RESEARCH PAPER

TEPHROSIA PURPUREA (L.) PERS.: A CLASSIFIED REVIEW OF ITS PHYTOCHEMISTRY AND PHARMACOLOGICAL ACTIVITIES

Akansha Singh*[1], Dr. Pushpendra Kannojia[2]
1. Ph.D. Research Scholar, BIU COP Bareilly International University, Pilibhit Bypass Road, Bareilly, Uttar Pradesh (U.P.) - 243006, India.
2. Principal, Professor, BIU COP Bareilly International University, Pilibhit Bypass Road, Bareilly, Uttar Pradesh (U.P.) - 243006, India.
*Corresponding author: akanksha_era@yahoo.co.in

Abstract
Tephrosia purpurea (L.) Pers. (Fabaceae), commonly known as Sarapunkha or wild indigo, is a medicinal herb used in traditional systems for liver and spleen disorders, wounds, gastrointestinal complaints, respiratory conditions and inflammatory disorders. This narrative review classifies the pharmacological and related biological activities reported across the supplied literature, including review papers, experimental animal studies, in-vitro assays, phytochemical investigations and analytical-standardization studies. The evidence is grouped into antioxidant and anti-lipid-peroxidative, anti-inflammatory and analgesic, hepatoprotective and chemopreventive, antidiabetic, antimicrobial and antifungal, antiparasitic and antiplasmodial, antiulcer and antacid, antidiarrheal, spasmolytic and bronchodilator, nephroprotective and anti-urolithiatic, anticancer, neuropharmacological, immunomodulatory and antiallergic, wound-healing and membrane-stabilizing, and insecticidal or other ecological activities. The literature also identifies diverse flavonoids, rotenoids, isoflavones, chalcones, sterols, triterpenoids and phenolic compounds. Examples of experimentally reported findings include α-glucosidase inhibition by root extracts and isolated pyranochromenes, antioxidant activity of root and whole-plant extracts, hepatoprotection in chemically induced injury models, and in-vitro anticestodal effects of methanolic root extract against Moniezia. However, most evidence remains preclinical, methods and extract compositions vary substantially, and the available uploaded sources do not establish clinical efficacy or a consistently defined safety profile. Standardized botanical material, quantitative chemical fingerprints, dose-ranging toxicology, mechanistic validation and well-designed clinical studies are needed before therapeutic claims can be translated into practice.

Keywords: Tephrosia purpurea; Sarapunkha; pharmacological activities; antioxidant; hepatoprotective; antidiabetic; flavonoids; rotenoids.
How to cite this article: Akansha Singh*1 Dr. Pushpendra Kannojia2. TEPHROSIA PURPUREA (L.) PERS.: A CLASSIFIED REVIEW OF ITS PHYTOCHEMISTRY AND PHARMACOLOGICAL ACTIVITIES. Int J Drug Deliv Technol. 2026;16(87s):499-509. DOI: 10.25258/ijddt.16.87s.49.

1. Introduction

Tephrosia purpurea (L.) Pers. is a perennial, highly branched herb of the family Fabaceae, widely reported from India and other tropical and subtropical regions. Traditional literature describes the plant under names including Sarapunkha, Sarpunkha, Sarphonk and wild indigo. Different plant parts—roots, leaves, stems, seeds, flowers and the whole plant have been used in ethnomedicinal practice. Traditional indications summarized in the uploaded reviews include liver and spleen enlargement, jaundice, ulcers, wounds, bronchitis, asthma, diarrhea, dyspepsia, boils, skin complaints and urinary disorders [1–5]. These records are evidence of traditional use, not proof of clinical effectiveness.

The pharmacological literature spans simple in-vitro assays, isolated-tissue experiments, chemically induced rodent models, cell-line experiments, compound isolation, computational predictions and narrative reviews. The evidence therefore needs to be interpreted by activity and by experimental level. Results obtained with different plant parts, extraction solvents, doses, models and endpoints should not be treated as directly interchangeable. The present review organizes the supplied material activity-wise and highlights where evidence is experimental, indirect, computational or based primarily on traditional claims.



Fig: 1: *Tephrosia purpurea* plant

2. Review approach and scope

This is a narrative review based on the articles and documents supplied in the conversation. The source set

included reviews of ethnopharmacology and phytochemistry; experimental studies on antioxidant, inflammatory, hepatic, metabolic, gastrointestinal, renal, respiratory, infectious and cancer-related endpoints; reports of isolated natural products; and pharmacognostic or chromatographic studies relevant to identity and standardization. Studies were grouped by the principal activity investigated. Numerical results are included only where they were clearly reported in the supplied article text. No independent database search, formal risk-of-bias assessment or meta-analysis was undertaken; consequently, this manuscript should not be described as a systematic review.

The uploaded collection contains overlapping review papers and several studies of different scope. Repeated claims across reviews are not counted as independent experimental confirmation. Where an article uses animal models, cell lines, isolated tissues or in-silico methods, that level is stated to avoid implying human clinical evidence.

3. Botanical identity, traditional use and phytochemical basis

3.1. Botanical and ethnomedicinal context

The supplied reviews describe *T. purpurea* as a small, erect to spreading, herbaceous perennial with pinnate leaves, pink-to-purple or pale flowers and pods containing several seeds. It grows in wastelands and in relatively dry, sandy, gravelly or rocky soils. The plant is referred to in Ayurvedic and Unani literature and is used traditionally as a tonic, diuretic, laxative, wound remedy and ingredient of formulations used for liver-related complaints [1–5]. These ethnomedicinal records provide hypotheses for experimental study, but they do not establish a treatment recommendation.

3.2. Reported classes of constituents

Across the phytochemical and pharmacognostic papers, the reported constituents include flavonoids and prenylated flavonoids, isoflavones, flavanones, chalcones, rotenoids, glycosides, sterols, triterpenoids, tannins, saponins, phenolics and other volatile or semi-volatile compounds. Specific compounds discussed in the supplied literature include rutin, quercetin, pongamol, purpurin, purpurenone, tephrosin, deguelin, rotenone, lupeol, β-sitosterol, lanceolatin derivatives, semiglabrin, tephrosone, tephropurpurin, terpurinflavone and trans-tephrostachin [2,6–11].

Kumar et al. used GC-HRMS to report compounds including 2-methoxy-4-vinylphenol, 4-(3-hydroxyprop-1-enyl)-2-methoxyphenol, n-hexadecanoic acid, 9,12-octadecadienoic acid and oleic acid in an aqueous whole-plant extract. The same paper reported essential metal ions by ICP-OES and examined antioxidant, antidiabetic, anti-inflammatory and antimicrobial endpoints [6]. These chemical identifications and activity assays provide candidate markers; they do not alone prove that each detected constituent causes the measured effect.

Constituent group / examples	Potential relevance discussed in the supplied literature	Interpretive note
Flavonoids and phenolics: rutin, quercetin, purpurin, purpurenone	Antioxidant, antimicrobial and anti-inflammatory research; possible contribution to multiple extract activities	Activity depends on concentration, extract matrix and assay; constituent-specific mechanisms need confirmation.
Rotenoids: rotenone, deguelin, tephrosin	Reported in phytochemical studies and discussed in bioactivity literature	Rotenone is biologically potent and associated with toxicological concerns; its presence is relevant to safety and quality control.
Pyranochromenes/ chromene-related compounds	Root-derived compounds studied for α-glucosidase inhibition and antioxidant capacity	Evidence is experimental and preclinical; do not infer antidiabetic efficacy in humans.
Chalcones and prenylated flavonoids: pongamol and related compounds	Natural-product isolation, chemical profiling and	Some papers concern chemical structure rather than direct

	activity-guided investigations	therapeutic testing.
Sterols / Triterpenoids: β-sitosterol, lupeol	Quantified in seasonal HPTLC studies; may be useful analytical markers	Season and plant material influence marker content.
Volatile / Semi-volatile phenolics	GC-HRMS and computational work linked candidate compounds to antioxidant activity	In-silico predictions support hypotheses, not clinical effects.

4. Activity-wise classification of reported evidence

The table below summarizes the principal activity categories found across the uploaded sources. The detailed subsections that follow distinguish experimental findings from review-level statements.

Activity category	**Evidence in supplied literature**	**Evidence type / key limitation**
Antioxidant/Anti-lipid-peroxidative	Radical-scavenging assays, antioxidant enzyme and lipid-peroxidation endpoints in extracts and isolated compounds	In vitro and animal studies; assays and extracts differ.
Anti-inflammatory/ Analgesic	Carrageenan paw edema, cotton-pellet granuloma and thermal nociception models	Rodent preclinical evidence.
Hepatoprotective/ Antihepatotoxic	Callus extract and plant extracts studied in toxicant-induced liver injury; aflatoxin and carcinogenesis models	Animal studies; preparation and doses are not standardized.
Antidiabetic/Metabolic	Seed and whole-plant extracts, flavonoid-rich fractions, α-glucosidase inhibition and diabetes-complication models	In-vitro and rodent studies; no evidence here of established clinical efficacy.
Antimicrobial/Antifungal	Extracts, isolated flavonoids and seed saponin tested against selected organisms; endophytic fungi also studied	In-vitro results do not determine clinical dose or effectiveness.
Antiparasitic/ Antisplasmodial	Root extract against goat tapeworm in vitro; isolated stem flavones against Plasmodium falciparum	In-vitro evidence; some studies concern a subspecies.
Antiulcer / Antacid	Rat ulcer models and modified artificial-	Animal and in-vitro

	stomach model using root extracts	model evidence.
Antidiarrheal	Methanolic whole-plant extract evaluated in experimental models	Preclinical; formulation and dose translation unresolved.
Spasmolytic/ bronchodilator/ Vasorelaxant	Isolated rabbit jejunum, trachea and vascular preparations	Isolated-tissue experiments; mechanism proposed, not clinically validated.
Nephroprotective / Anti-urolithiatic	Arsenic- and gentamicin-related renal injury, and ethylene-glycol-induced calculi in rats	Animal models; further safety and mechanistic work required.
Anticancer/ Chemo-preventive	MCF-7 and HepG2 cell studies; DMBA buccal pouch and NDEA liver carcinogenesis models; quinone-reductase induction	Cell and animal studies; not proof of cancer treatment in humans.
Neuropharmacological	Status epilepticus/oxidative stress model; trans-tephrostachin and derivatives assessed computationally against acetylcholinesterase	Animal and in-silico evidence of differing strength.
Antiallergic/ Immunomodulatory/ Membrane stabilizing	Review-level reports and experimental membrane-stabilization or immune-related investigations	Evidence varies; many claims need replicated, mechanistic studies.
Wound-healing/Other ecological activity	Traditional use and review-level wound claims; insecticidal, piscicidal and iron-solubilizing reports	Traditional and ecological activity must be separated from human therapeutic efficacy.

5. Antioxidant and anti-lipid-peroxidative activity

Antioxidant activity is among the most frequently reported endpoints in the uploaded literature. Studies have measured radical-scavenging activity, total phenolic and flavonoid content, and antioxidant capacity in leaves, roots and whole-plant extracts. A study of leaf extracts reported higher total phenolic and flavonoid content in ethanolic than in aqueous extract, alongside in-vitro radical-scavenging assays. Another study assessed methanolic root extract using UV-visible spectroscopy and FTIR and reported antioxidant activity as part of its phytochemical assessment [6,12–14].

Nile and Park investigated root extracts and two isolated compounds, a pyranochromene (reported as 9,10,11-trihydroxy-1H,7aH-pyrano[2,3-c;6,5-f] dichromen-7-one) and 5-hydroxytephroapollin. The paper reported antioxidant-capacity measurements using DPPH, ABTS, ORAC and FRAP assays. Compound 2 showed higher reported values than compound 1 across these assays, while both compounds were evaluated against Trolox as a

reference [8]. The same study reported α-glucosidase inhibition, discussed separately below.

Kumar et al. reported antioxidant potential above 65% for an aqueous whole-plant extract and used density functional theory (DFT) calculations to examine antioxidant-related properties of 2-methoxy-4-vinylphenol and 4-(3-hydroxyprop-1-enyl)-2-methoxyphenol [6]. Animal studies of toxicant-induced liver or kidney injury also reported changes in lipid peroxidation, glutathione and antioxidant-enzyme measures after extract treatment [15–18]. Such findings support antioxidant potential in experimental systems; they do not establish prevention or treatment of human disease.

Interpretation is complicated by differences in plant part, solvent, harvest season, assay system and concentration. Radical-scavenging percentages cannot be directly compared unless assay conditions, controls and extract concentrations are aligned. Future studies should quantify marker compounds, use orthogonal assays, include positive controls and establish reproducibility between independently prepared batches.

6. Anti-inflammatory and analgesic activity

The anti-inflammatory literature includes rodent models of acute and subacute inflammation. Gopalakrishnan and colleagues evaluated ethanolic extracts of aerial parts and roots at 250 and 500 mg/kg in carrageenan-induced paw edema and cotton-pellet granuloma models. The extracts produced dose-related inhibition of inflammatory endpoints, and analgesic activity was also reported using a tail-immersion method [19]. Shenoy et al. independently evaluated an ethanolic extract in acute and subacute rat inflammation models [20].

Kumar et al. reported anti-inflammatory activity for an aqueous whole-plant extract and a protein-binding endpoint with a reported binding constant of $5.23 \pm 0.04 \times 10^{-5}$ μM^{-1} [6]. Review papers also list anti-inflammatory and analgesic effects among the reported activities of T. purpurea [1–5]. These findings are compatible with further investigation of inflammatory pathways, but the available studies do not establish comparative clinical effectiveness or identify a single confirmed active principle.

Recommended next steps include blinded and adequately powered studies, standardization of extract composition, measurement of relevant inflammatory mediators, inclusion of dose-response curves and direct comparison with appropriate reference drugs. Potential adverse effects should be assessed at the same exposures used to demonstrate efficacy.

7. Hepatoprotective and chemo preventive activity

Hepatic protection is a recurring theme in the traditional and experimental literature. The uploaded articles include investigations of aqueous extracts from *T. purpurea* callus culture in a hepatotoxicity model, ethanolic extract against aflatoxin B1-induced liver injury, and extract treatment in N-nitrosodiethylamine (NDEA)-induced hepatocarcinogenesis [15,16,21].

In the aflatoxin B1 study, ethanolic extract was assessed in female Wistar rats at 100, 200 and 300 mg/kg. The authors reported that treatment shifted several altered indices—including serum transaminases, triglycerides, lipid peroxidation and antioxidant status—towards control values, with the strongest recovery described at 300 mg/kg [15]. This is a specific experimental result and should not be interpreted as a clinically recommended dose.

The NDEA/CCl_4 hepatocarcinogenesis study reported that oral *T. purpurea* extract at 200 and 400 mg/kg reduced liver-injury and cancer-marker changes in Wistar rats and normalized several antioxidant-enzyme measures [21]. A separate study on ethanolic root extract in DMBA-induced hamster buccal pouch carcinoma also reported chemopreventive and antilipid-peroxidative effects [22]. These are distinct models: hepatoprotection against toxic injury and suppression of carcinogenesis should not be conflated with treatment of established human cancer.

Mechanistic hypotheses include reduction of oxidative injury and modulation of detoxification responses. An activity-guided isolation paper identified compounds that induced quinone reductase in cultured Hepa 1c1c7 cells, an assay associated with phase-II enzyme induction [23]. This provides a mechanistic lead but does not establish the in-vivo contribution of each compound. Studies should clarify exposure, pharmacokinetics, liver histopathology, interactions and toxicology before clinical development.

8. Antidiabetic and metabolic activity

The antidiabetic evidence includes traditional use, seed-extract studies, whole-plant aqueous extract assays, flavonoid-rich fractions in streptozotocin (STZ)-induced diabetes, studies of pancreatic regeneration and experiments addressing cardiovascular or cataract complications associated with diabetes [1–6,24–26].

Nile and Park examined α-glucosidase inhibitory activity of root extracts prepared with petroleum ether, ethanol, ethyl acetate, methanol and butanol, together with two isolated compounds. Their table reported in-vitro IC_{50} values of 45.1 μg/mL for petroleum ether extract, 60.5 μg/mL for butanol extract, 98.2 μg/mL for compound 1 and 120.5 μg/mL for compound 2; the acarbose comparator was reported as 1250 μg/mL in that assay. In-vivo values were also reported in the paper [8]. These results are assay-specific and should not be generalized without replication and clarification of the experimental model.

Kumar et al. reported an antidiabetic assay result of 68.45 ± 4.23% for the aqueous whole-plant extract [6]. Bhadada and Goyal evaluated a flavonoid-rich fraction at 40 mg/kg/day orally for eight weeks in STZ-induced type I diabetic rats and reported improvement in hyperglycemia, dyslipidemia and selected cardiovascular and lenticular parameters [24]. Arora et al. studied the pancreatic effects of leaf extract in STZ-induced diabetic rats [25]. Other supplied work investigated cardiovascular complications and cataract associated with STZ-induced diabetes [26].

Together, these papers suggest several research directions: digestive-enzyme inhibition, oxidative-stress modulation and effects on diabetes-associated organ complications. Nevertheless, the studies use different preparations and models, and the uploaded literature does not provide

adequate clinical evidence to recommend *T. purpurea* for diabetes. Any further work should define the extract, assess effects alongside standard antidiabetic drugs, investigate hypoglycemia risk and interactions, and conduct formal toxicology.

9. Antimicrobial and antifungal activity

Antimicrobial activity is reported for crude extracts, flavonoid-rich material and isolated constituents. The uploaded sources include ethanolic, methanolic and hydroalcoholic extracts; a report on a flavonoid isolated from *T. purpurea*; and a study of a triterpenoid saponin from seed extract [27–30]. The tested organisms and methods vary, including standard bacterial strains, clinical bacterial isolates and Candida species. Some reports also investigate endophytic fungi associated with the plant and their antifungal activity; these studies concern microorganisms associated with *T. purpurea* rather than necessarily demonstrating that the plant extract itself is antifungal [31].

Kumar et al. reported minimum inhibitory concentration (MIC) values of 23–52 mg/mL for an aqueous whole-plant extract [6]. These concentrations are high relative to many purified antimicrobial agents and should be interpreted with attention to assay conditions, extract complexity and achievable exposure. An isolated flavonoid study reported antimicrobial testing of the extracted flavonoid [27]. Because crude-extract MICs are sensitive to solvent, organism, inoculum, endpoint and standardization, cross-study ranking is not appropriate unless the same protocol is used.

Priority research needs include authenticated strains, reference antibiotics, solvent controls, standardized inocula, replicated MIC/MBC testing, biofilm endpoints where justified, cytotoxicity counterscreens and bioassay-guided fractionation. Antimicrobial assay activity should not be described as proven treatment for bacterial or fungal infections.

10. Antiparasitic and antisplasmodial activity

Two distinct antiparasitic research strands appear in the uploaded sources: anticestodal testing of root extract against goat tapeworm and antiplasmodial testing of isolated stem flavones. Ghaywat et al. tested methanolic root extract against Moniezia obtained from goat intestines. The 125 mg/mL extract concentration produced paralysis and death times of 1.29 ± 0.17 h and 2.63 ± 0.36 h, respectively; the albendazole comparator at 10 mg/mL produced corresponding times of 1.43 ± 0.14 h and 2.3 ± 0.27 h. The authors described the extract as showing in-vitro anticestodal activity comparable to albendazole at the tested conditions. Histopathology showed shrinkage of the scolex and suckers, tegumental puffing and rupture, irregular folds and crumbling of segments [7].

Natural-product studies of *T. purpurea* stem and a related subspecies have reported antiplasmodial activity for isolated flavones. Juma et al. reported terpurinflavone as an antiplasmodial flavone from the stem [32]. Another paper isolated four prenylflavone derivatives from *T. purpurea* subsp. leptostachya; (E)-5-hydroxytephrostachin was the most active reported compound against the chloroquine-sensitive D6 strain of Plasmodium falciparum, with an IC_{50} of 1.7 ± 0.1 μM and cytotoxicity IC_{50} values above 21 μM against four cell lines [33]. The subspecies qualification is important; the result should not automatically be attributed to every *T. purpurea* preparation.

These findings are promising screening results but remain in-vitro evidence. Anthelmintic studies require replicated concentration-response testing, appropriate vehicle controls, mammalian-cell safety, in-vivo efficacy and residue assessment before any veterinary use. Antiplasmodial leads require selectivity, mechanism, resistance profiling, pharmacokinetic and in-vivo validation.

11. Antiulcer and antacid activity

Deshpande, Shah and Parmar studied the aqueous root extract in several rat models of gastric and duodenal ulceration, including ethanol-, hydrochloric-acid-, indomethacin- and pyloric-ligation-associated gastric injury and cysteamine-induced duodenal ulceration. The authors reported significantly lower ulcer indices in treated animals than in vehicle controls across the models and assessed gastric acid output and pepsin activity, using omeprazole as a reference [34].

A separate in-vitro investigation used a modified artificial-stomach model to compare root extracts prepared with solvents of differing polarity. The methanolic extract showed greater neutralizing effect, duration and capacity than sodium bicarbonate in the reported model, while ethyl acetate and chloroform extracts showed intermediate responses [35]. The model is useful for preliminary screening but does not reproduce the complexity of human gastric physiology. The antiulcer animal results and artificial-stomach antacid results therefore represent related but different types of evidence.

12. Antidiarrheal activity

A methanolic extract of *T. purpurea* was investigated for antidiarrheal activity in an experimental study by Janbaz and colleagues [36]. This research relates to traditional use of the plant in diarrhea and gastrointestinal complaints. The uploaded material does not justify a clinical treatment recommendation. Further work should report extract yield and chemical fingerprint, clarify the model and mechanism, assess electrolyte and hydration outcomes, and distinguish antidiarrheal effects from possible antimicrobial or spasmolytic effects.

13. Spasmolytic, bronchodilator and vasorelaxant activity

Janbaz et al. examined methanolic whole-plant extract in isolated rabbit jejunum, tracheal and vascular preparations. The extract produced concentration-dependent relaxation of spontaneous jejunal contractions and K^+ induced contractions. In tracheal preparations, relaxation of carbachol- and high-K^+-induced contractions was described as resembling the response to verapamil; the authors proposed a calcium-channel-blocking component to the nonspecific bronchodilator response [37].

These isolated-tissue results provide a pharmacological rationale for further examination of smooth-muscle

effects. They do not establish clinical benefit in asthma, bronchitis, hypertension or other conditions. Mechanistic confirmation, selectivity studies, cardiovascular safety and in-vivo respiratory endpoints would be needed.\

14. Nephroprotective and anti-urolithiasis activity

The uploaded studies include models of arsenic-induced nephrotoxicity, gentamicin-induced nephrotoxicity and ethylene-glycol-induced renal calculi [17,18,38]. In the arsenic study, rats receiving *T. purpurea* extract alongside sodium arsenite showed lower blood urea nitrogen and plasma creatinine, higher serum albumin, improved oxidative-stress indices and less renal necrosis than the arsenic-only group. The study did not report a significant reduction in kidney arsenic accumulation, an important distinction when considering the likely protective mechanism [17].

Shukla and Mourya evaluated ethanolic and aqueous root extracts in ethylene-glycol-induced urolithiasis in rats. Extract treatment significantly reduced urinary calcium, oxalate and phosphate excretion; microscopy indicated protection against calcium oxalate crystal deposition and tubular disruption, with the authors highlighting the 300 mg/kg ethanolic extract group [38]. A separate study examined the effect of leaves on gentamicin-associated renal injury [18]. These findings are preclinical and do not establish safety or effectiveness in human kidney disease or stone prevention.

15. Anticancer, antiproliferative and chemo-preventive activity

The cancer-related literature includes cell-line studies, animal carcinogenesis models and bioassays associated with phase-II enzyme induction. One study assessed *T. purpurea* fractions in MCF-7 human breast-cancer cells and reported activity for selected fractions [39]. Padmapriya et al. examined methanolic leaf and root extracts in HepG2 human hepatocellular carcinoma cells and reported antiproliferative effects with findings consistent with mitochondrial depolarization and increased caspase-3 expression, suggesting apoptosis in that in-vitro model [40].

In vivo, ethanolic root extract was investigated in DMBA-induced hamster buccal pouch carcinoma, with reported reductions in tumor incidence, volume and burden and an antilipid-peroxidative effect [22]. *T. purpurea* extract was also studied in NDEA-induced hepatocarcinogenesis in rats, with reported improvements in liver injury and cancer markers [21]. An activity-guided study isolated eight compounds that induced quinone reductase in cultured Hepa 1c1c7 cells, including 7,4′-dihydroxy-3′,5′-dimethoxyisoflavone and (+)-tephropurpurin [23].

These findings represent early discovery and chemoprevention research, not proof that *T. purpurea* treats cancer. Cell-line effects can result from nonspecific cytotoxicity; animal chemoprevention does not establish efficacy against established tumors. Further research needs authenticated extracts, selectivity against non-malignant cells, target validation, pharmacokinetics, dose-limiting toxicity and independent replication.

16. Neuropharmacological and enzyme-target studies

An uploaded study evaluated ethanolic extract in a lithium-pilocarpine-induced status epilepticus model with oxidative-stress endpoints [41]. A separate computational study examined molecular interactions of trans-tephrostachin and derivatives with human acetylcholinesterase in the context of Alzheimer's disease research [42]. These sources are not equivalent: an animal seizure model provides in-vivo preclinical evidence, while molecular docking or related computational analyses generate hypotheses about target binding. Neither is sufficient to support a clinical claim for epilepsy or Alzheimer's disease.

A useful follow-up programme would pair computational predictions with enzyme-inhibition assays, selectivity testing, cellular models, exposure measurements and appropriately designed in-vivo studies. For seizure-related work, behavioral, neurological and safety endpoints should be evaluated alongside oxidative-stress measures.

17. Antiallergic, immunomodulatory, membrane-stabilizing and wound-healing activity

Several uploaded reviews list antiallergic and immunomodulatory properties, and some describe mast-cell stabilization or erythrocyte-membrane integrity effects in experimental models [1–5]. A separate report investigates membrane-stabilizing potency of two Tephrosia species [43]. Because the uploaded source set contains a mixture of reviews and experimental reports, these claims should be treated as areas for further investigation rather than a single established pharmacological profile.

Wound-healing is prominent in traditional descriptions of Sarapunkha. Review articles summarize wound-related use, and the 2019 volatile-compound paper refers to earlier wound-healing research [1–6]. However, the supplied material does not provide a sufficiently consistent set of comparable wound-model data to quantify the magnitude of effect across extracts. Future studies should use standardized wound models, vehicle and positive controls, blinded planimetry, histology, microbial-load measurement where appropriate, and toxicity testing.

18. Insecticidal, piscicides and other non-clinical biological activities

Traditional and phytochemical reviews report insecticidal, piscicidal, vermifuge and insect-repellent uses for some parts or preparations of *T. purpurea* [1–5]. These uses are biologically distinct from therapeutic activities in humans. Rotenone and related rotenoids are relevant to this literature and also raise safety concerns because potent biological activity may be accompanied by toxicity. The uploaded article on a rock-iron-solubilizing compound from root exudates describes a phenolic/proteinaceous fraction that solubilized $Fe(OH)_3$ under acidic conditions and discusses a possible environmental or phytosiderophore role [44]. This is an ecological/biochemical finding, not evidence of a medicinal indication.

Similarly, the studies on endophytic fungi, plant chemical profiling for cosmetic ingredients and seasonal quantification of lupeol, β-sitosterol and rotenone

contribute to biological understanding and quality control, but should not be presented as direct evidence of therapeutic efficacy [9,31,45].

19. Pharmacognostical quality, analytical standardization and reproducibility

The uploaded collection contains pharmacognostic studies of roots, leaves, aerial parts and bark; preliminary phytochemical screening; TLC/HPTLC profiling; UV-visible and FTIR characterization; and a validated seasonal HPTLC method for lupeol, β-sitosterol and rotenone [9–14,45–48]. These works are important because efficacy findings are difficult to compare when plant identity, plant part, season, extraction procedure and chemical composition are not standardized.

The seasonal HPTLC study reported that marker concentrations varied with season and extract, and cautioned that winter-collected material may have a higher rotenone content. This finding makes toxicologically informed collection and batch release testing important [9]. Quality-control plans should include botanical authentication and voucher specimens, macroscopic and microscopic identification, physicochemical parameters, chromatographic fingerprints, quantitative marker ranges, contaminant testing and limits for constituents of toxicological concern.

The studies on phytochemical profiling also show why a single marker may not fully represent a multi-constituent extract. A validated fingerprint combined with quantitative markers and a bioassay linked to the intended activity would provide stronger batch comparability than qualitative screening alone.

20. Critical appraisal and research gaps

- Evidence level: Most activity claims derive from in-vitro assays, isolated tissues, cell lines, computational predictions or animal models. The uploaded sources do not establish clinical efficacy in humans.
- Heterogeneity: Plant parts, solvents, extract yields, doses, assay protocols, reference controls and outcome measures vary, preventing reliable quantitative pooling.
- Chemical attribution: Many papers test crude extracts. Activity-guided isolation and reconstitution studies are needed to identify active constituents and determine whether combinations are additive, synergistic or antagonistic.
- Safety: Rotenone and other potent constituents require attention to toxicity, therapeutic index, interactions and possible residues. Traditional use cannot substitute for formal toxicology.
- Reproducibility: Botanical authentication, voucher specimens, harvest season, storage, extraction details and chemical fingerprints should be reported consistently.
- Mechanistic validation: Antioxidant, enzyme-inhibition and docking results should be confirmed with orthogonal assays, target engagement and pharmacokinetic data.
- Translation: Standardized formulation development, stability studies, dose-ranging toxicology and carefully justified clinical research are needed before therapeutic claims can be made.
- Reporting quality: Studies should predefine outcomes, justify sample sizes, use appropriate randomization and blinding, report all groups and adverse events, and make raw or sufficiently detailed data available.

21. Proposed activity-based research roadmap

Priority	Suggested work	Rationale / deliverable
1. Botanical and chemical standardization	Authenticate root, leaf and aerial parts; prepare voucher specimens; validate HPTLC/HPLC/LC-MS fingerprints; quantify selected flavonoids, sterols and rotenoids.	Comparable, reproducible extracts and toxicologically informed marker specifications.
2. Replication of strongest screening signals	Repeat antioxidant, α-glucosidase, antimicrobial and anti-inflammatory assays using common protocols and positive controls.	Reliable concentration-response curves and inter-batch comparison.
3. Bioassay-guided fractionation	Fractionate the most reproducibly active extract; isolate compounds; test purified compounds and reconstituted mixtures.	Link chemistry to biological activity and reduce unsupported attribution.
4. Safety and selectivity	Conduct cytotoxicity counterscreens, acute/subchronic toxicity, interaction	Define margins between activity and toxicity.

	screening and exposure assessment; prioritize rotenone-related safety.	
5. Mechanistic and in-vivo validation	Validate targets and pathways, measure exposure, and use appropriately powered animal models only when justified by in-vitro evidence.	Distinguish genuine pharmacology from assay artifacts.
6. Translation	Develop standardized formulations and stability methods; proceed to clinical studies only after adequate nonclinical evidence and regulatory review.	Evidence-based development rather than premature therapeutic claims.

22. Conclusion

The uploaded literature portrays *T. purpurea* as a chemically diverse medicinal plant with a broad range of reported biological activities. The most developed experimental strands include antioxidant activity, anti-inflammatory and analgesic effects, hepatoprotection, antidiabetic and α-glucosidase-inhibitory activity, antimicrobial screening, gastrointestinal effects, renal protection, cancer-related assays and antiparasitic investigations. Natural-product studies have isolated structurally diverse flavonoids, pyranochromenes and other metabolites that may serve as leads for further research.

The breadth of reported activity should not be mistaken for a breadth of proven clinical benefit. Results are largely preclinical and are affected by substantial variation in extract preparation and analytical characterization. The most defensible path forward is activity-specific standardization, independent replication, safety assessment and mechanism-led development. Clinical use should not be inferred from the experimental findings summarized here.

Abbreviations

Abbreviation	Meaning
ABTS	2,2′-Azino-bis(3-ethylbenzothiazoline-6-sulfonic acid) assay
DFT	Density functional theory
DPPH	2,2-Diphenyl-1-picrylhydrazyl assay
FRAP	Ferric reducing antioxidant power
GC-HRMS	Gas chromatography–high-resolution mass spectrometry
HPTLC	High-performance thin-layer chromatography
IC_{50}	Concentration producing 50% inhibition
MIC	Minimum inhibitory concentration
NDEA	N-nitrosodiethylamine
ORAC	Oxygen radical absorbance capacity
STZ	Streptozotocin
T. purpurea	*Tephrosia purpurea* (L.) Pers.

References

[1] Palbag S, Dey BK, Singh NK. Ethnopharmacology, phytochemistry and pharmacology of Tephrosia purpurea. Chinese Journal of Natural Medicines. 2014;12(1):1–7.

[2] Dalwadi PP, Patel JL, Patani PV. *Tephrosia purpurea* Linn (Sharpunkha, Wild Indigo): A review on phytochemistry and pharmacological studies. Indian Journal of Pharmaceutical and Biological Research. 2014;2(1):108–121.

[3] Negi N, Kalia AN, Brar S, Gauttam VK. Therapeutic potential of Tephrosia purpurea. Journal of Chemical and Pharmaceutical Research. 2015;7(2):574–580.

[4] Baranwal A, Mazumder A, Chakraborthy GS, Gupta S. Phytopharmacological uses of Tephrosia purpurea—A review. Pharmacophore. 2014;5(4):658–665.

[5] Rajakkani T, Vijayabharathi R, Radha R. Review on Tephrosia purpurea. World Journal of Pharmacy and Pharmaceutical Sciences. 2019;8(4):466–477. doi:10.20959/wjpps20194-13435.

[6] Kumar V, Singh S, Srivastava B, Patial PK, Kondalkar SA, Bharthi V. Volatile and semi-volatile compounds of *Tephrosia purpurea* and its medicinal activities: Experimental and computational studies. Biocatalysis and Agricultural Biotechnology. 2019; 20:101222. doi: 10.1016/j.bcab.2019.101222.

[7] Ghaywat LD, Bhavare VV, Borgave SS, Bhagde RV, Mahale LM. Therapeutic study of *Tephrosia purpurea* (Linn.) root extract on goat tapeworm with special reference to histopathological parameters. Journal of

Veterinary and Animal Sciences. 2021;52(3):286–291. doi:10.51966/jvas.2021.52.3.286-291.

[8] Nile SH, Park SW. α-Glucosidase inhibitory activity of pyranochromenes and root extracts of Tephrosia purpurea. Chemistry of Natural Compounds. 2014;50(6):1113–1115. doi:10.1007/s10600-014-1175-6.

[9] Khatoon S, Irshad S, Pandey MM, Rastogi S, Rawat AKS. A validated HPTLC densitometric method for determination of lupeol, β-sitosterol and rotenone in Tephrosia purpurea: A seasonal study. Journal of Chromatographic Science. 2019;57(8):688–696. doi:10.1093/chromsci/bmz041.

[10] Peng Y, Gao C, Yan T, et al. Two new secondary metabolites from Tephrosia purpurea. Natural Product Communications. 2015. doi:10.1177/1934578X1501000631.

[11] Juma WP, Akala HM, Eyase FL, et al. Terpurinflavone: An antiplasmodial flavone from the stem of Tephrosia purpurea. Phytochemistry Letters. 2011; 4:176–178.

[12] Jirvankar PS, Khobragade D, Chandewar S, Pimpale A, Gawali R, Lokade A, Maske A, Agrawal R. Phytochemical, UV-visible and FTIR assessment along with in vitro antioxidant activity of methanolic extract of *Tephrosia purpurea* Linn root. Journal of Pharmaceutical Research International. 2022;34(3B):26–33. doi:10.9734/JPRI/2022/v34i3B35389.

[13] Nigam S, Leo P. Phytochemical screening and TLC profiling of *Tephrosia purpurea* leaves and stem. European Journal of Biomedical and Pharmaceutical Sciences. 2015;2(7):162–165.

[14] Estimation of flavonoid, polyphenolic content and in-vitro antioxidant capacity of leaves of *Tephrosia purpurea* Linn. (Leguminosae). (Full author/journal metadata should be checked against the source PDF before submission.)

[15] Gupte SS, Rathour A, Gupta D, et al. *Tephrosia purpurea* ameliorates oxidative and histological alterations induced by aflatoxin B1 in rats. International Journal of Pharmaceutical Investigation. 2022;12(3):340–345. doi:10.5530/ijpi.2022.3.

[16] Mujeeb M, Zafar R, Husain A, Ahmad A. Antihepatotoxic activity of aqueous extracts of callus culture of *Tephrosia purpurea* (L.) Pers. Acta Poloniae Pharmaceutica—Drug Research. 2012;69(3):545–549.

[17] Gora RH, Kerketta P, Baxla SL, Toppo R, Prasad R, Patra PH, Roy BK. Ameliorative effect of *Tephrosia purpurea* in arsenic-induced nephrotoxicity in rats. (Original article; journal and year should be confirmed from the supplied PDF before submission.)

[18] Effect of *Tephrosia purpurea* (L.) Pers. leaves on gentamicin-induced nephrotoxicity in rats. (Bibliographic metadata should be checked against the supplied PDF before submission.)

[19] Gopalakrishnan S, Vadivel E, Dhanalakshmi K. Anti-inflammatory and analgesic activities of *Tephrosia purpurea* Linn. aerial and root extracts. Journal of Pharmacy Research. 2010;3(5):1103–1106.

[20] Shenoy S, Shwetha K, Prabhu K, Maradi R, Bairy KL, Shanbhag T. Evaluation of antiinflammatory activity of *Tephrosia purpurea* in rats. Asian Pacific Journal of Tropical Medicine. 2010:193–195.

[21] Chemopreventive evaluation of *Tephrosia purpurea* against N-nitrosodiethylamine-induced hepatocarcinogenesis in Wistar rats. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[22] Anticarcinogenic and antilipidperoxidative effects of Tephrosia purpurea. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[23] Activity-guided isolation of constituents of *Tephrosia purpurea* with the potential to induce the Phase II enzyme, quinone reductase. (Full bibliographic metadata should be checked against the supplied DOCX before submission.)

[24] Bhadada SV, Goyal RK. Effect of flavonoid-rich fraction of *Tephrosia purpurea* (Linn.) Pers. on complications associated with streptozotocin-induced type I diabetes mellitus. Indian Journal of Experimental Biology. 2016; 54:457–466.

[25] Arora SK, Verma PR, Itankar PR, et al. Evaluation of pancreatic regeneration activity of *Tephrosia purpurea* leaves in rats with streptozotocin-induced diabetes. Journal of Traditional and Complementary Medicine. 2021; 11:435–445.

[26] Bhadada S, Goyal RK. Effect of aqueous extract of *Tephrosia purpurea* on cardiovascular complications and cataract associated with streptozotocin-induced diabetes in rats. Indian Journal of Pharmaceutical Sciences. 2015. doi:10.4103/0250-474X.169037.

[27] Singh V, Saxena RC, Singh AK. A flavonoid out of *Tephrosia purpurea* extract and its antimicrobial effect. Biomedical & Pharmacology Journal. 2008;1(2):465–468.

[28] Antimicrobial activity of *Tephrosia purpurea* (Linn.) Pers. and Mimusops elengi (Linn.) against some clinical bacterial isolates. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[29] Extraction and antimicrobial activity of hydroalcoholic extract of Tephrosia purpurea. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[30] In vitro antimicrobial activity of triterpenoid saponin from *Tephrosia purpurea* seeds extract. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[31] Phylogenetic diversity and antifungal activity of endophytic fungi associated with Tephrosia purpurea. (Full bibliographic metadata should be checked against the supplied PDF before submission.)

[32] Juma WP, Akala HM, Eyase FL, et al. Terpurinflavone: An antiplasmodial flavone from the

stem of Tephrosia purpurea. Phytochemistry Letters. 2011; 4:176–178.

[33] Four prenylflavone derivatives with antiplasmodial activities from the stem of *Tephrosia purpurea* subsp. leptostachya. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[34] Deshpande SS, Shah GB, Parmar NS. Antiulcer activity of *Tephrosia purpurea* in rats. Indian Journal of Pharmacology. 2003; 35:168–172.

[35] Sandhya S, Venkata Ramana K, Vinod KR, Chaitanya RSN. Assessment of in vitro antacid activity of different root extracts of *Tephrosia purpurea* (L.) Pers. by modified artificial stomach model. Asian Pacific Journal of Tropical Biomedicine. 2012: S1487–S1492.

[36] Janbaz KH, Jan A, Qadir MI, Gilani AH. Anti-diarrheal activity of methanolic extract of Tephrosia purpurea. Acta Poloniae Pharmaceutica—Drug Research. 2013;70(2):261–269.

[37] Janbaz KH, Jan A, Qadir MI, Gilani AH. Spasmolytic, bronchodilator and vasorelaxant activity of methanolic extract of Tephrosia purpurea. Acta Poloniae Pharmaceutica—Drug Research. 2013;70(2):261–269.

[38] Shukla A, Mourya P. Investigations for anti-urolithiatic activity of *Tephrosia purpurea* roots against ethylene glycol-induced renal calculi in rats. Asian Journal of Pharmacy and Pharmacology. 2016;2(2):40–43.

[39] Anticancer activity of *Tephrosia purpurea* and Ficus religiosa using MCF-7 cell lines. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[40] Padmapriya R, Gayathri L, Ronsard L, Akbarsha MA, Raveendran R. In vitro anti-proliferative effect of *Tephrosia purpurea* on human hepatocellular carcinoma cells. Pharmacognosy Magazine. 2017.

[41] Assessment of effect of ethanolic extract of *Tephrosia purpurea* (L.) Pers. on lithium-pilocarpine-induced status epilepticus and oxidative stress in Wistar rats. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[42] Molecular interaction of human acetylcholinesterase with trans-tephrostachin and derivatives for Alzheimer's disease. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[43] Membrane stabilizing potency of two Tephrosia species. (Full author/journal metadata should be checked against the supplied PDF before submission.)

[44] Aggarwal KK, Rajiv J, Babu CR. A rock-iron-solubilizing compound from root exudates of Tephrosia purpurea. Journal of Chemical Ecology. 1999;25(10).

[45] Exploiting the complementarity between dereplication and computer-assisted structure elucidation for the chemical profiling of natural cosmetic ingredients: *Tephrosia purpurea* as a case study. (Full bibliographic metadata should be checked against the supplied PDF before submission.)

[46] Pharmacognostical and phytochemical evaluation of *Tephrosia purpurea* (Linn.) Pers. (Supplied document; full bibliographic metadata should be checked before submission.)

[47] Pharmacognostical standardization of *Tephrosia purpurea* Pers root. (Supplied document; full bibliographic metadata should be checked before submission.)

[48] Phytochemical investigation and quantitative estimation of flavonoid and phenolic contents of the root, stem and leaves of *Tephrosia purpurea* Linn. (Full bibliographic metadata should be checked against the supplied PDF before submission.