Research Article

Bambusa Vulgaris Leaf Extracts and Inflammatory Targets Relevant To Cystic Fibrosis in Adolescents: GC–MS Profiling, Cell-Free Anti-Inflammatory Screening and Molecular Docking

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Abstract

Objective: To profile Bambusa vulgaris leaf extracts by gas chromatography–mass spectrometry, to screen the ethanolic extract in cell-free anti-inflammatory assays, and to explore by molecular docking how library-suggested constituents interact with inflammatory proteins relevant to cystic fibrosis, a chronic airway disease whose inflammation begins early and persists through adolescence.
Methods: Leaf powder was extracted successively with petroleum ether and ethanol. Both extracts underwent phytochemical screening and gas chromatography–mass spectrometry. The ethanolic extract was tested for inhibition of protein denaturation, nitric oxide scavenging and ferric reducing power (50–1000 µg/mL). Five library-suggested compounds were docked (AutoDock Vina) against Toll-like receptor 4, nuclear factor kappa B, cyclooxygenase-2 and tumour necrosis factor-alpha.
Results: Yields were 4.8% and 5.6%. The ethanolic extract was positive in 10 of 11 phytochemical tests. Protein-denaturation inhibition rose from 18.40% to 84.33%, and nitric oxide scavenging from 15.14% to 86.86%, across the concentration range. Best docking scores were −8.478 kcal/mol (hentriacontane-10,14,16-trione, Toll-like receptor 4), −8.397 (propanoic acid, 3,3′-thiobis-, ditetradecyl ester, nuclear factor kappa B), −7.290 (n-propyl 9-tetradecenoate, cyclooxygenase-2) and −6.448 (Glyasperin C, tumour necrosis factor-alpha). Library similarity of the docked compounds was low (35–54), and all came from the petroleum ether extract, not the extract assayed.
Conclusion: The findings are exploratory. They do not show activity against cystic fibrosis, or efficacy or safety in adolescents. Compound identification, docking validation, cell-model testing and age-appropriate safety studies are required.

Keywords: Cystic Fibrosis; Adolescent; Plant Extracts; Gas Chromatography-Mass Spectrometry; Molecular Docking Simulation; Inflammation.

Introduction

Cystic fibrosis (CF) is an autosomal recessive disorder of the cystic fibrosis transmembrane conductance regulator (CFTR). Defective anion transport dehydrates airway mucus, impairs mucociliary clearance and leads to chronic infection and inflammation, and lung disease remains the main cause of morbidity and mortality.[1,2] Inflammation begins early in life and drives disease progression.[3] Lavage studies in young children show neutrophil-dominated inflammation, with interleukin-1 associated with early structural lung disease even without infection,[4,5] and chronic Pseudomonas aeruginosa infection with a hyperinflammatory host response accelerates decline in lung function.[6] In airway epithelial cells, Toll-like receptor 4 (TLR4) engagement is linked to release of protease-bearing exosomes, elevated in sputum from people with CF colonised by P. aeruginosa,[7] and P. aeruginosa infection of CF bronchial epithelial cells activates TLR4 signalling, IκBα phosphorylation and nuclear binding of the nuclear factor kappa B (NF-κB) subunit p65.[8] NF-κB controls transcription of mediators including tumour necrosis factor-alpha (TNF-α) and cyclooxygenase-2 (COX-2).

CFTR modulators have improved outcomes, but anti-inflammatory therapy is still needed to complement them.[3] Adolescents with CF carry years of cumulative airway injury while taking on greater responsibility for a demanding daily regimen. Objectively measured adherence to CFTR modulators is suboptimal, although age under 18 years was associated with better adherence,[9] and self-efficacy, studied mainly in adolescent and adult cohorts, is associated with adherence.[10] Any adjunct anti-inflammatory approach for this age group must therefore be low-burden, tolerable and safe, and a botanical candidate would have to show this through formal pharmacological and safety evaluation, not through traditional use.

Bambusa vulgaris Schrad. ex J.C. Wendl. (Poaceae) is a widely distributed pantropical bamboo whose leaf phytochemistry and bioactivity are sparsely reported in the indexed biomedical literature. This exploratory study profiled petroleum ether and ethanolic leaf extracts by gas chromatography–mass spectrometry (GC–MS), screened the ethanolic extract in three

cell-free anti-inflammatory assays, and docked candidate constituents against four proteins spanning the inflammatory axis of the CF airway: a pattern-recognition receptor (TLR4), a transcription factor (NF-κB), a cytokine (TNF-α) and an effector enzyme (COX-2). No CF model was used and no adolescents were involved; CF in adolescents is the translational question, not the study population. The work is hypothesis-generating.

Methods

Plant material, extraction and phytochemical screening

Leaves of B. vulgaris were collected from Kapputhi R.F., Salem district, Tamil Nadu, India (11.715499° N, 78.178705° E), on 10–12 December 2025, and authenticated at the Siddha Medicinal Plants Garden, Central Council for Research in Siddha, Ministry of Ayush, Mettur Dam, Tamil Nadu (certificate No. 210126249, 21 January 2026; Dr. P. Radha, Research Officer, Botany). Leaves were washed, shade-dried (28 ± 2 °C), powdered, sieved (mesh no. 40) and stored in amber containers. The powder was extracted successively in a Soxhlet apparatus with petroleum ether (40–60 °C, 72 h; extract E1) and then ethanol (99% v/v, 72 h; extract E2). Extracts were concentrated under reduced pressure and dried in a vacuum desiccator, and yield was calculated against dry plant weight. Both extracts were screened by standard qualitative tests for resins, tannins, steroids, flavonoids, glycosides, proteins, phenols, saponins, gums and mucilage, alkaloids and carbohydrates (Supplementary Methods).

GC–MS

E1 and E2 were analysed at CIMF, Periyar University (injection volume 1.00 µL; analysis date 10 April 2026), and the NIST20M1, NIST20M2, NIST20R and WILEY8 libraries were searched. Peak areas are relative percentages, not concentrations. No authentic standards or experimental retention indices were available, so the first library suggestion and its similarity index (SI) are reported for each peak (Table 1; Supplementary Tables S1–S2).

Cell-free anti-inflammatory assays

E2 (50–1000 µg/mL) was assessed by inhibition of heat-induced bovine serum albumin denaturation (Mizushima and Kobayashi, modified; 660 nm),[11] nitric oxide scavenging by the sodium nitroprusside–Griess method (Marcocci et al., modified; 546 nm)[12] and ferric reducing power (Oyaizu; 700 nm).[13] Inhibition (%) = [(absorbance of control − absorbance of sample)/absorbance of control] × 100. Procedures are in the Supplementary Methods. The laboratory procedure states concentrations in µg/mL, whereas the laboratory tables label them µL; the procedure is followed here. Each assay report gives one summary response per concentration without replicate absorbances, so no inferential statistics are presented. Half-maximal inhibitory concentrations (IC_{50}) are laboratory-reported and unverified, and none was calculated for ferric reducing power because absorbances and the response definition are unavailable.

Molecular docking and in silico properties

Docking was run in the in-house MUMO pipeline (run 8aaeabdc-9981-469b-af6e-21181307c89b, 17 October 2026). Five first-ranked GC–MS suggestions with unambiguous structures were docked: hentriacontane-10,14,16-trione, propanoic acid, 3,3′-thiobis-, ditetradecyl ester (thiobis ester), n-propyl 9-tetradecenoate, Glyasperin C and hexestrol. Three of these were suggested by the library as trimethylsilyl derivatives and were docked as underivatised parents from their SMILES strings. Three further entries in the same run (phthalic acid, an entry named only “diethyl ester”, and fumaric acid) did not match GC–MS suggestions and were excluded.

Targets were TLR4 (UniProt O00206), the NF-κB p50 homodimer–DNA complex (PDB 1NFK; chains A and B), COX-2 (UniProt P35354) and TNF-α (UniProt P01375), using the structure recorded in the pipeline for each. Receptors were cleaned and converted with Meeko, and coordinates were checked by Ramachandran analysis,[14] ERRAT[15] and ProSA[16] (Supplementary Table S4). Ligands were protonated near pH 7.4, and the lowest-energy RDKit MMFF conformer was used. AutoDock Vina[17] was run with a rigid receptor, exhaustiveness 16, three replicas (base seed 42) and the best replica retained. Search boxes came from geometric pocket detection (TLR4, NF-κB, TNF-α) or UniProt-annotated active-site residues (COX-2) (Table S4). Scores are in kcal/mol, with more negative values more favourable; ligand efficiency (LE) is the score divided by the number of heavy atoms. Contacts were detected with ProLIF;[18] where ProLIF failed (COX-2 and TNF-α top poses), contacts were read from BIOVIA Discovery Studio 2025 diagrams made from the retained pose without redocking or minimisation. No co-crystallised ligand was redocked, so the protocol is not validated. Inducible nitric oxide synthase was part of the initial panel but could not be docked because no run records were available. Physicochemical descriptors were calculated with RDKit and ADMET (absorption, distribution, metabolism, excretion, toxicity) endpoints predicted with ADMET-AI.[19]

Results

Extraction, phytochemistry and GC–MS

Yields were 4.8% (w/w) for E1 and 5.6% for E2. E2 was positive for resins, tannins, steroids, flavonoids, glycosides, proteins, phenols, saponins, alkaloids and carbohydrates (10 of 11 tests) and negative for gums and mucilage; E1 was positive only for steroids and carbohydrates (Supplementary Table S3). These are qualitative tests.

E1 and E2 each gave 20 integrated peaks, with 17 and 15 named suggestions respectively (Supplementary Tables S1–S2). The largest E1 peak (8.83%, 16.629 min) was diethyl phthalate (SI 76). Only the E1 dodecane suggestion reached SI 80, and no named E2 suggestion did; the highest in E2 was phytol (5.62%, SI 79). The five docked compounds were all E1 suggestions with SI 35–54 (Table 1).

Table 1. Selected GC–MS library suggestions (full lists in Supplementary Tables S1–S2).

Extract	RT (min)	Area (%)	First library suggestion	SI
E1	14.205	3.90	Propanoic acid, 3,3'-thiobis-, ditetradecyl ester	54
E1	16.629	8.83	Diethyl phthalate	76
E1	35.734	3.69	n-Propyl 9-tetradecenoate	39
E1	41.535	3.63	Glyasperin C, 3TMS	35
E1	41.662	4.89	Hentriacontane-10,14,16-trione, TMS derivative	35
E1	44.205	6.12	Hexestrol, 2TMS derivative	53
E2	16.634	6.34	Diethyl phthalate	75
E2	22.069	5.62	Phytol	79

RT = retention time; SI = library similarity index, not a probability of correct identification; TMS = trimethylsilyl. Names do not confirm compound identity. E1 = petroleum ether extract; E2 = ethanolic extract.

Cell-free anti-inflammatory activity of the ethanolic extract

Protein-denaturation inhibition was 18.40%, 44.58%, 64.14%, 76.03% and 84.33%, and nitric oxide scavenging 15.14%, 38.59%, 60.88%, 76.24% and 86.86%, at 50, 250, 500, 750 and 1000 µg/mL (Figure 1). Laboratory-reported IC_{50} values were 398.51 and 435.80; the fitting method and replicate data are unavailable. Ferric reducing-power values (13.67 to 85.17) are on an undefined scale and cannot be read as inhibition (Supplementary Table S5).

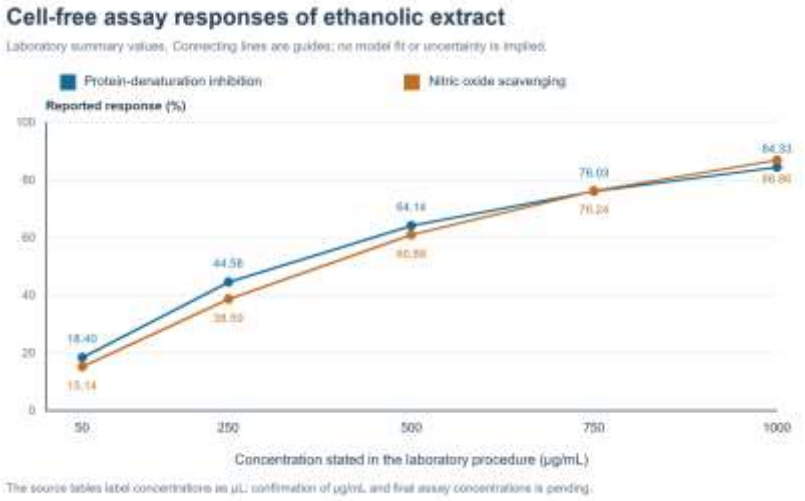


Figure 1. Laboratory-reported protein-denaturation inhibition and nitric oxide scavenging by the ethanolic extract (µg/mL). Points are single summary responses; lines connect observations and are not fitted models; no error bars or significance are implied.

Molecular docking

Receptor checks gave ERRAT quality factors of 74.658 to 96.335 and ProSA Z-scores of −4.24 to −8.87 (Supplementary Table S4); these describe the coordinates, not docking accuracy. Vina scores ranged from −4.827 to −8.478 kcal/mol, and the

leading ligand differed by target (Table 2). Hentriacontane-10,14,16-trione led on TLR4 (−8.478; LE 0.249), the thiobis ester on NF-κB (−8.397; LE 0.215), n-propyl 9-tetradecenoate on COX-2 (−7.290; LE 0.384) and Glyasperin C on TNF-α (−6.448; LE 0.248). The trione and thiobis ester were not docked against COX-2 in the recorded run. Contacts of the top poses are in Table 3 and Figure 2.

Table 2. AutoDock Vina scores (kcal/mol) of GC–MS-derived ligands.

Ligand (extract; SI)	TLR4	NF-κB	COX-2	TNF-α
Hentriacontane-10,14,16-trione (E1; 35)	**−8.478**	−7.767	ND	−5.915
Thiobis ester (E1; 54)	−7.272	**−8.397**	ND	−4.827
n-Propyl 9-tetradecenoate (E1; 39)	−6.641	−6.359	**−7.290**	−5.901
Glyasperin C (E1; 35)	−6.554	−6.183	−6.290	**−6.448**
Hexestrol (E1; 53)	−5.934	−5.423	−7.056	−5.563

Bold = most favourable per target. ND = not docked against this target in the recorded run. Best of three replicas.

Table 3. Top-ranked pose for each target.

Target	Ligand	Contacts and key residues
TLR4	Trione	ProLIF: 43 contacts (1 hydrogen bond, THR756; 42 hydrophobic, including ILE723, ILE753, PHE656, TRP757, PHE759)
NF-κB (chain B)	Thiobis ester	ProLIF: 24 contacts (2 hydrogen bonds, ARG161; 22 hydrophobic, including GLN177, GLU179, PHE217, ARG227, ARG228)
COX-2	n-Propyl ester	Discovery Studio: hydrogen bonds HIS200, THR198; alkyl/π contacts HIS193, HIS372, HIS374, TRP373, LEU376, ALA188
TNF-α	Glyasperin C	Discovery Studio: hydrogen bonds ASP219, ALA109; π ARG108; hydrophobic VAL93, ALA94, PRO96, VAL226; unfavourable contact GLY224

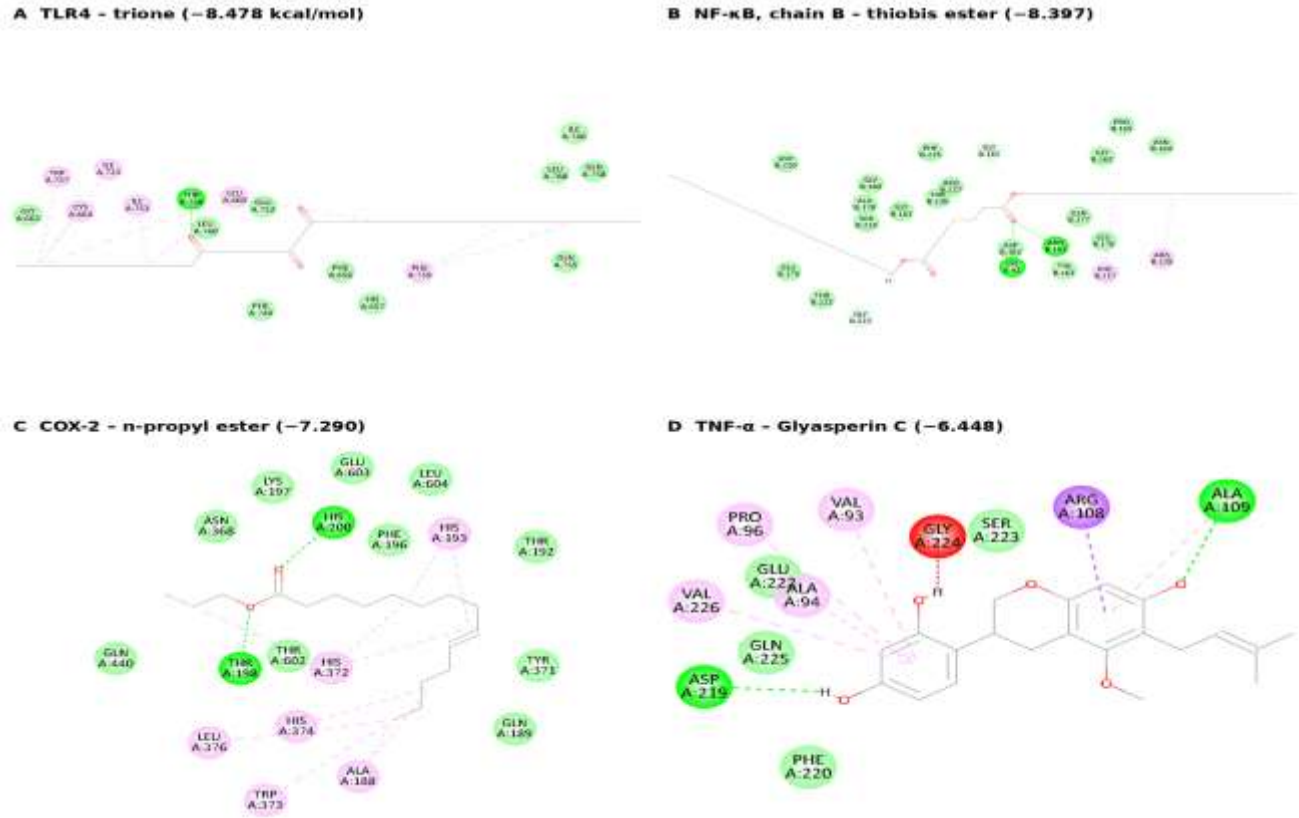


Figure 2. Two-dimensional interaction diagrams (Discovery Studio) of the top pose for each target: (A) hentriacontane-10,14,16-trione in TLR4; (B) propanoic acid, 3,3′-thiobis-, ditetradecyl ester in NF-κB (chain B); (C) n-propyl 9-tetradecenoate in COX-2; (D) Glyasperin C in TNF-α. Whole-receptor and pocket views are in Supplementary Figures S1–S4.

ADMET predictions were completed for the trione, thiobis ester and hexestrol (Supplementary Table S6). The two leading long-chain ligands were extremely lipophilic (logP 9.88 and 10.99), had quantitative estimate of drug-likeness values of 0.083 and 0.059, predicted aqueous solubility (logS) near −7, and failed the Veber filter. Hexestrol had favourable drug-likeness (0.82) but a higher predicted liver-injury probability (0.319). All values are model predictions.

Discussion

The ethanolic extract showed concentration-related responses in two cell-free assays, and the docking screen ranked five library-suggested compounds differently across four targets. These readouts describe protein-stabilising and radical-quenching behaviour and do not measure cytokine production, receptor activation or signalling. They are not irrelevant to CF, since an oxidant–antioxidant imbalance is among the mechanisms proposed for its excessive airway inflammation,[2] but no constituent was tested alone and the ferric reducing-power data cannot yet be interpreted.

The central constraint is that what was docked is not what was assayed. All five docked ligands come from the petroleum ether GC–MS report, whereas the assays used the ethanolic extract. Their similarity indices (35–54) are low, three are derivative suggestions docked as parents, and no standard or retention index supports any of them. Hexestrol is a synthetic oestrogen and is more likely a library mismatch than a plant constituent, so its score should not be used to argue for a bioactive component, least of all in a paper about adolescents. Diethyl phthalate is a ubiquitous plasticiser and needs a procedural blank. Phytol, the best E2 suggestion (SI 79), was not docked and is the obvious next candidate.

Docking results need cautious reading. The protocol was not validated by redocking, and Vina scores tend to favour large flexible lipophilic ligands, which probably explains why the two long-chain compounds lead on TLR4 and NF-κB; their LE values (0.249, 0.215) are lower than that of the smaller n-propyl ester on COX-2 (0.384). Pockets were mostly found geometrically. The TLR4 top-pose contact residues (656–759) lie in the C-terminal region that UniProt annotates as cytoplasmic, so this is not the extracellular lipopolysaccharide-recognition interface; TNF-α was docked as a monomer although the active cytokine is a trimer; and the COX-2 and TNF-α contacts come from display diagrams that, for Glyasperin C, include an unfavourable contact. A favourable score therefore does not imply interference with TLR4 recognition, NF-κB activation or TNF-α signalling. The predicted properties of the two best-scoring ligands (logP about 10–11, logS near −7, failed Veber filter) resemble aliphatic lipids, not tractable oral candidates.

For CF in adolescents, the implications are prospective only. A botanical adjunct would have to be low-burden, well tolerated and safe in a growing population, and evaluated for interactions with CFTR modulators;[9] none of this can be inferred here. The study does not support use of B. vulgaris extracts by or for adolescents with CF. Limitations also include the absence of replicate assay data, an unresolved concentration unit in the laboratory tables, no iNOS result, no CF cell, tissue or animal model, and no cytotoxicity or other safety testing. Next steps are compound identification (standards, retention indices, LC–MS, bioassay-guided fractionation), corrected docking including phytol, validated redocking and known functional sites, and testing in CF epithelial models with IL-8 and NF-κB readouts alongside cytotoxicity.

Conclusion

Ethanolic B. vulgaris leaf extract showed concentration-related responses in cell-free protein-denaturation and nitric oxide scavenging assays, and docking of five low-confidence petroleum ether-derived GC–MS suggestions gave Vina scores of −4.827 to −8.478 kcal/mol, with different leading ligands per target. The protocol was not validated, the best-scoring ligands have poor predicted drug-likeness, and no docked compound is linked to the assayed extract. These results are hypothesis-generating and provide no evidence of activity against CF, or of efficacy or safety in adolescents.

Declarations

Ethics. The study used plant material, chemical analysis, cell-free assays and computational methods only. No human participants, human samples, animals or patient data were used, and no adolescents were enrolled; Resolution 466/12 of the National Health Council is therefore not applicable.

Competing interests. The authors declare no competing interests.

Funding. This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Author contributions. S. Ruby (guide): conceptualisation, supervision, writing – review and editing. M. G. Mowriss and C. Mugilarasi: investigation and formal analysis of the in silico studies (molecular docking, interaction analysis, ADMET prediction), methodology, visualisation. N. Mohammed Kaja Muhaideen, A. Mohammed Tanish, K. R. Mohan Ram and A. Murugan: literature review, manuscript proofreading and supporting work. All authors read and approved the final manuscript.

Data availability. The original chromatographic files, assay records and prepared docking files are available from the corresponding author on reasonable request.

Use of generative AI. Generative AI tools were used to assist with revising and restructuring the text and checking numerical values against source reports (ChatGPT, OpenAI, in earlier drafts; Claude, Anthropic, in this version). The authors reviewed and edited the content and take full responsibility for all reported data and interpretations.

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