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Anti-ulcer potential of *Fioria vitifolia*: Phytochemical characterization by GC-MS and *in silico* molecular docking studies against gastric targets

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Abstract

Fioria vitifolia (L.) Mattei (family Malvaceae) is a medicinal herb traditionally used for its analgesic, anti-inflammatory, hepatoprotective, and gastrointestinal benefits. The present study aimed to evaluate its anti-ulcer potential through phytochemical profiling and in silico molecular docking approaches. Whole plant parts, including leaves, stems, roots, and flowers, were extracted using 99% ethanol by maceration for 96 hours, yielding 2.27% w/w extract. The ethanolic extract was analyzed using Gas Chromatography-Mass Spectrometry (GC-MS), which identified nine phytoconstituents. Among them, 5-(4-Nitrobenzylidene)-3-(m-Toluoyl) Rhodanine (38.61%), Ribitol 1,3:2,4-Di-O-Benzylidene (14.54%), and Eicosane, 1-Iodo- (14.30%) were the major compounds detected.

To investigate their anti-ulcer activity, molecular docking studies were carried out using AutoDock Vina against six ulcer-related therapeutic targets: H+/K+-ATPase (PDB: 5YLU), Histamine H2 receptor (PDB: 9IXJ), Pig Gastric H,K-ATPase (PDB: 2XZB), Cyclooxygenase-2 (PDB: 3LN1), Beta-1 Adrenergic Receptor (PDB: 2VT4), and Carbonic Anhydrase-II (PDB: 1A42). The compound 5-(4-Nitrobenzylidene)-3-(m-Toluoyl) Rhodanine exhibited the strongest overall binding affinity, with a maximum docking score of −8.057 kcal/mol against 2XZB. Ribitol 1,3:2,4-Di-O-Benzylidene showed notable affinity toward Carbonic Anhydrase-II, with a docking score of −8.181 kcal/mol.

Preliminary phytochemical screening confirmed the presence of alkaloids, steroids, phenols, tannins, and terpenoids. These findings support the potential of *F. vitifolia* as a promising natural source for the development of anti-ulcer agents.

Keywords: *Fioria vitifolia*, GC-MS, molecular docking, anti-ulcer, peptic ulcer disease, proton pump inhibitor, COX-2, Malvaceae, phytochemistry.

1. Introduction

Pharmaceutical chemistry bridges synthetic organic chemistry and biological pharmacology, aiming to generate novel chemical entities with improved therapeutic profiles. Within this discipline, natural product-guided drug discovery remains one of the most productive strategies, with plant-derived compounds contributing substantially to modern pharmacopoeias [1,9]. Plants have been the cornerstone of traditional medicine across diverse global cultures, and their phytochemical constituents represent an evolving reservoir for anti-infective, analgesic, anti-inflammatory, and gastroprotective drug leads[15]. Peptic Ulcer Disease (PUD) is a globally significant gastroduodenal disorder characterized by a breach in the mucosal lining that penetrates the muscularis mucosae, arising from an imbalance between aggressive factorshydrochloric acid, pepsin, Helicobacter pylori, and non-steroidal anti-inflammatory drugs (NSAIDs)and defensive mucosal mechanisms [22,23]. Despite therapeutic advancements including proton pump inhibitors (PPIs) and H. pylori eradication regimens, PUD continues to impose a substantial clinical and economic burden, with U.S. healthcare costs reaching up to $10 billion annually. Furthermore, long-term NSAIDinduced mucosal damage and the rising prevalence of antibiotic-resistant H. pylori strains underline the urgent need for new therapeutic alternatives [17].

India's biodiversity, encompassing Ayurvedic, Siddha, and Unani systems, offers a vast and largely unexplored reservoir for anti-ulcer drug discovery [15]. *Fioria vitifolia* (L.) Mattei (synonym: *Hibiscus vitifolius* L.), a perennial herb of the family Malvaceae, is a medicinally important species distributed across tropical and subtropical Asia, Africa, and Oceania [3]. Historically, it has been employed in traditional practice for its analgesic, sedative, anti-inflammatory, and hepatoprotective properties, and ethnopharmacological reports confirm its utilization for treating bacterial infections and inflammation-related gastric distress [2, 16].

Phytochemical investigations have revealed a diverse metabolite profile in *F. vitifolia*, including sterols, flavonoids (notably gossypin), triterpenes (friedelin, epifriedelinol), alkaloids, and saponins [3]. Significantly, the plant has been identified as a natural source of plant protease inhibitors (PIs), with a trypsin inhibition rate of up to 91.79%a property of particular relevance to anti-ulcer research as it may mitigate proteolytic degradation of the gastric mucosa [4]. To rigorously characterize and validate the bioactive phytoconstituents of this plant, modern analytical and computational tools are essential[15]. Gas Chromatography-Mass Spectrometry (GC-MS) provides the resolution and identification power necessary for complex botanical extracts, while molecular docking enables structure-based prediction of binding affinity at specific ulcer-related protein targets [8, 9, 11].

The present study integrates these two methodological pillarsanalytical phytochemistry via GC-MS and computational biology via molecular dockingto systematically evaluate the anti-ulcer potential of F. vitifolia. Nine bioactive compounds identified from the ethanolic extract were subjected to in silico docking against six clinically relevant gastric targets. The results presented here serve as a foundation for the rational development of this species as a source of novel gastroprotective drug candidates.

2. Background: Peptic ulcer disease

2.1 EpidemiologyandGlobal Burden

Peptic Ulcer Disease (PUD) historically affected approximately 10% of the U.S. population at some point in their lives, though modern eradication of H. pylori has reduced overall incidence. A critical demographic shift is underway: PUD prevalence and complications are rising among the elderly, driven by chronic NSAID use for cardiovascular and musculoskeletal conditions [24, 25]. Annual U.S. healthcare expenditures for PUD remain as high as $10 billion, illustrating the persistent socioeconomic burden of this disease.

2.2 Pathophysiology

Ulceration represents the ultimate consequence of disequilibrium between the aggressive factors (hydrochloric acid, pepsin, H. pylori toxins, NSAIDs) and the multi-layered mucosal defense system [23]. The first defense tierthe mucus-bicarbonate gelmaintains a pH gradient from luminal pH 1.5 to a near-neutral pH ~7.0 at the epithelial surface [22, 24]. The epithelial layer, reinforced by tight junctions and Trefoil Factor Peptides enabling rapid restitution, constitutes the second tier, while the post-epithelial microvascular network delivers nutrients and neutralizes back-diffusing protons [23, 25].

2.3 Primary Etiology

H. pylori remain the leading cause of PUD globally. Its survival mechanism relies on urease-mediated conversion of urea to ammonia, neutralizing the gastric microenvironment. Once established, the organism induces chronic active inflammation, disrupts gastrin regulation leading to acid hypersecretion, and directly reduces mucus hydrophobicity [16, 22]. NSAIDs constitute the second major etiological factor through dual mechanisms: direct topical mucosal injury and systemic COX-1 inhibition that depletes cytoprotective prostaglandins [17, 24].

Psychological and physiological stress further exacerbate mucosal vulnerability through HPA axis activation and reduced mucosal perfusion [23].

2.4 Therapeutic Landscape and Unmet Need

PPIs, acting by irreversible binding to the H+/K+-ATPase parietal cell pump, are the cornerstone of modern PUD therapy [17, 22]. Standard H. pylori eradication employs triple therapy (PPI + amoxicillin + clarithromycin) for 10–14 days, with quadruple regimens used in high-resistance regions [17]. Despite these advances, therapeutic gaps persist: NSAID-associated gastric damage often continues despite PPI prophylaxis, antibiotic resistance rates for clarithromycin are rising globally, and long-term PPI use carries risks of hypomagnesemia, C. difficile infection, and kidney disease. These limitations provide strong justification for the development of plant-derived, multi-target anti-ulcer agents [22].

3. Plant Profile: *Fioria vitifolia*

3.1 Taxonomy and Nomenclature

Fioria vitifolia (L.) Mattei (synonym: *Hibiscus vitifolius* L.) is classified within the family Malvaceae, subfamily Malvoideae, identified through specific morphological attributes including the epicalyx, staminal tube, and trichomes [3, 5]. Commonly known as Grape-leaved Mallow or Wild Okra, it is distributed natively across tropical Asia (India, Thailand, Myanmar, southern China), Africa (Ethiopia, Kenya, Angola, Nigeria), and northern Australia, with introduced populations in the Caribbean [6, 15].

3.2 Morphology

F. vitifolia is a perennial herb or subshrub growing to approximately 1 meter in height. The stem is greenish-yellow and terete, characterized by stellate pubescence. Leaves are simple, alternate, broadly ovate to orbicular, with palmately 3–5 lobed configuration, cordate base, and serrate-dentate margins. Flowers are large, solitary, and axillary, featuring a pale-yellow

corolla with a distinctive dark purple/chocolate Center. The epicalyx consists of 8–10 free linear segments (4–8 mm); the staminal tube is Monadelphous, 14–15 mm long, and deep purple [2, 5]. Pollen grains are spheroidal with ~54 pores and an echinate exine (spines 20–25 µm), measuring 105–113 µm in diameter [6]. Fruits are depressed globose capsules (~1.5 cm) bearing reniform, tubercles seeds.

3.3 Phytochemistry and Bioactivity

The plant's pharmacological properties are underpinned by a rich metabolite profile. Major isolated compounds include the triterpenes friedelin and epifriedelinol, and the flavonoid glycoside gossypin [3, 13]. Broad phytochemical screening has confirmed phenolics, tannins, saponins, alkaloids, steroids, triterpenoids, and amino acids [2, 3]. Reported pharmacological activities span central analgesia, anti-inflammation, antipyresis, hepatoprotection, antioxidant activity, and trypsin inhibitionall directly relevant to gastric ulcer pathophysiology [2, 3, 4].

4. Materials and Methods

4.1 Plant Collection and Authentication

Fioria vitifolia was collected from wild sources in Namakkal District, Tamil Nadu, India. Plant material was washed thoroughly under running tap water and rinsed thrice with distilled water. All partsflower, root, stem, and leafwere shade-dried to constant weight and powdered using a mechanical blender. [Note: Ethics committee permission and institutional approval statement to be included as per IJPR requirements.]

4.2 Extraction by Maceration

Maceration is a passive diffusion-based extraction technique in which powdered plant material is immersed in a solvent over an extended period at room temperature, enabling solvent penetration, solute dissolution, and diffusion-driven mass transfer. This method was selected to preserve thermolabile bioactive constituents susceptible to degradation under heat-based extraction methods. Powdered plant parts were extracted using 99% ethanol as solvent in the following ratios (Table 1):

Plant Part	Powder Weight (g)	Ethanol Volume (mL)
Flower	8	100
Root	12	100
Stem	12	100
Leaf	12	100
Total	44	400

Table 1: Maceration Extraction Parameters for *F. vitifolia*

Each sample was placed in a sealed container and macerated for 96 hours with manual shaking every 4 hours at 35–45°C. Post-maceration, plant debris was removed using muslin cloth; the filtrate was further clarified through Whatman No. 1 filter paper. The combined filtrate (253 mL recovered; recovery efficiency = 63.25%) was evaporated to yield a dry ethanolic extract. The percentage yield was calculated as 2.27% w/w (1.0 g dry extract / 44 g total powder × 100).

4.3 Preliminary Phytochemical Screening

The extract was subjected to standard phytochemical tests for alkaloids (Dragendorff's, Mayer's, Wagner's, and Hager's reagents), phenols (ferric chloride test), steroids (Salkowski test), tannins, and terpenoids. Results were recorded as positive (+) or negative (−).

4.4 GC-MS Analysis

GC-MS analysis was performed on a PerkinElmer GC 2400 gas chromatograph coupled with an MS 2400SQT single-quadrupole mass spectrometer (GC manufactured in Singapore; MS in USA). Chromatographic separation was achieved on an Elite-5MS capillary column (30 m × 0.25 mm ID × 0.25 µm film thickness; 5% diphenyl/95% dimethylpolysiloxane stationary phase). Helium served as the carrier gas with a 20:1 split ratio and 1.0 µL injection volume. Inlet, transfer line, and source temperatures were maintained at 250°C. Mass spectrometric detection used electron impact (EI+) ionization with a scan range of m/z 50–300. The Total Ion Chromatogram (TIC) was 1.38×10^8. Data processing was performed using PerkinElmer SimplicityChrom™ CDS software, and compound identification was achieved by comparing fragmentation patterns with the NIST MS Search v3.0 library (>394,000 spectra).

4.5 Molecular Docking Protocol

Structures of nine GC-MS-identified compounds were retrieved by PubChem ID and converted to SMILES format. Three-dimensional ligand structures were prepared by generating 3D coordinates, assigning bond orders, enumerating tautomers, and determining protonation states at physiological pH. Six gastric ulcer target proteins were selected from the RCSB Protein Data Bank (PDB) based on their established roles in ulcer pathogenesis (Table 2):

Target Protein	PDB ID	Mechanism of Relevance
H+/K+-ATPase (Proton Pump)	5YLU	Direct inhibition of gastric acid secretion
Histamine H2R (Gs complex)	9IXJ	Blocking histamine-induced acid release
Pig Gastric H,K-ATPase with SCH28080	2XZB	Anti-secretory validation
Cyclooxygenase-2 (COX-2)	3LN1	Reducing gastric mucosal inflammation
Beta-1 Adrenergic Receptor	2VT4	Modulating stress-induced gastric lesions
Carbonic Anhydrase-II	1A42	Regulating bicarbonate for mucosal cytoprotection

Table 2: Gastric Ulcer Target Proteins Used in Molecular Docking

Protein structures were prepared by removing water molecules and co-crystallized ligands, adding missing hydrogens, and assigning partial charges. Docking was performed using Auto Dock Vina with sampling exhaustivity set to 4. Results were ranked by calculated binding affinity (kcal/mol), with more negative values indicating higher predicted binding strength [9].

5. Results and Discussion

5.1 Preliminary Phytochemical Screening

Preliminary phytochemical screening of the ethanolic extract of *F. vitifolia* confirmed the presence of alkaloids, steroids, phenols, tannins, and terpenoids (Table 3). The detection of these broad phytochemical classes corroborates prior reports [2, 3] and provides a chemical basis for the plant's traditional anti-inflammatory and gastroprotective uses. Alkaloids are known to exert anti-secretory effects; phenols and tannins confer astringent and mucosal-protective properties; while terpenoidsparticularly the isolated friedelin and epifriedelinolare recognized anti-inflammatory agents.

S. No.	Test	Observation
1	Alkaloids (Dragendorff's, Mayer's, Wagner's, Hager's tests)	Positive (+)
2	Steroids (Salkowski test)	Positive (+)
3	Phenols (Ferric Chloride test)	Positive (+)
4	Tannins	Positive (+)
5	Terpenoids	Positive (+)

Table 3: Preliminary Phytochemical Screening Results of F. vitifolia Ethanolic Extract

5.2 GC-MS Phytochemical Profiling

GC-MS analysis of the ethanolic extract resolved nine distinct peaks, collectively accounting for 100% of the Total Ion Chromatogram (TIC = 1.38 × 10^8). Compound identification against the NIST v3.0 library yielded nine bioactive constituents (Table 4). The dominant peak at retention time 2.317 min was attributed to 5-(4-Nitrobenzylidene)-3-(m-Toluoyl) Rhodanine ($C_{18}H_{12}O_4N_2S_2$; MW 384; area 38.61%), a rhodanine derivative. Rhodanine-based scaffolds have been reported as inhibitors of multiple enzyme classes relevant to infectious and inflammatory diseases [9]. Ribitol, 1,3:2,4-Di-O-Benzylidene (RT 4.177 min; area 14.54%) is a cyclic acetal derivative of ribitol, a naturally occurring sugar alcohol associated with anti-inflammatory and cytoprotective biological activity. Eicosane, 1-Iodo- (RT 24.547 min; area 14.30%) is a long-chain iodoalkane; its presence reflects the lipophilic composition of the extract. Hexadecanoic Acid, Ethyl Ester (ethyl palmitate; RT 19.061 min; area 4.53%) is a fatty acid ester with known membrane-stabilizing properties. The chalcone derivative 2,4-Dimethoxy-2'-Ethoxychalcone (RT 7.354 min; area 0.71%) belongs to a class of flavonoid precursors with established COX inhibitory and anti-ulcer activity [13].

S.No.	Compound Name	RT (min)	Formula	MW	Area %
1	5-(4-Nitrobenzylidene)-3-(m-Toluoyl) Rhodanine	2.317	$C_{18}H_{12}O_4N_2S_2$	384	38.61
2	Methyl 3-Methoxy-3-Phenylpropanoate	2.492	$C_{11}H_{14}O_3$	194	7.26
3	Ribitol, 1,3:2,4-Di-O-Benzylidene	4.177	$C_{19}H_{20}O_5$	328	14.54
4	Cyanamide, Di-2-Propenyl-	6.866	$C_7H_{10}N_2$	122	1.56
5	2,4-Dimethoxy-2'-Ethoxychalcone (Isomer 2)	7.354	$C_{19}H_{20}O_4$	312	0.71
6	Silane, 2-Butenylmethoxymethylphenyl-	7.854	$C_{12}H_{18}Osi$	206	14.84
7	Methanone, (2-Methoxyphenyl)(4-Methylphenyl)	10.418	$C_{15}H_{14}O_2$	226	3.65
8	Hexadecanoic Acid, Ethyl Ester	19.061	$C_{18}H_{36}O_2$	284	4.53
9	Eicosane, 1-Iodo-	24.547	$C_{20}H_{41}I$	408	14.30

Table 4: GC-MS Identified Bioactive Compounds from F. vitifolia Ethanolic Extract

Chromatogram Plot

File: C:\TurboMass\GCMS-CCE.pro\Data\20022026_05.raw
Acquired: 20-Feb-26 05:00:50 PM
Description: Mani
GC/MS Method: BASICANALYSIS METHOD
Sample ID: HV

Printed: 23-Feb-26 11:27 AM

Page 1 of 1
Vial Number: 17

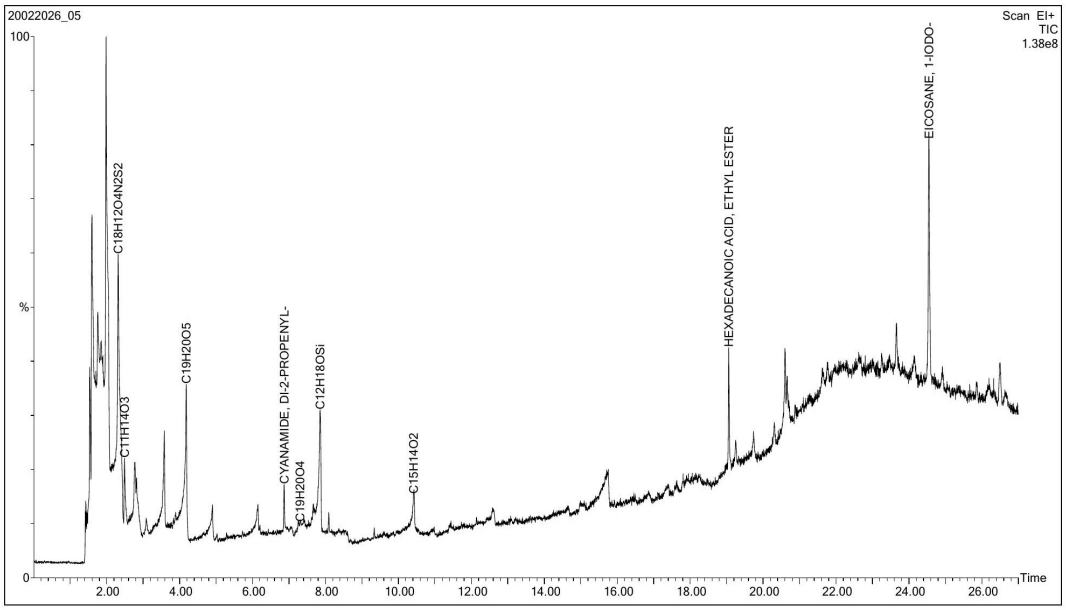


Figure 1: GC-MS Chromatogram Plot of F. vitifolia Ethanolic Extract (PerkinElmer GC 2400; TIC = 1.38 × 10⁸)

Quantitation Report

File Name: C:\TurboMass\GCMS-CCE.pro\Data\20022026_05.raw
Sample ID: HV
Description: Mani
Inject Date/Time: February 20, 2026 5:00:50 PM
Instrument: 2400-GCMS
GC Method: BASICANALYSIS METHOD
Quantify Method: HV MANI
Calibration File: METH1
GC Column: ID: 250 um
Matrix:
Sample wt/vol: 1.000
Level (low/med):

Operator:
Vial: 17
Tune File: Jan 2026
MS Method: BASICANALYSIS METHOD
Last Updated: February 23, 2026 11:25:18 AM
Last Updated: March 26, 2025 5:44:45 PM
Date Received:
Dilution Factor: 0.0
% Moisture: 0.0 not decanted 0

	Internal Standards	RT	m/z	Area		RT Dev	Man

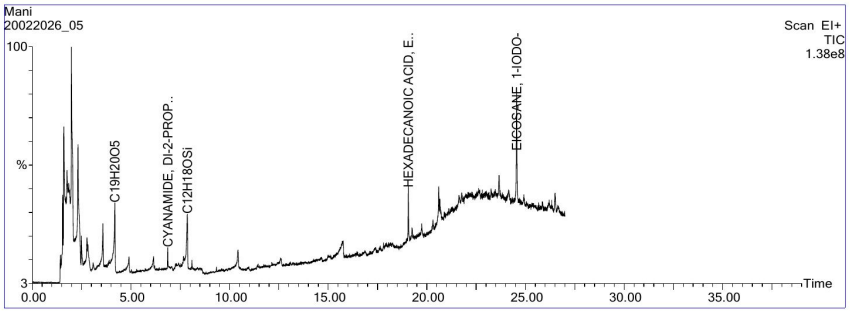
	Surrogate Compounds	RT	m/z	Area	%Recovery	RT Dev	Man

	Target Compounds	RT	m/z	Area		Rpt Limit	Man
1	C18H12O4N2S2	2.32	119	1,303,063.0		0.00	
2	C11H14O3	2.49	121	244,865.5		0.00	
3	C19H20O5	4.18	149	490,812.3		0.00	
4	CYANAMIDE, DI-2-PROPENYL-	6.87	81	52,676.6		0.00	
5	C19H20O4	7.30	67	23,912.3		0.00	
6	C12H18OSi	7.85	151	500,790.9		0.00	
7	C15H14O2	10.42	225	123,248.8		0.00	
8	HEXADECANOIC ACID, ETHYL ESTER	19.06	88	152,989.3		0.00	
9	EICOSANE, 1-IODO-	24.55	57	482,612.6		0.00	

Figure 2: Quantitation Report – Target Compounds Identified from GC-MS Analysis

Quantitative Report

File: C:\TurboMass\GCMS-CCE.pro\Data\20022026_05.raw
Acquired: 20-Feb-26 05:00:50 PM Printed: 23-Feb-26 11:28 AM
Description: Mani
GC/MS Method: GC: BASICANALYSIS METHOD MS: BASICANALYSIS METH Page 1 of 1
Sample ID: HV Vial Number: 17



#	Name	RT	Area	Area %	Height
1	C18H12O4N2S2	2.317	1,303,063.0	38.61	18,780,176.0
2	C11H14O3	2.492	244,865.5	7.26	4,773,064.0
3	C19H20O5	4.177	490,812.3	14.54	8,068,899.0
4	CYANAMIDE, DI-2-PROPENYL-	6.866	52,676.6	1.56	1,248,109.0
5	C19H20O4	7.304	23,912.3	0.71	267,955.0
6	C12H18OSi	7.854	500,790.9	14.84	8,326,923.0
7	C15H14O2	10.418	123,248.8	3.65	1,916,645.0
8	HEXADECANOIC ACID, ETHYL ESTER	19.061	152,989.3	4.53	3,595,091.0
9	EICOSANE, 1-IODO-	24.547	482,612.6	14.30	9,544,421.0
		TOTALS	3,374,971.3	100.0	56,521,283.0

Inst(2400-GCMS) ACQUISITION PARAMETERS
Refer to method interface to review Oven steps, Inj man.=50°C, Volume=1.00 µL, Split=:1, Carrier Gas=, Solvent Delay= min,
Transfer Temp=250°C, Source Temp=250°C, Scan: 50 to 300Da, Column 15.0m x 250µm

Figure 3: Quantitative Report with Peak Area Summary and Acquisition Parameters

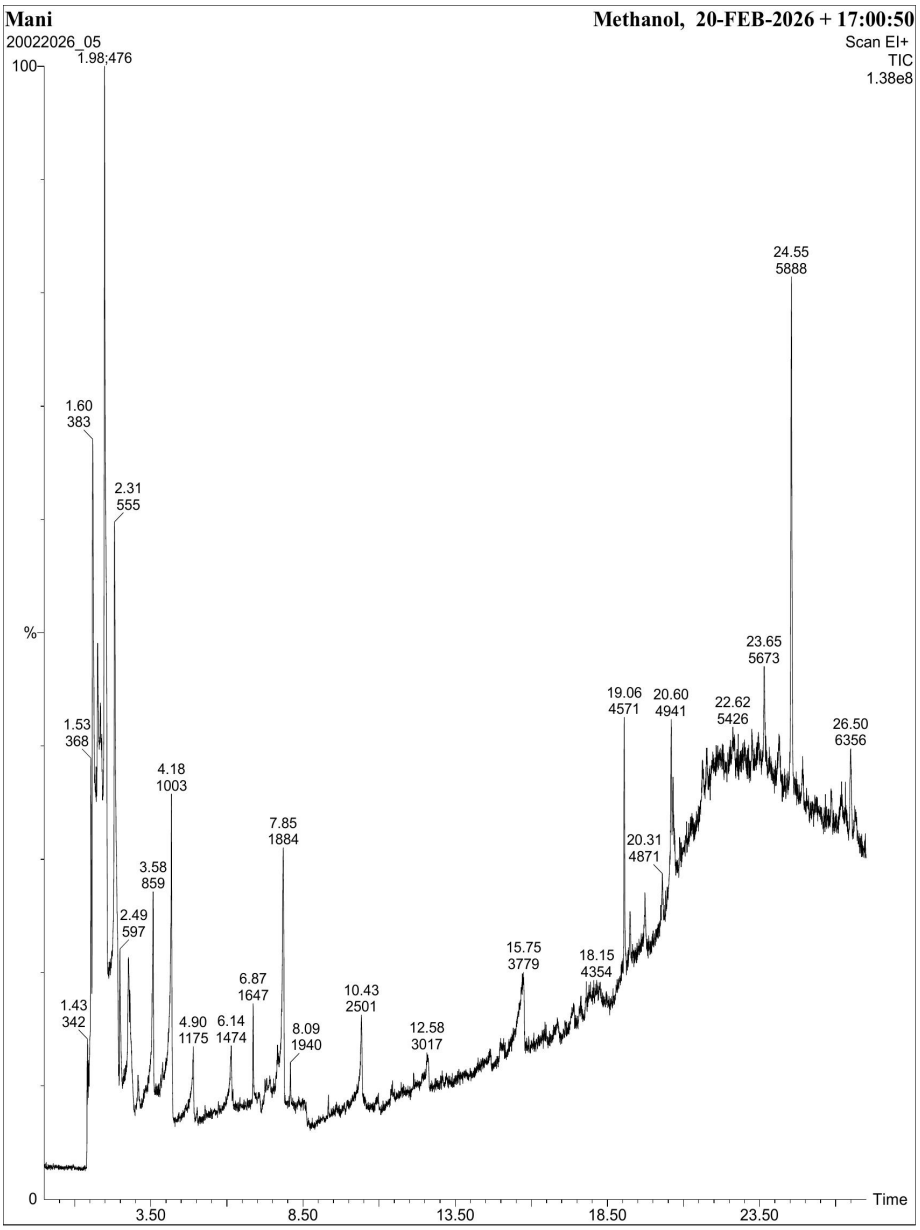


Figure 4: Total Ion Chromatogram (TIC) of F. vitifolia Ethanolic Extract – Scan EI+, TIC 1.38e8 (20-Feb-2026)

5.3 Molecular Docking Results

The binding affinities (kcal/mol) of all nine compounds across the six ulcer-related protein targets are presented in Table 5. Lower (more negative) values indicate stronger predicted binding interactions.

Compound	5YLU	9IXJ	2XZB	3LN1	2VT4	1A42
5-(4-Nitrobenzylidene)-3-(m-Toluoyl) Rhodanine	−7.207	−6.598	−8.057	−6.692	−6.680	−7.341
Methyl 3-Methoxy-3-Phenylpropanoate	−5.360	−4.616	−5.308	−4.557	−4.017	−5.283
Ribitol, 1,3:2,4-Di-O-Benzylidene	−7.429	−6.409	−7.984	−6.961	−5.920	−8.181
Cyanamide, Di-2-Propenyl-	−3.967	−3.045	−3.838	−3.164	−3.880	−3.494
2,4-Dimethoxy-2'-Ethoxychalcone	−6.390	−5.480	−6.845	−5.488	−5.308	−5.464
Silane, Butenylmethoxymethylphenyl-	−5.657	−4.348	−5.375	−4.103	−4.294	−4.121
Methanone, (2-Methoxyphenyl)(4-Methylphenyl)	−6.783	−5.299	−7.059	−5.273	−4.729	−5.272
Hexadecanoic Acid, Ethyl Ester	−5.030	−4.305	−5.021	−3.628	−3.311	−3.237
Eicosane, 1-Iodo-	−4.749	−3.954	−5.182	−3.703	−3.264	−3.577

Table 5: Calculated Binding Affinities (kcal/mol) of GC-MS Compounds Against Gastric Ulcer Targets

The rhodanine derivative 5-(4-Nitrobenzylidene)-3-(m-Toluoyl) Rhodanine emerged as the most broadly active lead compound, exhibiting the highest affinity for Pig Gastric H,K-ATPase (PDB: 2XZB; −8.057 kcal/mol) and favorable affinities across all six targets, ranging from −6.598 to −8.057 kcal/mol. The 2XZB structurean H,K-ATPase co-crystallized with the competitive inhibitor SCH28080—represents the classical binding site for proton pump inhibitors. The strong docking score of this rhodanine compound suggests potential as a novel class of anti-secretory agent, acting via a mechanism analogous to competitive pump inhibition. Its affinity for Carbonic Anhydrase-II (1A42; −7.341 kcal/mol) further implicates a cytoprotective mechanism, as CA-II regulates bicarbonate secretion essential for mucosal pH buffering [9, 22]. Ribitol, 1,3:2,4-Di-O-Benzylidene demonstrated the highest binding affinity observed in this study for Carbonic Anhydrase-II (1A42; −8.181 kcal/mol), surpassing the rhodanine lead. Its strong interactions with both 2XZB (−7.984 kcal/mol) and COX-2 (3LN1; −6.961 kcal/mol) establish this compound as a multi-mechanistic anti-ulcer candidate: simultaneously supporting mucosal bicarbonate defense, inhibiting the gastric proton pump, and reducing prostaglandin-mediated inflammation. The chalcone 2,4-Dimethoxy-2'-Ethoxychalcone (2XZB; −6.845 kcal/mol) also demonstrated moderate multi-target affinity consistent with chalcones' known COX and acid-pump inhibitory activity [13]. Methanone, (2-Methoxyphenyl) (4-Methylphenyl) demonstrated selective binding to 2XZB (−7.059 kcal/mol) and 5YLU (−6.783 kcal/mol), both anti-secretory targets, suggesting a targeted anti-acid mechanism.

Compounds with the weakest binding affinities Cyanamide, Di-2-Propenyl- (best score: −3.967 kcal/mol) and Eicosane, 1-Iodo- (best: −5.182 kcal/mol)are unlikely to contribute meaningfully to the anti-ulcer bioactivity of the extract and may represent minor or inert fractions [11]. Overall, the docking results establish a clear structure-activity hierarchy within the F. vitifolia extract, with the rhodanine and ribitol derivatives constituting the priority lead scaffolds for further pharmacological investigation [9, 14].

6. Conclusion

The present study represents a comprehensive analytical and computational evaluation of *Fioria vitifolia* as a source of novel anti-ulcer therapeutics. GC-MS profiling of the ethanolic whole-plant extract identified nine bioactive constituents, prominently featuring 5-(4-Nitrobenzylidene)-3-(m-Toluoyl) Rhodanine (38.61% area) and Ribitol, 1,3:2,4-Di-O-Benzylidene (14.54% area), alongside a chalcone, a fatty acid ester, and several aromatic derivatives.

Molecular docking investigations against six clinically validated gastric ulcer targets revealed that the rhodanine lead achieves a peak binding affinity of −8.057 kcal/mol against the Pig Gastric H,K-ATPase (2XZB)—comparable to established competitive pump inhibitors—while demonstrating broad-target activity across all six receptors. Ribitol Di-O-Benzylidene exhibited the highest affinity recorded in this study for Carbonic Anhydrase-II (−8.181 kcal/mol), implicating a potent cytoprotective mechanism. These computational findings are fully corroborated by the positive preliminary phytochemical profile and are consistent with the extensive pharmacological literature reporting analgesic, anti-inflammatory, and gastroprotective activities for *F. vitifolia* extracts [2, 3].

In conclusion, this study provides a robust scientific validation for the ethnopharmacological uses of *F. vitifolia* and identifies specific molecular scaffolds—rhodanine and ribitol derivatives—as priority candidates for rational anti-ulcer drug development. Future studies should focus on in vitro enzyme inhibition assays for H,K-ATPase and CA-II, in vivo gastric ulcer models (e.g., HCl/ethanol and pylorus ligation), ADMET profiling of the lead compounds, and clinical-grade standardization of the extract. The results collectively establish *F. vitifolia* as a scientifically credible and pharmacologically promising reservoir for novel gastroprotective agents.

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References

1. Reddy KN, Subbaraju GV, Reddy CS, Raju VS. Ethnoveterinary medicine for treating livestock in Eastern Ghats of Andhra Pradesh. Indian Journal of Traditional Knowledge. 2006;5(3):368–72.
2. Mahalakshmi K, Senthilkumar N, Suresh A, Parthiban KG. Analgesic activity of methanolic extract of Fioria vitifolia (L.).
3. Mahalakshmi K, Natesan SK, Parthiban KG, Sekar G. Anti-inflammatory and antipyretic activity of Fioria vitifolia. International Current Pharmaceutical Journal. 2013;2(7):119–21.
4. Sreejina SK, Kannan VM. Exploring plant seed extracts as potential sources of trypsin inhibitors for insect pest management. Entomon. 2025;50(1):107–12.
5. Naskar S, Mandal R. Characterization of common members of Malvaceae based on selective morphological attributes. Indian Journal of Plant Sciences. 2014;4(3):79–86.
6. Linn SS. Pollen morphology of ten species in Nyaung Myint Village, Meiktila Township [Doctoral dissertation]. MERAL Portal.
7. Imath M, Ragavendran C, Kamaraj C, Raja RK. Eco-friendly synthesis of chitosan NPs from Fioria vitifolia (L.): antibacterial, antioxidant, cytotoxicity and zebrafish embryo safety evaluation. Results in Engineering. 2025;25:104076.
8. Shoichet BK, Kuntz ID. Matching chemistry and shape in molecular docking. Protein Engineering, Design and Selection. 1993;6(7):723–32.
9. Ferreira LG, Dos Santos RN, Oliva G, Andricopulo AD. Molecular docking and structure-based drug design strategies. Molecules. 2015;20(7):13384–421.
10. Elokely KM, Doerksen RJ. Docking challenge: protein sampling and molecular docking performance. Journal of Chemical Information and Modeling. 2013;53(8):1934–45.

11. Agarwal S, Mehrotra RJ. An overview of molecular docking. JSM Chemistry. 2016;4(2):1024–8.
12. Meena R, Saipraba S, Natarajan D, et al. Fioria vitifolia leaf extract-derived zinc oxide nanoparticles: green synthesis, characterization, and biological activities. Biomass Conversion and Biorefinery. 2024;14(24):31687–702.
13. Sadeghi M, Miroliaei M, Ghanadian M. Inhibitory effect of flavonoid glycosides on digestive enzymes: in silico, in vitro, and in vivo studies. International Journal of Biological Macromolecules. 2022;217:714–30.
14. Paggi JM, Pandit A, Dror RO. The art and science of molecular docking. Annual Review of Biochemistry. 2024;93(1):389–410.
15. Duraisamy R, Kalaivanan S, Paramashivam MD. Development of pharmacopoeial standards and HPTLC fingerprint analysis of roots of *Hibiscus vitifolius* Linn. Journal of Scientific and Innovative Research. 2015;4(4):182–6.
16. Peterson WL. Helicobacter pylori and peptic ulcer disease. New England Journal of Medicine. 1991;324(15):1043–8.
17. Soll AH, et al. Medical treatment of peptic ulcer disease: practice guidelines. JAMA. 1996;275(8):622–9.
18. Sverdén E, Agréus L, Dunn JM, Lagergren J. Peptic ulcer disease. BMJ. 2019;367.
19. Milosavljevic T, et al. Complications of peptic ulcer disease. Digestive Diseases. 2011;29(5):491–3.
20. Zittel TT, Jehle EC, Becker HD. Surgical management of peptic ulcer disease today. Langenbeck's Archives of Surgery. 2000;385(2):84–96.
21. Yamada T, et al. Helicobacter pylori in peptic ulcer disease. JAMA. 1994;272(1):65–9.
22. Malfertheiner P, Chan FK, McColl KE. Peptic ulcer disease. The Lancet. 2009;374(9699):1449–61.
23. Tytgat GN. Etiopathogenetic principles and peptic ulcer disease classification. Digestive Diseases. 2011;29(5):454–8.
24. Wallace JL. How do NSAIDs cause ulcer disease? Best Practice & Research Clinical Gastroenterology. 2000;14(1):147–59.
25. Najm WI. Peptic ulcer disease. Primary Care: Clinics in Office Practice. 2011;38(3):383–94.

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