous study.[1,3,23] pH was measured and then neutralized with a sodium hydroxide solution.

Collection of Serum

The procedure began by withdrawing blood from the tail veins of the animals, without the use of anticoagulants. This blood was then collected. By tilting these tubes and then centrifuging. The supernatant was then centrifuged to obtain the serum, which was then stored in the refrigerator at temperatures between 2 and 8°C. The next day, the serum was thoroughly analyzed using an enzymatic immunoassay (ELISA). This test specifically measured the levels of various pro-inflammatory cytokines.

Serum inflammatory cytokines

Cytokines were quantified using the sandwich ELISA technique.[23] Specific ELISA kits from MyBioSource and Abcam, USA, were used for this purpose. This method accurately measured the concentration of the individual cytokines in the serum. The ELx800 ELISA reader, also from the USA, was used to read the final measurements. The data collected was then plotted on a standard curve to accurately determine the concentrations of these pro-inflammatory cytokines in serum. The concentrations were then expressed in picograms per milliliter (pg/mL).

Serum apoptosis markers

The concentrations of apoptosis markers, in particular caspase-3 and caspase-9, were quantified in rat serum using a special ELISA kit from MyBioSource, USA. These markers were detected at an optical density of 450 nm.

Determination of antibacterial effect

Bacterial strains used

The study employed bacterial strains: *Staphylococcus aureus* ATCC 512477, *Staphylococcus epidermidis* ATCC 12228, *Proteus mirabilis* ATCC 299, *Salmonella choleraesuis* ATCC 10708, *Enterococcus faecalis* ATCC 29212, *Pseudomonas aeruginosa* ATCC 27853, *Klebsiella pneumoniae* ATCC 700603, *Escherichia coli* ATCC 25922.

Standardization of bacterial culture

This was conducted following the protocol outlined by Syed *et al.,*[24]

Determination of antibacterial susceptibility

The test was conducted following the protocol outlined by Syed *et al.,*[24]

Statistical analysis

The statistical analysis was done via Prism 9 using one-way ANOVA, followed by Tukey's test.

RESULTS

Determination of bioactive constituents

Figure 1, the chromatogram depicting the analysis of TLP methanolic extract shows clear peaks, as evidenced by GC-MS analysis. GC-MS revealed the presence of over 100 compounds, mainly at trace levels, with 12 major compounds being particularly prominent (Table 1) (Fig. 2). This study detected the fatty alcohols 1-hexadecanol and 2-hexadecanol with retenton times (RT) of 28.19 min and 38.54 min, respectively. Hexadecanoic acid methyl ester (methyl palmitate) was identified at an RT of 32.49 min, accounting for 6.90% of the chromatogram. In addition, 7,10,13-hexadecatrienoic acid methyl ester, ageratriol and methyl tetradecanoate were also identified in TLP methanolic extract with characteristic RT values (see Table 1). Ageratriol, a sesquiterpene, was detected at a RT of 21.56 minute. Methyl tetradecanoate, the methyl ester of tetradecanoic acid (myristic acid), was detected together with 2-myristynoylpantheline at an RT of 25.31 min, accounting for 2.04% of the chromatogram. Furthermore, 9,12,15-octadecatrienoic acid methyl ester (methyl linoleate) or alpha-methyl linoleate, was identified within the extract at an RT of 40.43 min with a probability index of 12.44% and accounted for 10.23% of the chromatogram. Linalool, a naturally occurring terpene alcohol found in TLP extract, was determined at an RT of 12.90 minute. In addition, 9-hexadecenoic acid (palmitoleic acid) was detected at an RT of 16.86 min, accounting for 1.27% of the chromatogram. Finally, ethyl iso-alcoholate and dasycarpidan-1-methanol acetate (esters) were identified in the extract, albeit to a lesser extent.

Table 1: Major chemical constituents of CMETL analyzed through GC-MS

Sr. No	Compound name	Molecular formula	Molecular weight	Retention time(Min)	Probability Index	Percent area of curve
1	1-Hexadecanol, 2-methyl-	$C_{17}H_{36}O$	256	28.19	8.63	0.17
2	2-Hexadecanol	$C_{16}H_{34}O$	242	38.54	75.95	4.15
3	Hexadecanoic acid, methyl ester	$C_{17}H_{34}O_2$	270	32.49	37.60	6.90
4	7,10,13-Hexadecatrienoic acid, methyl ester	$C_{17}H_{28}O_2$	264	30.65	7.97	3.97
5	Ageratriol	$C_{15}H_{24}O_3$	252	21.56	37.72	8.83
6	Methyl tetradecanoate	$C_{15}H_{30}O_2$	242	22.82	32.18	3.64
7	2-Myristynoyl pantetheine	$C_{25}H_{44}N_2O_5S$	484	25.31	38.32	2.04
8	9,12,15-Octadecatrienoic acid, methyl ester, (Z,Z,Z)-	$C_{19}H_{32}O_2$	292	40.43	12.44	10.23
9	Linalool	$C_{10}H_{18}O$	154	12.90	37.52	1.39
10	9-Hexadecenoic acid	$C_{16}H_{30}O_2$	254	16.86	7.04	1.27
11	Ethyl iso-allocholate	$C_{26}H_{44}O_5$	436	20.42	10.21	0.72
12	Dasycarpidan-1-methanol, acetate (ester)	$C_{20}H_{26}N_2O_2$	326	46.36	12.72	0.09

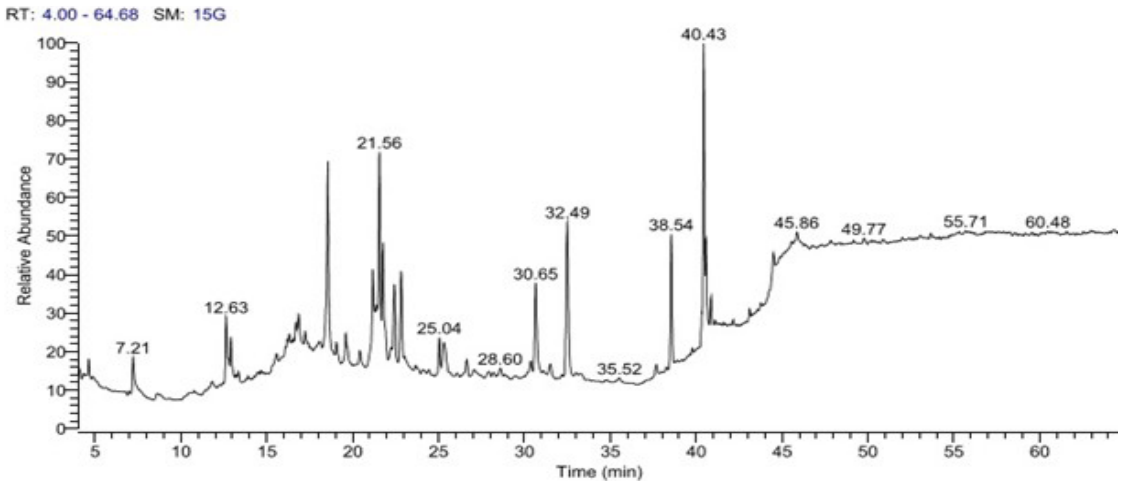

Fig. 1. GC-MS chromatogram of CMETL

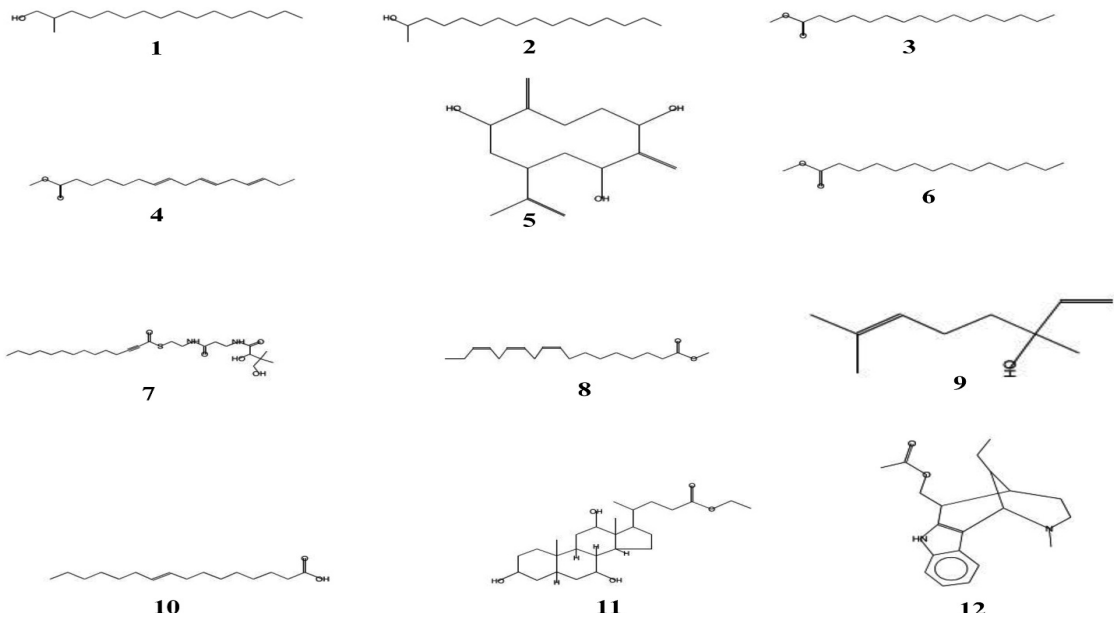

Fig. 2. Possible bioactive compounds and their chemical structures (1) 1-Hexadecanol, 2-methyl- (2) 2-Hexadecanol (3) Hexadecanoic acid, methyl ester (4) 7,10,13-Hexadecatrienoic acid, methyl ester (5) Ageratriol (6) Methyl tetradecanoate (7) 2-Myristynoyl pantetheine (8) 9,12,15-Octadecatrienoic acid, methyl ester, (Z,Z,Z)-(9) Linalool (10) 9-Hexadecenoic acid (11) Ethyl iso-allocholate (12) Dasycarpidan-1-methanol, acetate (ester)

Study on ulceration and treatment

Table 2 presents data on the effects of TLP on the healing of gastric ulcers in rats. In addition, Fig. 3 shows the macroscopic view of rat stomachs, highlighting hemorrhagic lesions in the glandular region. The results show that the control group had an average size of 611±32 mm². In contrast, rats treated with 400 mg/kg TLP showed a significant reduction in ulcer size to 124± 6.21 mm², a decrease of 79.7±2.97%. Treatment with omeprazole resulted in an even smaller ulcer size of 90±3.5 mm², corresponding to a healing rate of 85±1.9%.

Table 2: Effects of intra esophageal administration of Turnip leaves powder (TLP) and omeprazole on biochemical parameters of gastric juice recovered from rats

Groups	Treatment	Ulcer area(mm²)	% of inhibition	Mucus weight	pH
1	Normal control	0.00	0.00	2.8 ± 0.11	3.61 ± 0.09
2	Ulcer Control	611 ± 32	NA	0.95 ± 0.2	3.61 ± 0.21
3	Omeprazole	90 ± 3.5	85 ± 1.9	1.45 ± 0.3	6.54 ± 0.09
4	TLP (400 mg/kg)	124 ± 6.21	79.7 ± 2.97	2.94 ± 0.14	7.02 ± 0.55

TLP: Turnip leaves powder

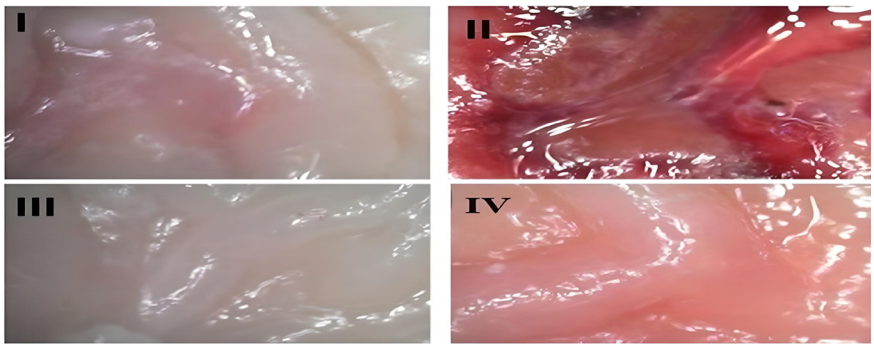

Fig. 3. Macroscopic inspection of hemorrhagic lesions in rat stomach. (I–IV) are representative photos from G 1, 2, 3, and 4, respectively. Photo II showed the most severe hemorrhagic lesions from G2

Study on Biomarkers

In G2, which represents the diseased animals, there was a significant increase in inflammatory cytokine levels compared to G1 (the normal group). Conversely, inflammatory cytokine levels decreased significantly in G3, treated with the standard OMZ, and in G4, receiving the test drug TLP, with a significant decrease observed (Table 3).

Table 3. Cytokine network study on the effect of TLP treatment in ulcerated rats

Type of cytokines	Cytokine level (pg/mL)						
	G1	G2	%increase	G3	%decrease$	G4	%decrease$$
IL-1β	36.149 ± 7.13	501.99 ± 83.3	1288.67	83.792 ± 7.7	83.308	64.232 ± 2.69	87.204
IL-6	47.22 ± 3.3	510.66 ± 100.5	981.449	226.072 ± 54	55.729	207.92 ± 53.58	59.284
IL-8R	0.445 ± 0.3	6.145 ± 0.49	1280.9	2.27 ± 0.49	63.059	1.445 ± 0.4	76.484
IL-2	0.594 ± 0.09	30.06 ± 2.82	4960.61	3.672 ± 1.46	87.784	3.198 ± 1	89.361
IL-4	0.9188 ± 0.06	5.997 ± 1.8	552.764	2.962 ± 0.164	50.605	2.739 ± 0.128	54.327
IL-10	44.902 ± 3.6	288.668 ± 63.66	542.885	75.772 ± 12.68	73.751	72.912 ± 8.52	74.741
IFN-γ	3.48 ± 1.45	39.26 ± 10	1028.16	28.12 ± 4.4	28.374	28.84 ± 3.4	26.541
TNF-α	21.71 ± 7.7	939.319 ± 39.24	4226.67	142.29 ± 16	84.851	97.168 ± 9.5	89.655

Values are mean±SD (n=5). $Percentage decrease after treating with OMZ 20 mg/kg body weight; $$Percentage decrease after treating with turnip leaves powder (TLP) at 400 mg/kg body weight

Remarkably, after ulcer induction, TNF-α exhibited the greatest increase among the cytokines, with IL-1β and IL-6 also showing a notable increase. However, after administration of TLP for treatment, TNF-α showed the greatest decrease at 89.6%, closely followed by IL-2 and IL-1β, which decreased by approximately 89.4% and 87.2% with TLP treatment, respectively. The decrease in IL-6 and IFN-γ levels was much less pronounced than that of TNF-α and IL-1β, as shown in Table 3. Meanwhile, IL-4 and IL-10 were surprisingly increased upon induction of ulcer in the rats, however, TLP treatment resulted in a decrease in both cytokines by 54.3% and 74.7%, respectively (Table 3). It is crucial to note that omeprazole treated G3 also decreased inflammatory cytokines, however, its effect was generally less than that resulting from TLP treatment. These results were summarized and illustrated in Table 3, Fig. 4 and Figure 5.

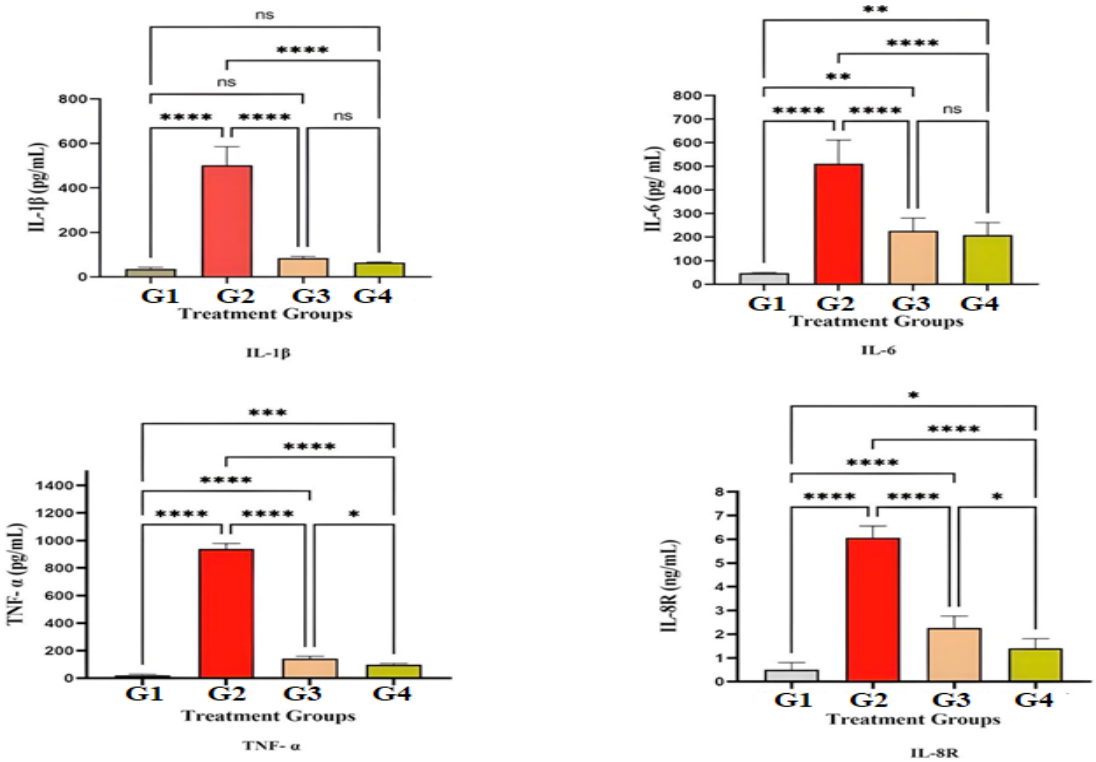

Fig. 4. Study on proinflammatory cytokines. ** Extremely highly significant; *** Very highly significant; ** Highly significant; * Significant; ns: not significant at p > 0.05 level**

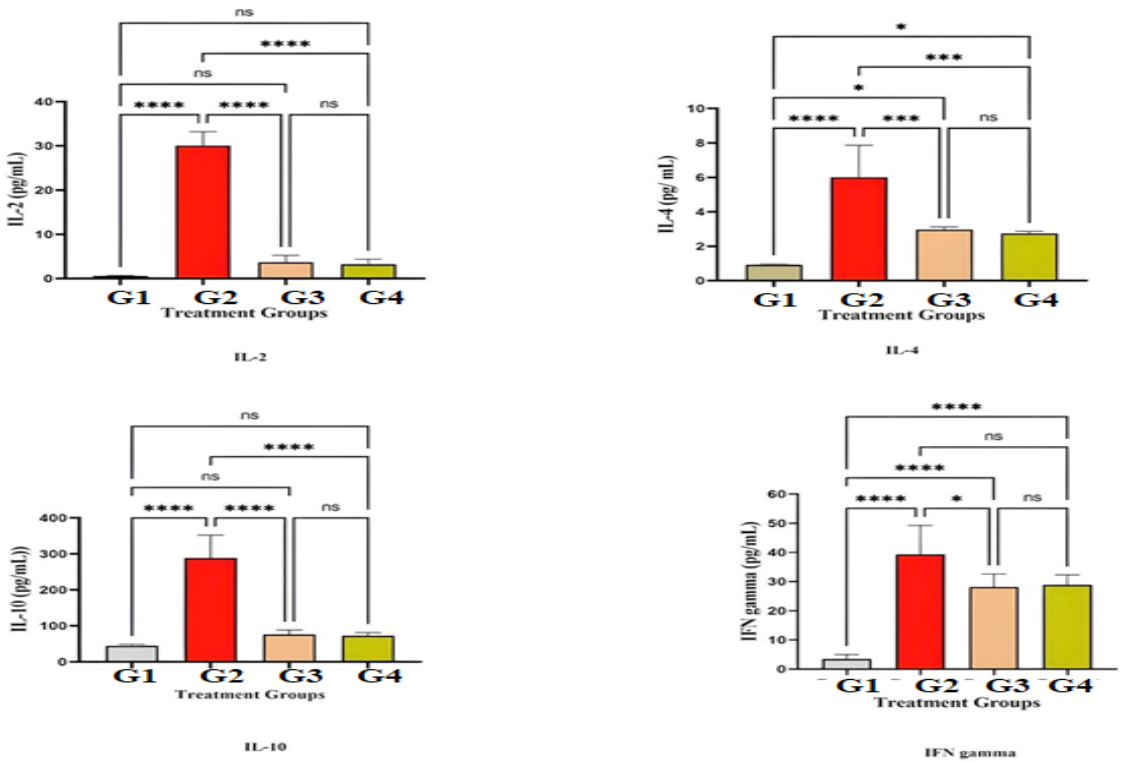

Fig. 5. Study on anti-inflammatory cytokines. ****Extremely highly significant; ***Very highly significant; **Highly significant; *Significant; ns: not significant at p>0.05 level

Regarding apoptotic markers, caspases-3 and -9 in G2 showed a significant increase after ulcer induction. However, both caspase-3 and caspase-9 levels decreased significantly after treatment with TLP (G3) relative to disease G2, as shown in Fig. 6. It is noteworthy that the reduction in caspase-3 and caspase-9 levels was not significant in the TLP-treated animals (G4) compared to the OMZ-treated group (G3). The study highlights the immunomodulatory effect of TLP, which contributes to the treatment of ulcers.

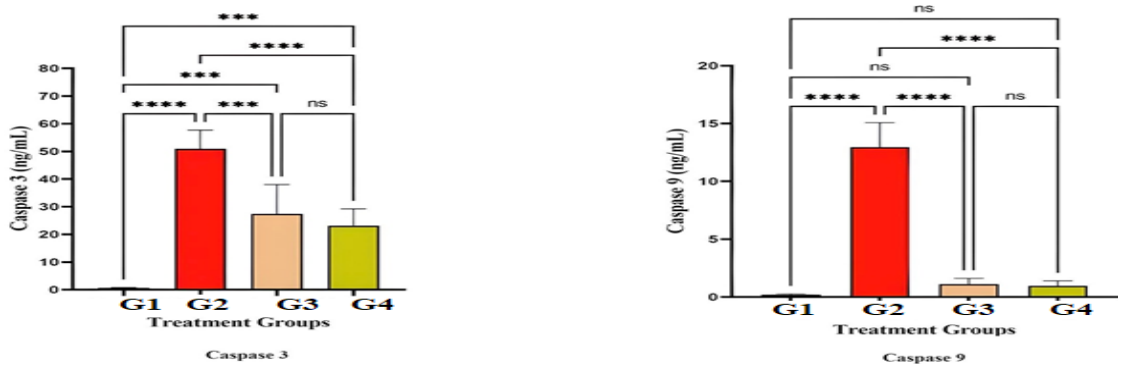

Fig. 6. Study on apoptosis markers. ****Extremely highly significant; ***Very highly significant ; **Highly significant ; *Significant; ns: not significant at p>0.05 level

Study on antibacterial effects

TLP exhibited its strongest antibacterial activity against *Escherichia coli*, while *Staphylococcus aureus* was the second most potent. It is noteworthy that the efficacy of the TLP extract was comparable for both *Gram-positive* and *Gram-negative* bacteria (Fig. 7). As shown in Table 4, the efficacy of the methanolic TLP extract was lower than ciprofloxacin. Though TLP showed moderate activity in crude form, its performance can be increased by further purification of the compound.

Table 4: Antibacterial activity of CMETL

Organisms	CFU	Zone of inhibition. (mm)	
		Extract	Ciprofloxacin (5 µg/Disc)
Staphylococcus aureus ATCC 512477	3×10^4	18.3 ± 2.5	34 ± 2
Staphylococcus epidermidis ATCC 12228	2×10^3	15.3 ± 1.5	32.3 ± 1.5
Escherichia coli ATCC 25922	4×10^5	20.3 ± 2	37.3 ± 1.15
Proteus mirabilis ATCC 299	2×10^3	17.3 ± 3.2	27 ± 1
Salmonella choleraesuis ATCC 10708	4×10^5	17.6 ± 3.5	29.3 ± 2.5
Klebsiella pneumoniae ATCC 700603	3×10^3	16.3 ± 1.5	29.3 ± 1.5
Enterococcus faecalis ATCC 29212	4×10^5	18.3 ± 1.5	30.6 ± 2.5
Pseudomonas aeruginosa ATCC 27853	2×10^4	15.6 ± 1.5	29.33 + 2.5

CFU: Colony Forming Unit. #Values are mean±SD (n=6)

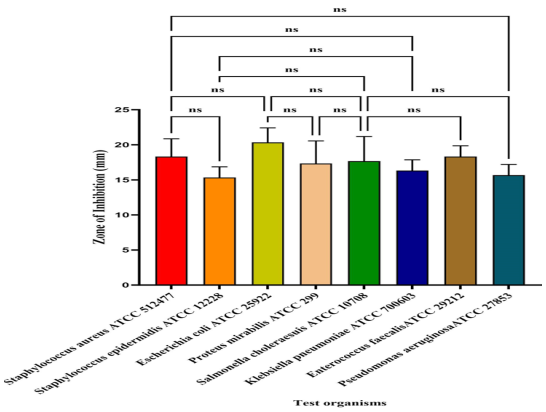

Fig. 7. Comparative antibacterial study of CMETL. All the results were not significant (ns) to each other at p > 0.05 level

DISCUSSION

The comprehensive study presented sheds light on the multifaceted role of bioactive constituents in TLP in the context of gastric ulcer treatment, with a particular focus on their impact on inflammatory and apoptotic markers. The identification of over 50 compounds in TLP's methanolic extract, with a focus on 12 major constituents, underscores the complexity and richness of natural extracts in offering a spectrum of bioactive molecules. Many bioactive compounds have been identified in the extracts which are displayed in Table 1. Research on turnips has revealed their potential in various health domains, including their anticancer, antimicrobial, anti-hypoxia, anti-diabetic, antioxidant, and nephroprotective properties. Consequently, turnips have become a focal point of significant interest in scientific investigations.[9] 9,12,15-Octadecatrienoic acid methyl ester, occurring naturally, has been detected in various plant species including *Allamanda cathartica* L. and *Pithecellobium dulce*.[25,26] It serves as a significant constituent in plants like *Lepidium sativum*, which display antibacterial properties.[27] Furthermore, it is a major component of several plants demonstrating antibacterial properties, such as *Lepidium sativum* and *Plumeria alba*.[27,28] *Octadecadienoic acid* (Z,Z)-(α-linoleic acid), may reduce the risk of heart disease.3,23 n-Hexadecanoic acid (palmitic acid) has been identified as a significant bioactive compound. Recent research indicates that palmitic acid significantly inhibits prostate cancer.[29] It also showed anti-inflammatory properties and potent antibacterial effects against biofilm-forming bacteria.[30]

The present study was meant to check the therapeutic efficacy of TLP in treating gastric ulcers by primarily investigating its effects on the cytokine network. The results showed that TLP has greater efficacy in modulating inflammatory cytokine levels than the usual administration of omeprazole. The importance of IL-1β, a vital cytokine, in regulating inflammation-related processes in the intestinal mucosa has been demonstrated in previous studies.[31,32] Interleukin-1β (IL-1β) maintains the integrity of the gastrointestinal mucosa. Its primary role is to inhibit the migration of neutrophils induced by chemotaxis. The present study observed a marked increase in IL-1β levels in G2 ulcer-affected animals.

Interleukin-6 (IL-6) is another proinflammatory cytokine. According to Petrasek *et al.,*[33] the production of this cytokine occurs in response to tissue injury. It is a soluble mediator with multiple roles in inflammation and immunological response, particularly in peptic ulcers. TNF-α is a proinflammatory cytokine that is produced by macrophages and plays a central role in regulating inflammation and immunological responses, as well as in the modulation of immune cells and the

body's response to tissue damage or infection. It also combats in gastric ulcers, essentially gastric mucosa lesions. Promoting inflammation, stimulating cell apoptosis, and disrupting the protective mucosal barrier of the gastric mucosa can potentially exacerbate tissue damage. Studies have shown that gastric ulcers often have elevated TNF- α levels.[34,35] Neutrophils are the primary hosts of the IL-8 receptor (IL-8R), which significantly influences the inflammatory process. The receptor in question has a pronounced affinity for IL-8, a central component of the natural immune system that is synthesized by macrophages and an essential protein in the inflammatory response. In gastric ulcers, especially those due to Helicobacter pylori infection, the IL-8 gene shows the highest upregulation, highlighting its importance in the physiological response to these infections.[36-38] The effects of this phenomenon encompass all facets of the body's immune system, emphasizing its importance in treating and responding to peptic ulcers.[39,40] IL-2 and IFN-γ are also pro-inflammatory cytokines that were investigated in this study. It can be clearly seen from the results that all these proinflammatory cytokines were upregulated in the ulcerated rats as expected, but treatment with TLP reduced their levels in a satisfactory extent.

The study also investigated the levels of IL-4 and IL-10 in ulcerated rats, and these are classified as anti-inflammatory cytokines.[41] Surprisingly, these cytokines were actually significantly upregulated in ulcerated rats, however, significant upregulation of anti-inflammatory cytokines might result in a reverse effect and worsen inflammatory injury.[42] Treatment with TLP also resulted in significantly decreasing IL-4 and IL-10 in the ulcerated rats, which further contributed towards the antiulcer effect of turnip. In general, the study underscored the immunoregulatory properties of TLP, attributed to its content of palmitic acid and alpha-linolenic acid, with the former associated with the promotion of pro-inflammatory responses and the latter with their suppression, as noted in previous studies.[43,44] The role of caspase-3 in the apoptotic pathway, essential for the breakdown of various cellular proteins and removing old, damaged, or dysfunctional cells, was also examined. Caspase-9, another critical enzyme in apoptosis, particularly regarding mitochondrial damage, was investigated. Research by Morandi *et al.,*[45] demonstrated that an aqueous extract of olive leaves can mitigate apoptosis by lowering caspase-3 and -9 levels, an effect attributed to hydroxytyrosol and oleuropein. This aligns with findings from Yaguchi *et al.,*[46], who reported that linoleic acid suppresses these caspases. These findings suggest a balance between pro-inflammatory and anti-inflammatory actions, which may contribute to the therapeutic potential of the extract in ulcer treatment. Additionally, TLP's methanolic extract displayed a broad spectrum of antibacterial activities, supporting previous findings that turnip extract exhibits antibacterial properties.[46,47] The antibacterial properties of TLP could be due to hexadecanoic acid methyl ester, alpha-methyl linoleate, linalool and palmitoleic acid.[48-50]

This comprehensive study on the effect of turnip leaves in treating gastric ulcers and bacterial infections illustrates the complexity of natural extracts and their potential therapeutic benefits. However, a limitation of the study is the inherent complexity of natural extracts, which contain various compounds that can interact in unpredictable ways, making it difficult to isolate the effects of individual constituents. Furthermore, while the study provides valuable insight into the anti-inflammatory activity of turnip and its effects on biomarkers associated with peptic ulcer, translating these findings to clinical practice requires additional research to fully understand the plant extract's bioavailability, efficacy, and safety in human populations. The fact that the study relies on animal models and specific biomarkers also emphasizes the need for comprehensive clinical studies to validate these findings in humans and establish a clear therapeutic protocol for using turnip to treat peptic ulcers. It is also possible to continue this research in the future by conducting further isolation studies in order to develop a highly bioactive molecule instead of using the whole plant's extract for treatment, and this is expected to increase efficacy and reduce side-effects.

CONCLUSION

This investigation firmly advocates for the therapeutic efficacy of TLP in addressing gastric ulcers, underscoring its significant potential. GC-MS analysis disclosed the presence of over 100 compounds, with 12 identified as necessary, demonstrating the natural extract's rich and intricate nature in offering numerous bioactive molecules

with profound health benefits. Particularly striking was the observed in reduction of ulcer sizes in rats treated with TLP, highlighting TLP's substantial therapeutic promise, especially in its capacity to modulate cytokine networks crucial in the inflammatory processes linked to gastric ulcers. These findings align with existing research on these cytokines' roles in inflammation and ulcer healing, reinforcing TLP's therapeutic value. Moreover, the observed decrease in caspase-3 and -9 levels post-TLP treatment points to its potential to reduce apoptosis, which is vital for repairing and regenerating ulcerated tissues.

Additionally, TLP's antibacterial properties against prevalent human bacterial pathogens add an extra dimension to its therapeutic profile, advocating for a comprehensive approach to ulcer treatment encompassing anti-inflammatory, immunomodulatory, and antibacterial actions. These outcomes not only bolster the accumulating evidence for the utility of botanicals in healthcare but also beckon further investigation into the precise mechanisms through which these bioactive components act and their broader medical applications. The study accentuates the promising role of TLP in developing new strategies for peptic ulcer therapy, offering insights that could lead to innovative, nature-based medical treatments.

Ethical Approval

Animal research commenced following approval from Research Ethical Committee at the University of Ha'il (Approval number: H-2024-102).

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Conflict of interest

The author declare that we have no conflict of interest.

REFERENCES

1. Rahamat Unissa S.; Mohd.Abdul H.; Aisha M.A.; Amal M.A.; Yusra H.A.; Asia A A. Alsagri.; Danah L.; Mohammed K.B.B. *Current Plant Biology*., **2024**.https://10.1016/J.CPB.2024.10042
2. Unissa Syed, R.; Moni, S. S.; Huwaimel, B.; Alobaida, A.; Abdulkareem Almarshdi, A.; S. Abouzied, A.; S. Abu Lila, A.; Abdallah, M. H.; Banu, H.; Abdul Hadi, M.; E. El-Horany, H.; Ibrahim Abdelwahab, S.; Taha, M. M. E., *Arab. J. Chem.*, **2022**, *15*(12), 104308. https://10.1016/j.arabjc.2022.104308
3. Syed RU.; Moni SS.; Break MKB.; Khojali WMA.; Jafar M.; Alshammari MD.; Abdelsalam K.; Taymour S.; Alreshidi KSM.; Elhassan Taha MM., *Antibiotics*., **2023**, *12*(7), 1157. https://doi.org/10.3390/antibiotics12071157
4. Salari, N.; Darvishi, N.; Shohaimi, S.; Bartina, Y.; Ahmadipanah, M.; Salari, H. R.; Mohammadi, M., *Indian J. Surg.*, **2021**, *84*(5), 913–921. https://10.1007/s12262-021-03189-z
5. Paul, S.; Geng, C.; Yang, T.; Yang, Y.; Chen, J., *J. Food Sci.*, **2018**, *84*(1), 19–30.https://10.1111/1750-3841.14417
6. Yang, J.; Lou, J.; Zhong, W.; Li, Y.; He, Y.; Su, S.; Chen, X.; Zhu, B., *Foods*., **2023**, *12*(17), 3195. https://10.3390/foods12173195
7. Gairola, S.; Sharma, J.; Bedi, Y. S., *J. Ethnopharmacol.*, **2014**, *155*(2), 925–986. https://10.1016/j.jep.2014.06.029
8. Javed, A.; Ahmad, A.; Nouman, M.; Hameed, A.; Tahir, A.; Shabbir, U., *Braz. J. Food Technol.*, **2019**, *22*. https://10.1590/1981-6723.25318
9. Cao, Q.; Wang, G.; Peng, Y., *Front. Nutr.*, **2021**, *8*, 721733. https://10.3389/fnut.2021.721733
10. Jideani, A. I. O.; Silungwe, H.; Takalani, T.; Omolola, A. O.; Udeh, H. O.; Anyasi, T. A., *Int. J. Food Prop.*, **2021**, *24*(1), 41–67. https://10.1080/10942912.2020.1866597
11. Raigond, P.; Kaundal, B.; Sood, A.; Devi, S.; Dutt, S.; Singh, B., *J. Food Compos. Anal.*, **2018**, *74*, 82–88.https://10.1016/j.jfca.2018.09.001
12. Chihoub, W.; Dias, M. I.; Barros, L.; Calhelha, R. C.; Alves, M. J.; Harzallah-Skhiri, F.; Ferreira, I. C. F. R., *Food Res. Int.*, **2019**, *126*, 108651. https://10.1016/j.foodres.2019.108651
13. Liberal, Â.; Fernandes, Â.; Polyzos, N.; Petropoulos, S. A.; Dias, M. I.; Pinela, J.; Petrovi, J.; Soković, M.; Ferreira, I. C. F. R.; Barros, L., *Molecules*., **2020**, *25*(23), 5606. https://10.3390/molecules25235606
14. Raza, Q.-U.-A.; Bashir, M. A.; Rehim, A.; Geng, Y.; Raza, H. M. A.; Hussain, S.; Ahmad, I.; Wasif, M., *Sustainability*., **2023**, *15*(15), 11851. https://10.3390/su151511851

15. Akasha. R; Moni S.K.S; Alshammari. M.D ; Alhaidan E.M.; Albsher.B.N; Alreshidi. K.M; Syed, R.U., *Orient. J. Chem.*, **2025**, *41*(4), DOI:
16. Gupta, S.; Nagar.L; Singla.M.; Smriti; Singh.S;Gupta.G; Bhojraj.S; U.S. Rahamat; Alshammari. A.D; D.A. Maali; Alshammari. N.N.O; Alshammari, A.N.O; Alhaidan.E.M., *Current Pharamceutical Design.,* **2025**, *31*, 1-17. htts://10.2174/0113816128362999250605093917
17. Syed, R. U.; Moni, S. S.; Alfaisal, R. H.; Alrashidi, R. H.; Alrashidi, N. F.; Wadeed, K. M.; Alshammary, F. N.; Habib, A. M.; Alharbi, F. M.; ur Rehman, Z.; Shamsher Alam, M.; Basode, V. K.; Abdulhaq, A. A., *Arab. J. Chem.*, **2022**, *15*(8), 104006. https://10.1016/j.arabjc.2022.104006
18. Lebda, M. A.; Mostafa, R. E.; Taha, N. M.; Abd El-Maksoud, E. M.; Tohamy, H. G.; Al Jaouni, S. K.; El-Far, A. H.; Elfeky, M. S., *Antioxidants.*, **2021**, *10*(11), 1836. DOI:10.3390/antiox10111836
19. Wahyuni, I. S.; Sufiawati, I.; Nittayananta, W.; Levita, J., *J. Inflamm. Res.*, **2022**, *15*, 2281–2294. https://10.2147/JIR.S359042
20. Hassanzadeh-Taheri, M.; Hassanpour-Fard, M.; Doostabadi, M.; Moodi, H.; Vazifeshenas-Darmiyan, K.; Hosseini, M., *J. Tradit. Complement. Med.*, **2017**, *8*(1), 178–183. https://10.1016/j.jtcme.2017.05.010
21. Rahman, M. S.; Jahan, N.; Rahman, S. M. A.; Rashid, M. A., *Bangladesh Med. Res. Counc. Bull.*, **2016**, *41*(3), 114–120.https://10.3329/bmrcb.v41i3.29886
22. Choi, E.-Y.; Hwang, H.-J.; Kim, I.-H.; Nam, T., *J. Food Chem. Toxicol.*, **2009**, *47*(1), 134–139. https://10.1016/j.fct.2008.10.026
23. Syed, R. U.; Moni, S. S.; Lila, A. S. A.; Abdallah, M. H.; Abouzied, A. S.; Banu, H.; Alreshidi, K. S. M.; Alrashidi, B. M. W.; Hadi, M. A.; El-Horany, H.; Abdelwahab, S. I.; Taha, M. M. E., *Life.,* **2022**, *12*(10), 1641. https://10.3390/life12101641
24. Syed, R. U.; Moni, S. S.; Nawaz, M.; Bin Break, M. K.; Khalifa, N. E.; Abdelwahab, S. I.; Alharbi, R. M.; Alfaisal, R. H.; Al Basher, B. N.; Alhaidan, E. M., *Pharmaceutics.*, **2023**, *15*(4), 1082. https://10.3390/pharmaceutics15041082
25. Prabhadevi, V.; Sahaya, S. S.; Johnson, M.; Venkatramani, B.; Janakiraman, N., *Asian Pac. J. Trop. Biomed.*, **2012**, *2*(2), S550–S554. https://10.1016/S2221-1691(12)60272-X
26. Saeed Kotb, S. S.; Ayoub, I. M.; El-Moghazy, S. A.; Singab, A. N. B., *Chem. Biodivers.*, **2020**, *17*(7). https://10.1002/cbdv.202000048
27. Alqahtani, F. Y.; Aleanizy, F. S.; Mahmoud, A. Z.; Farshori, N. N.; Alfaraj, R.; Al-Sheddi, E. S.; Alsarra, I. A., *Saudi J. Biol. Sci.*, **2019**, *26*(5), 1089–1092.https://10.1016/j.sjbs.2018.05.007
28. Ferdosi, M. F. H.; Kaleem Naseem, M. K.; Afzal, A.; Haider Khan, I.; Javaid, A., *Arab. J. Chem.*, **2023**, *16*(6), 104719. https://10.1016/j.arabjc.2023.104719
29. Zhu, S.; Jiao, W.; Xu, Y.; Hou, L.; Li, H.; Shao, J.; Zhang, X.; Wang, R.; Kong, D., *Life Sci.*, **2021**, *286*, 120046.https://10.1016/j.lfs.2021.120046
30. Aparna, V.; Dileep, K. V.; Mandal, P. K.; Karthe, P.; Sadasivan, C.; Haridas, M., *Chem. Biol. Drug Des.*, **2012**, *80*(3), 434–439. https://10.1111/j.1747-0285.2012.01418.x
31. Mantovani, A.; Dinarello, C. A.; Molgora, M.; Garlanda, C., *Immunity.*, **2019**, *50*(4), 778–795. https://10.1016/j.immuni.2019.03.012
32. Zhao, H.; Wu, L.; Yan, G.; Chen, Y.; Zhou, M.; Wu, Y.; Li, Y., *Signal Transduct. Target Ther.,* **2021**, *6*(1), 263. https://10.1038/s41392-021-00658-5
33. Petrasek, J.; Bala, S.; Csak, T.; Lippai, D.; Kodys, K.; Menashy, V.; Barrieau, M.; Min, S.-Y.; Kurt-Jones, E. A.; Szabo, G., *J. Clin. Invest.*, **2012**, *122*(10), 3476–3489. https://10.1172/JCI60777
34. Ruder, B.; Atreya, R.; Becker, C., *Int. J. Mol. Sci.*, **2019**, *20*(8), 1887.https://10.3390/ijms20081887
35. Datta De, D.; Bhattacharjya, S.; Maitra, M.; Datta, A.; Choudhury, A.; Dhali, G. K.; Roychoudhury, S., *PLoS One.*, **2011**, *6*(3), e14775. https://10.1371/journal.pone.0014775
36. Bakkari, M. A.; Moni, S. S.; Alshammari, A.; Sultan, M. H.; Madkhali, O. A.; Almoshari, Y.; Alam, M. F.; Elmobark, M. E., *Vaccines.*, **2023**, *11*(2), 457. https://10.3390/vaccines11020457
37. Beck, P. L.; Cotton, J. A.; Platnich, J. M.; Muruve, D. A.; Buret, A. G.; Jijon, H., *Int. J. Interferon Cytokine Mediat. Res.*, **2016**, *13*, https://10.2147/IJICMR.S63682
38. David, J. M.; Dominguez, C.; Hamilton, D. H.; Palena, C., *Vaccines.*, **2016**, *4*(3), 22. https://10.3390/vaccines4030022

39. Kinoshita, H.; Hirata, Y.; Nakagawa, H.; Sakamoto, K.; Hayakawa, Y.; Takahashi, R.; Nakata, W.; Sakitani, K.; Serizawa, T.; Hikiba, Y.; Akanuma, M.; Shibata, W.; Maeda, S.; Koike, K., *PLoS One*., **2013**, *8*(4), e60914. https://10.1371/journal.pone.0060914
40. Tanaka, T.; Narazaki, M.; Kishimoto, T., *Cold Spring Harb. Perspect. Biol*., **2014**, *6*(10), a016295.https://10.1101/cshperspect.a016295
41. Armah, F. A.; Henneh, I. T.; Alake, J.; Ahlidja, W.; Amoani, B.; Ofori, E. G.; Asante-Kyei, B.; Temitayo, G. I.; Adokoh, C. K., *Biomed. Res. Int*., **2021**, *2021*, 6694572. https://10.1155/2021/6694572
42. Beserra, A. M. S. e S.; Calegari, P. I.; Souza, M. do C.; dos Santos, R. A. N.; Lima, J. C. da S.; Silva, R. M.; Balogun, S. O.; Martins, D. T. de O., *J. Agric. Food Chem*., **2011**, *59*(13), 6957–6965. https://10.1021/jf2003267
43. Lee, J.-Y.; Lee, H.-J.; Jeong, J.-A.; Jung, J.-W., *Orient. Pharm. Exp. Med*., **2010**, *10*(1), 37–43. https://10.3742/OPEM.2010.10.1.037
44. Zhao, G.; Etherton, T. D.; Martin, K. R.; Gillies, P. J.; West, S. G.; Kris-Etherton, P. M., *Am. J. Clin. Nutr*., **2007**, *85*(2), 385–391. https://10.1093/ajcn/85.2.385
45. Morandi, F.; Bensa, V.; Calarco, E.; Pastorino, F.; Perri, P.; Corrias, M. V.; Ponzoni, M.; Brignole, C., *Nutrients.,* **2021**, *13*(7), 2178. https://10.3390/nu13072178
46. Yaguchi, T.; Fujikawa, H.; Nishizaki, T., *Neurochem. Res*., **2010**, *35*(5), 712–717. https://10.1007/s11064-010-0124-4
47. Alotaibi, B.; Mokhtar, F. A.; El-Masry, T. A.; Elekhnawy, E.; Mostafa, S. A.; Abdelkader, D. H.; Elharty, M. E.; Saleh, A.; Negm, W. A., *J. Inflamm. Res*., **2021**, *14*, 7411–7430. https://10.2147/JIR.S345780
48. Kusumah, D.; Wakui, M.; Murakami, M.; Xie, X.; Yukihito, K.; Maeda, I., *Biosci. Biotechnol. Biochem.,* **2020**, *84*(6), 1285–1290.https://10.1080/09168451.2020.1731299
49. Wang, F.; Guo, Y.; Cao, Y.; Zhang, C., *J. Ocean Univ. China.,* **2022**, *21*(6), 1615–1621. https://10.1007/s11802-022-5047-6
50. Shaaban, M. T. ; Ghaly, M. F.; Fahmi, S. M., *J. Basic Microbiol*., **2021**, *61*(6), 557–568. https://10.1002/jobm.202100061