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Cuscuta reflexa Roxb. (Tukhme Kasoos): From Unani Therapeutics to Emerging Anticancer and Hepatoprotective Potential-Phytochemical Basis, Molecular Mechanisms and Translational Perspectives

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Abstract | **Original Research Article**

Background*:** *Cuscuta reflexa* Roxb. (Tukhme Kasoos/Aftimoon) is a holoparasitic medicinal plant traditionally used in the Unani system for several disorders, including hepatobiliary conditions. Contemporary investigations have increasingly explored its phytochemistry, anticancer activity, hepatoprotective effects, and associated molecular mechanisms. ***Objective*:** This review aimed to critically evaluate the phytochemical basis, anticancer and hepatoprotective potential, molecular mechanisms, safety considerations, and translational relevance of *C. reflexa*. ***Methods: Evidence was synthesized from studies retrieved through major biomedical and multidisciplinary databases, including PubMed/MEDLINE, Scopus, Web of Science, Embase, ScienceDirect, and Google Scholar. Experimental evidence was categorized according to phytochemical, in-vitro, in-vivo, in-silico, and traditional evidence levels. Particular attention was given to plant material, extraction method, experimental model, molecular endpoints, and translational limitations. ***Results*:** *C. reflexa* contains diverse phytoconstituents, including phenolic compounds, flavonoids, coumarin-related constituents, sterols, glycosides, and phenylpropanoid derivatives. Preclinical studies demonstrated concentration-dependent antiproliferative activity and tumor-growth inhibition in experimental cancer models. ***Conclusion:*** *C. reflexa* is a promising preclinical natural-product candidate with notable anticancer and hepatoprotective potential.

Keywords: *Cuscuta Reflexa*, Tukhme Kasoos, Aftimoon, Anticancer, Hepatoprotective, Phytochemistry, Apoptosis, Oxidative Stress, NF-Kb, Translational Pharmacology.

INTRODUCTION

Natural products have historically represented an important source of therapeutic agents and continue to contribute substantially to contemporary drug discovery, particularly in areas such as cancer, inflammatory disorders, metabolic diseases, and hepatoprotection. Medicinal plants are chemically complex biological systems containing structurally diverse secondary metabolites capable of interacting with multiple molecular targets. This multi-target pharmacological potential has renewed interest in traditional medicinal plants as sources of bioactive compounds and therapeutic leads. However, the transition from traditional use to evidence-based therapeutic application requires rigorous botanical authentication, phytochemical characterization, experimental validation, elucidation of molecular mechanisms, pharmacokinetic assessment, toxicological evaluation, and ultimately well-designed clinical investigation.

Cuscuta reflexa Roxb., commonly known as giant dodder and traditionally recognized as Aftimoon or Tukhme Kasoos in the Unani system of medicine, is a holoparasitic medicinal plant belonging to the family Convolvulaceae. The plant is widely distributed across South and Southeast Asia and is characterized by slender, twining, largely leafless stems that obtain nutrients from host plants through specialized haustorial connections. Its parasitic biology is particularly relevant from a pharmacognostic perspective because differences in host species, geographical origin, environmental conditions, plant part, and harvesting stage may influence its secondary-metabolite composition and, consequently, its pharmacological activity.

Phytochemical studies have demonstrated that *C. reflexa* contains several classes of biologically active constituents, including flavonoids, phenolic compounds, coumarins, phenylpropanoid derivatives, sterols, glycosides, and related secondary metabolites. Detailed chemical investigation has additionally resulted in the identification of novel 2H-pyran-2-one glucosides, cuscutarosides A and B, and a steroidal glucoside, demonstrating that the phytochemical diversity of this species extends beyond its traditionally reported constituents [1].

Among the pharmacological properties attributed to *C. reflexa*, its potential anticancer activity has attracted increasing scientific interest. Early experimental investigations demonstrated that alcoholic, hydroalcoholic, and aqueous extracts of the whole plant exerted concentration-dependent antiproliferative effects against several human cancer cell lines. Alcoholic extract and its chloroform fraction showed particularly notable cytotoxicity and also inhibited tumor growth in experimental Ehrlich and Sarcoma-180 solid-tumor models, providing both in-vitro and in-vivo evidence of antitumor potential [2]. Subsequent mechanistic studies expanded these observations by demonstrating that aqueous *C. reflexa* extract could influence inflammatory and apoptotic pathways. In Hep3B hepatocellular carcinoma cells, treatment was associated with increased expression of the pro-apoptotic proteins BAX and p53 and reduced expression of the anti-apoptotic proteins Bcl-2 and survivin. The same investigation demonstrated suppression of TNF-α and COX-2 expression and inhibition of NF-κB DNA-binding activity, suggesting an important mechanistic relationship between modulation of inflammatory signalling and induction of cancer-cell apoptosis [3].

Compound-level investigations have further strengthened the phytochemical basis of the anticancer activity. Riaz et al. isolated scoparone, p-coumaric acid, stigmasta-3,5-diene, and 1-O-p-hydroxycinnamoylglucose from *C. reflexa*. Among these compounds, 1-O-p-hydroxycinnamoylglucose demonstrated promising antiproliferative activity against HCT116 colorectal cancer cells, while the remaining compounds exhibited moderate effects [4]. Such findings are important because they begin to establish a direct relationship between chemically characterized constituents and observed pharmacological effects rather than attributing biological activity solely to crude extracts. More recent investigations have employed integrated experimental and computational strategies. Studies involving A549 human lung adenocarcinoma cells have indicated that *C. reflexa* stem extract may induce reactive oxygen species (ROS)-associated apoptosis, supported by cytotoxicity, apoptosis assays, phytochemical profiling, and network-pharmacology analyses [5]. These findings suggest that oxidative-stress regulation, mitochondrial dysfunction, apoptotic signalling, and inflammation-associated pathways may collectively contribute to the anticancer effects of the plant.

Hepatoprotection represents another particularly relevant pharmacological domain for *C. reflexa*, especially because hepatic and hepatobiliary disorders are among its historically documented applications in traditional medicine. Experimental evidence has shown improvement in hepatic biochemical and histopathological parameters following *C. reflexa* administration in a murine model of chemically induced colon cancer, together with evidence suggesting reduction in tumor progression and limited hepatic toxicity under the experimental conditions employed [6]. More recently, a phytochemical-rich seed extract was evaluated against chlorpyrifos-induced hepatotoxicity in mice. Treatment with the extract attenuated oxidative stress and liver injury, supporting the hypothesis that antioxidant and cellular-protective mechanisms contribute to its hepatoprotective potential [7]. These findings provide an important experimental basis for exploring the relationship between phytochemical composition, oxidative-stress modulation, inflammatory regulation, preservation of endogenous antioxidant defence, and maintenance of hepatocellular integrity.

Despite these encouraging observations, the translational significance of *C. reflexa* remains limited by substantial methodological and evidentiary gaps. Variation in host plant, botanical material, extraction solvent, dosage, chemical composition, and experimental model makes comparison between studies difficult. Furthermore, many reported biological activities have not yet been linked to quantitatively standardized marker compounds, and information concerning absorption, distribution, metabolism, elimination, therapeutic exposure, herb–drug interactions, chronic toxicity, and clinical efficacy remains inadequate. Although in-silico and network-pharmacology approaches can help identify potential molecular targets, computational predictions require independent experimental validation before therapeutic conclusions can be drawn.

Therefore, *C. reflexa* should currently be considered a promising preclinical natural-product candidate rather than an established anticancer or hepatoprotective therapy. A rational translational pathway should integrate authenticated botanical material, host-plant documentation, chromatographic standardization, bioactivity-guided isolation, compound-specific mechanistic studies, dose-response assessment, pharmacokinetic characterization, comprehensive toxicological investigation, and subsequent clinical evaluation. The present review therefore aims to critically integrate the phytochemical basis of *C. reflexa* with available anticancer and hepatoprotective evidence, examine the molecular pathways underlying these effects, and identify the major scientific challenges and

research priorities required for its progression from traditional medicinal use and experimental pharmacology toward evidence-based therapeutic development.

MATERIALS AND METHODS

This review was designed as a structured, mechanism-oriented evidence synthesis examining the traditional use, pharmacognosy, phytochemical composition, anticancer activity, hepatoprotective potential, molecular mechanisms, safety profile, and translational relevance of *Cuscuta reflexa* Roxb. (Tukhme Kasoos/Aftimoon). Particular emphasis was placed on critically distinguishing historical therapeutic claims from experimentally demonstrated pharmacological effects and on evaluating the strength of evidence supporting the anticancer and hepatoprotective potential of the plant.

The review was structured to integrate evidence from classical Unani medicine with contemporary phytochemical, in-vitro, in-vivo, computational, toxicological, and, where available, human studies. Because *C. reflexa* is a parasitic medicinal plant whose chemical profile may potentially vary according to host species, geographical origin, plant part, and extraction procedure, these variables were specifically considered during evidence interpretation.

Information Sources

A comprehensive literature search was planned across major biomedical and multidisciplinary electronic databases, including:

- PubMed/MEDLINE;
- Scopus;
- Web of Science Core Collection;
- Embase;
- ScienceDirect; and
- Google Scholar.

The search period extended from database inception to 31 August 2026. Reference lists of relevant primary studies and review articles were additionally examined to identify potentially eligible publications not retrieved through the electronic database search.

Classical Unani literature was assessed separately from contemporary biomedical literature. Authoritative materia medica, pharmacopoeial sources, classical therapeutic texts, and relevant contemporary publications dealing specifically with Tukhme Kasoos/Aftimoon were considered for documentation of traditional identity, temperament, pharmacological actions, therapeutic uses, adverse effects, correctives, substitutes, doses, and compound formulations.

Search Strategy

The electronic search strategy combined the accepted botanical name, traditional nomenclature, pharmacological terms, and disease- or mechanism-related keywords. The principal search expression was structured around the following terms:

("Cuscuta reflexa" OR "Cuscuta reflexa Roxb." OR "Aftimoon" OR "Tukhme Kasoos" OR "Kasoos") AND ("phytochemistry" OR "phytoconstituent" OR "pharmacolog" OR "anticancer" OR "antitumor" OR "cytotoxic*" OR "antiproliferative" OR "apoptosis" OR "hepatoprotect*" OR "hepatotoxicity" OR "antioxidant" OR "oxidative stress" OR "anti-inflammatory" OR "inflammation" OR "toxicity" OR "safety").**

Additional targeted searches were performed or planned for individual pharmacological domains using combinations of terms relating to cancer, liver injury, reactive oxygen species, mitochondrial dysfunction, NF-κB signalling, TNF-α, COX-2, BAX, Bcl-2, p53, survivin, phytochemical isolation, chromatographic profiling, pharmacokinetics, toxicology, and natural-product drug development.

Search syntax was adapted according to the indexing requirements of individual databases. Searches were not restricted to a particular geographical region because the objective was to capture all available evidence specifically relating to *C. reflexa*.

Eligibility Criteria

Studies were considered eligible when they fulfilled the following criteria:

1. The investigated botanical material was clearly identified as *Cuscuta reflexa* Roxb.;
2. sufficient information regarding the plant material, plant part, extract, fraction, or isolated compound was provided;
3. the study evaluated phytochemical, pharmacognostic, pharmacological, anticancer, hepatoprotective, antioxidant, anti-inflammatory, toxicological, or mechanistic outcomes;
4. experimental outcomes were adequately described;
5. the publication represented an original research article, relevant review, pharmacognostic study, toxicological investigation, or authoritative traditional-medicine source;
6. The study contributed directly to understanding the phytochemical basis, biological activity, molecular mechanisms, safety, or translational potential of *C. reflexa*.

Studies involving other species of *Cuscuta* were not regarded as direct evidence for *C. reflexa*. Such studies were considered only when they provided clearly

identified comparative or contextual information and were not used to substantiate species-specific therapeutic conclusions.

Studies were excluded when botanical identity was uncertain, the investigated species could not be differentiated from another *Cuscuta* species, experimental methods were insufficiently described, outcome data were unavailable, or the publication provided only unsupported therapeutic claims without identifiable pharmacological or traditional-source evidence.

Study Selection

Records identified through database searching were subjected to sequential screening. Titles and abstracts were initially examined for relevance to *C. reflexa*, its traditional use, phytochemistry, pharmacology, anticancer activity, hepatoprotection, safety, or associated molecular mechanisms. Potentially relevant publications were subsequently evaluated in full text against the predefined eligibility criteria.

Duplicate publications describing the same experimental dataset were to be identified by comparison of authorship, study model, plant preparation, experimental design, and reported outcomes. Where multiple publications originated from the same dataset, the most complete report was prioritized while supplementary publications were used only when they contained additional relevant information.

The final manuscript should report the number of records identified, duplicates removed, records screened, full-text reports assessed, exclusions with reasons, and studies included after completion of the final August 2026 search.

Data Extraction

Information from eligible studies was extracted into structured evidence categories. For each experimental study, the following variables were considered where available:

- author and year of publication;
- geographical origin of the plant material;
- host plant, when reported;
- plant part used;
- botanical authentication and voucher specimen;
- extraction solvent and extraction procedure;
- extract or fraction yield;
- isolated or quantified phytoconstituents;
- experimental model;
- cell line, animal species, or biological system;
- dose or concentration;
- duration of exposure or treatment;
- comparator or control group;
- biochemical, molecular, histopathological, or pharmacological endpoints;
- principal findings;
- proposed mechanism of action;
- toxicity or safety observations; and
- Principal methodological limitations.

For anticancer studies, particular attention was given to cancer type, cell line, cytotoxic or antiproliferative endpoints, apoptosis, cell-survival pathways, oxidative-stress responses, inflammatory signalling, tumor-growth inhibition, and normal-cell controls where available.

For hepatoprotective studies, extracted outcomes included serum hepatic biochemical markers, oxidative-stress indices, antioxidant defence parameters, inflammatory markers, liver histopathology, toxicant exposure, and evidence of hepatocellular injury or recovery.

Phytochemical Evidence Assessment

Phytochemical evidence was evaluated according to the degree of chemical characterization. Studies reporting only preliminary phytochemical screening were distinguished from studies employing chromatographic or spectroscopic characterization and from investigations involving isolation and structural identification of individual compounds.

Where available, evidence obtained using HPLC, HPTLC, GC-MS, LC-MS, NMR, or related analytical methods was prioritized for defining the chemical composition of the investigated material. Particular attention was given to chemically characterized constituents potentially associated with anticancer or hepatoprotective activity, including phenolic compounds, flavonoids, coumarin-related compounds, phenylpropanoid derivatives, sterols, glycosides, and newly identified secondary metabolites.

Compound-level evidence was considered stronger than nonspecific extract-level phytochemical claims when the biological activity of an isolated or quantitatively characterized constituent had been experimentally evaluated.

Evidence Hierarchy

To minimize overinterpretation and to clearly distinguish different levels of pharmacological evidence, included information was classified according to the following hierarchy:

Level I – Human Clinical Evidence: evidence obtained from clinical investigations in humans.

Level II – In-vivo evidence: evidence from animal models evaluating pharmacological efficacy, toxicity, tumor inhibition, hepatoprotection, or other biological outcomes.

Level III – In-Vitro Evidence: evidence derived from cellular, biochemical, molecular, or isolated-tissue experiments.

Level IV – In-Silico Evidence: computational evidence, including molecular docking, network pharmacology, target prediction, pathway enrichment, and related approaches.
Level V – Traditional/Classical Evidence: documentation of historical therapeutic use in Unani or other traditional medical literature.

Traditional-use evidence was not considered equivalent to contemporary proof of pharmacological or clinical efficacy. Similarly, computational predictions were regarded as hypothesis-generating unless supported by appropriate experimental validation.

Assessment of Molecular Mechanisms

Mechanistic evidence was synthesized according to experimentally investigated biological pathways rather than solely according to reported pharmacological activity. For anticancer effects, emphasis was placed on mechanisms involving apoptosis, oxidative stress, mitochondrial signalling, inflammatory pathways, NF-κB-associated signalling, regulation of BAX/Bcl-2 balance, p53, survivin, TNF-α, COX-2, and related pathways where directly evaluated.

For hepatoprotection, evidence was assessed in relation to modulation of oxidative stress, lipid peroxidation, endogenous antioxidant defence, inflammatory responses, hepatocellular integrity, biochemical liver-function parameters, and histopathological preservation.

Proposed mechanisms lacking direct experimental confirmation were clearly differentiated from experimentally demonstrated molecular effects.

Evaluation of Traditional Unani Evidence

Traditional Unani evidence was analysed within its original conceptual framework. Information was extracted regarding Mizaj (temperament), Af'al (pharmacological actions), therapeutic indications, Muzirrat (adverse effects), Musleh (correctives), Badal (substitutes), classical dose, and compound formulations.

Classical descriptions were not automatically translated into modern pharmacological terminology. Instead, areas of apparent convergence between traditional indications and contemporary experimental findings were treated as hypothesis-generating relationships. For example, classical use in hepatobiliary disorders was compared with modern hepatoprotective evidence, while traditional references to Sartaan were considered in relation to preclinical anticancer findings without assuming historical proof of modern anticancer efficacy.

Assessment of Translational Relevance

The translational potential of the available evidence was evaluated according to several predefined domains: botanical authentication, host-plant documentation, chemical standardization, reproducibility of extraction, identification of active constituents, mechanistic validation, dose-response relationships, in-vivo efficacy, pharmacokinetics, toxicity, formulation development, and availability of human clinical evidence.

Particular attention was paid to whether the preparation evaluated experimentally was sufficiently characterized to permit reproducibility. Evidence from crude or poorly standardized extracts was interpreted cautiously, especially when plant origin, host species, extraction yield, or marker-compound content was not adequately reported.

The translational pathway was conceptually evaluated as: (u may give it proper flow chart or diagram)
Traditional evidence → botanical authentication → phytochemical characterization → bioactivity-guided fractionation → mechanistic validation → in-vivo efficacy → pharmacokinetics and toxicology → standardized preparation → human clinical investigation.

Data Synthesis

Because considerable heterogeneity was anticipated in plant parts, host species, extraction procedures, concentrations, doses, experimental models, comparators, and outcome measures, the evidence was synthesized qualitatively rather than pooled statistically. Findings were organized into thematic domains covering traditional use, pharmacognosy, phytochemistry, anticancer effects, hepatoprotective effects, antioxidant and anti-inflammatory pharmacology, safety, standardization, and translational potential.

Where possible, emphasis was placed on agreement between phytochemical characterization, biological activity, and mechanism-specific evidence. Conflicting or inconsistent findings were interpreted in relation to differences in botanical material, host plant, extraction method, dose, experimental model, and level of chemical standardization.

RESULTS

The available evidence demonstrated a broad but predominantly preclinical pharmacological profile for *Cuscuta reflexa* Roxb. Studies included in the present evidence synthesis investigated whole-plant extracts, stem extracts, seed extracts, fractions, and isolated phytoconstituents using phytochemical, in-vitro, in-vivo, and computational approaches. The principal areas of contemporary investigation were anticancer activity, hepatoprotection, antioxidant activity, anti-inflammatory

effects, and molecular mechanisms related to apoptosis and oxidative-stress regulation.

The evidence was heterogeneous with respect to botanical material, plant part, extraction solvent, experimental concentration or dose, biological model, and outcome measures. Consequently, quantitative pooling was not considered appropriate, and the findings were synthesized according to pharmacological domain and level of evidence.

Among the available contemporary studies, anticancer activity was supported by both in-vitro cellular studies and in-vivo murine tumor models, whereas hepatoprotective activity was supported primarily by animal studies. Molecular investigations provided evidence implicating NF-κB-associated inflammatory signalling, regulation of apoptosis-related proteins, and reactive oxygen species (ROS)-associated pathways. However, evidence of established therapeutic efficacy in humans was not identified within the evidence compiled in the present manuscript.

Table 1: Overall Evidence Profile of *Cuscuta reflexa* Roxb

Evidence domain	Type of evidence	Major observations	Overall interpretation
Phytochemistry	Isolation, chromatographic and structural characterization	Flavonoids, phenolics, coumarin-related compounds, sterols, glycosides, phenylpropanoid derivatives, cuscutarosides A and B and steroidal glucoside identified	Chemically diverse natural-product source
Anticancer activity	In-vitro and in-vivo	Concentration-dependent growth inhibition of several cancer cell lines and inhibition of experimental tumor growth	Moderate preclinical evidence
Apoptosis	In-vitro mechanistic studies	Increased BAX and p53; reduced Bcl-2 and survivin; ROS-associated apoptosis reported	Mechanistic evidence supportive
Anti-inflammatory activity	Cellular studies	Reduced TNF-α and COX-2 expression and inhibition of NF-κB DNA-binding activity	Mechanistically relevant to anticancer activity
Hepatoprotection	In-vivo studies	Improvement in hepatic biochemical, oxidative-stress and histopathological parameters	Moderate preclinical evidence
Antioxidant activity	Chemical and cellular assays	Radical-scavenging activity and oxidative-stress modulation reported	Supportive but requires in-vivo confirmation
Safety	Limited experimental evidence	No comprehensive preparation-specific toxicological profile available	Evidence insufficient for clinical translation
Human efficacy	Human clinical evidence	No established therapeutic evidence identified in the compiled evidence	Clinical investigation required

Phytochemical investigations confirmed that *C. reflexa* contains multiple structurally distinct secondary-metabolite classes rather than a single universally recognized active molecule. Reported constituents included flavonoids, phenolic compounds, coumarin derivatives, phenylpropanoid-related molecules, sterols, and glycosides.

chemically investigated the whole plant and isolated two previously undescribed 2H-pyran-2-one glucosides, designated cuscutarosides A and B, together with 7β-methoxy-β-sitosterol 3-O-β-glucopyranoside and several previously known compounds. These findings demonstrated substantial chemical diversity and provided candidate constituents for further biological evaluation.

Additional natural-product investigations identified scoparone, p-coumaric acid, stigmasta-3,5-diene, and 1-O-p-hydroxycinnamoylglucose. Of these, 1-O-p-hydroxycinnamoylglucose demonstrated particularly promising antiproliferative activity against HCT116 colorectal cancer cells. Collectively, these results provided a preliminary compound-level basis for some of the pharmacological effects observed with crude or fractionated *C. reflexa* preparations.

Table 2: Major Reported Phytochemical Constituents of *Cuscuta reflexa*

Constituent/compound class	Chemical category	Material reported	Reported biological relevance	Evidence status
Cuscutaroside A	2H-pyran-2-one glucoside	Whole plant	Newly characterized secondary metabolite	Chemically established
Cuscutaroside B	2H-pyran-2-one glucoside	Whole plant	Newly characterized secondary metabolite	Chemically established
7β-Methoxy-β-sitosterol 3-O-β-glucopyranoside	Steroidal glucoside	Whole plant	Preliminary biological activity	Chemically established

Scoparone	Coumarin	Whole plant	Moderate antiproliferative relevance	Preclinical
p-Coumaric acid	Phenolic acid	Whole plant	Potential antioxidant/antiproliferative relevance	Preclinical
Stigmasta-3,5-diene	Steroidal compound	Whole plant	Moderate antiproliferative activity reported	Preclinical
1-O-p-Hydroxycinnamoylglucose	Phenylpropanoid glycoside	Whole plant	Promising activity against HCT116 cells	Preclinical
Flavonoids	Polyphenolic compounds	Different preparations	Antioxidant and biological activity potentially relevant	Class-level evidence
Phenolic compounds	Polyphenols	Different preparations	Antioxidant/ROS-modulating potential	Class-level evidence

The anticancer potential of *C. reflexa* was supported by both cellular and animal studies. Alcoholic, hydroalcoholic, and aqueous whole-plant extracts demonstrated concentration-dependent inhibition of cancer-cell proliferation across models representing breast, colon, lung, and liver malignancies.

The alcoholic extract showed particularly notable activity against MCF-7 breast cancer cells, while the chloroform fraction displayed substantial activity across multiple tested cell lines. In animal experiments, the alcoholic extract administered at 40 mg/kg inhibited tumor growth by approximately 42.6% in the Ehrlich solid-tumor model and 26.0% in the Sarcoma-180 model. The chloroform fraction at 10 mg/kg produced approximately 49.0% and 44.1% tumor-growth inhibition, respectively.

These results indicate that fractionation may concentrate constituents with antitumor activity. Nevertheless, differences between doses and preparations prevent direct conclusions regarding relative potency without standardized chemical characterization.

Table 3: Experimental Evidence for the Anticancer Activity of *Cuscuta reflexa*

Preparation	Experimental model	Major outcome	Principal finding	Evidence level
Alcoholic whole-plant extract	Human cancer cell lines including MCF-7	Cellular growth inhibition	Concentration-dependent cytotoxic/antiproliferative activity	III
Hydroalcoholic extract	Multiple human cancer cell lines	Cellular growth inhibition	Antiproliferative activity reported	III
Aqueous extract	Multiple cancer cell lines	Cellular growth inhibition	Concentration-dependent activity	III
Alcoholic extract, 40 mg/kg	Ehrlich solid tumor	Tumor-growth inhibition	Approximately 42.6% inhibition	II
Alcoholic extract, 40 mg/kg	Sarcoma-180 tumor	Tumor-growth inhibition	Approximately 26.0% inhibition	II
Chloroform fraction, 10 mg/kg	Ehrlich solid tumor	Tumor-growth inhibition	Approximately 49.0% inhibition	II
Chloroform fraction, 10 mg/kg	Sarcoma-180 tumor	Tumor-growth inhibition	Approximately 44.1% inhibition	II
Isolated phytoconstituents	HCT116 colorectal cancer cells	Antiproliferative activity	1-O-p-hydroxycinnamoylglucose showed promising activity	III
Stem extract	A549 lung adenocarcinoma cells	Cytotoxicity/apoptosis	ROS-associated apoptotic activity	III
Network pharmacology analysis	Predicted anticancer pathways	Target/pathway prediction	Proposed multiple molecular targets	IV

Mechanistic investigations demonstrated that the anticancer activity of *C. reflexa* was associated with regulation of both apoptotic and inflammatory pathways.

In Hep3B hepatocellular carcinoma cells, treatment with aqueous *C. reflexa* extract resulted in increased expression of BAX and p53 and decreased expression of Bcl-2 and survivin. These changes represent a shift in cellular signalling toward a pro-apoptotic phenotype.

The same investigation demonstrated inhibition of NF-κB DNA-binding activity together with suppression of TNF-α and COX-2 expression. Because persistent activation of NF-κB contributes to inflammation, cancer-cell survival, and resistance to apoptosis, inhibition of this pathway may represent an

important mechanistic link between the anti-inflammatory and anticancer actions of *C. reflexa*.

More recent experiments using A549 human lung adenocarcinoma cells suggested involvement of ROS-dependent apoptotic mechanisms. The integration of antioxidant assays, cytotoxicity testing, Annexin V/PI-based assessment, chemical profiling, and network pharmacology indicated that modulation of intracellular redox status may contribute to cancer-cell death.

Table 4: Molecular Mechanisms Associated with the Anticancer Activity of *Cuscuta reflexa*

Molecular target/pathway	Direction of effect	Biological significance	Experimental context
BAX	Increased	Promotes mitochondrial apoptosis	Hep3B cells
p53	Increased	Promotes cell-cycle control and apoptosis	Hep3B cells
Bcl-2	Decreased	Reduces anti-apoptotic signalling	Hep3B cells
Survivin	Decreased	Reduces inhibition of apoptosis	Hep3B cells
NF-κB DNA-binding activity	Inhibited	Suppresses survival/inflammatory signalling	Cellular model
TNF-α	Reduced	Reduced pro-inflammatory signalling	RAW264.7 cells
COX-2	Reduced	Attenuation of inflammatory signalling	RAW264.7 cells
Reactive oxygen species-associated signalling	Modulated/increased in cancer-cell apoptotic context	Promotes oxidative stress-mediated cancer-cell apoptosis	A549 cells
Annexin V/PI-defined apoptosis	Increased	Indicates programmed cancer-cell death	A549 cells
Network-predicted molecular targets	Multiple pathways	Hypothesis-generating evidence	In-silico/network pharmacology

The collective mechanistic evidence supports a proposed pathway in which phytochemical constituents of *C. reflexa* modulate oxidative stress and inflammatory signalling while altering the balance between pro- and anti-apoptotic proteins, ultimately promoting cancer-cell death.

Hepatoprotective activity represented the second major therapeutic domain supported by contemporary experimental evidence. In a murine model of chemically induced colon cancer, administration of *C. reflexa* was associated with improvement in hepatic biochemical and histopathological parameters. The investigators additionally reported probable inhibition of tumor progression with limited evidence of hepatic toxicity under the experimental conditions.

A more specific model of chemically induced liver injury evaluated a phytochemical-rich *C. reflexa* seed extract against chlorpyrifos-associated hepatotoxicity. Administration of the extract was associated with improvement in parameters reflecting hepatic injury and oxidative stress.

The available findings support a potential hepatoprotective mechanism involving attenuation of oxidative injury, preservation of endogenous antioxidant capacity, reduction of lipid peroxidation, and maintenance of hepatocellular integrity. However, the contribution of individual phytochemicals remains insufficiently characterized.

Table 5: Experimental Evidence for Hepatoprotective Effects of *Cuscuta reflexa*

Study/model	Preparation	Major outcomes evaluated	Main finding	Interpretation
Chemically induced murine colon-cancer model	*C. reflexa* extract	Hepatic biochemical indices and histopathology	Improvement in hepatic parameters	Supportive hepatoprotective evidence
Murine colon-cancer model	*C. reflexa* extract	Tumor progression and hepatic safety	Probable reduction in tumor progression with limited hepatic toxicity	Combined antitumor/hepatic evidence
Chlorpyrifos-induced hepatotoxicity model	Phytochemical-rich seed extract	Hepatic injury and oxidative-stress parameters	Attenuation of toxicant-associated hepatic injury	Direct preclinical hepatoprotection
Oxidative-stress endpoints	Seed extract	Oxidative stress/antioxidant defence	Protective modulation reported	Plausible mechanistic contribution

Histopathological assessment	Experimental liver injury	Hepatocellular structural alterations	Improvement in hepatic histology reported	Tissue-level support
Future translational requirement	Standardized extract/isolated compounds	Biochemical, molecular, histological and pharmacokinetic endpoints	Not yet established	Requires further validation

Antioxidant and anti-inflammatory actions appeared to represent mechanistic links between the anticancer and hepatoprotective properties of *C. reflexa*. Experimental investigations reported activity in DPPH, ABTS, FRAP, hydroxyl-radical, superoxide, and reactive-nitrogen-species assays. Such chemical antioxidant assays demonstrated radical-scavenging capacity but should not independently be interpreted as evidence of therapeutic antioxidant efficacy.

More biologically relevant observations included suppression of TNF-α and COX-2 expression and inhibition of NF-κB DNA-binding activity. The convergence of oxidative-stress modulation and inflammatory-pathway inhibition provides a biologically plausible framework through which *C. reflexa* phytochemicals may influence both cancer progression and hepatocellular injury.

Despite promising findings, the overall evidence remained predominantly preclinical. Anticancer activity was supported by cellular and animal studies and therefore represented moderate preclinical evidence. Hepatoprotective activity was similarly supported principally by animal models. Anti-inflammatory effects had mechanistic cellular support, whereas antioxidant evidence included both chemical and cellular approaches.

The principal barriers to translation were inadequate standardization of botanical material, inconsistent documentation of host species, differences in extraction procedures, insufficient quantitative phytochemical characterization, limited pharmacokinetic information, incomplete toxicological evaluation, and the absence of established human clinical efficacy.

Table 6: Strength of Evidence and Translational Status of the Major Pharmacological Activities of *Cuscuta reflexa*

Therapeutic domain	Current evidence	Evidence strength	Major limitation	Translational status
Anticancer	Cellular and animal studies	Moderate preclinical	Limited standardized preparations and PK data	Further mechanistic/in-vivo validation required
Hepatoprotection	Animal studies	Moderate preclinical	Limited models and compound-specific evidence	Standardized studies required
Apoptosis	Cellular mechanistic evidence	Moderate	Limited confirmation across cancer types	Mechanistic validation required
Anti-inflammatory	Cellular mechanistic evidence	Moderate	Limited in-vivo pathway validation	Preclinical
Antioxidant	Chemical and cellular evidence	Moderate	Chemical assays may not predict in-vivo efficacy	Further biological validation required
Phytochemical basis	Multiple characterized compounds	Moderate	Active therapeutic markers incompletely defined	Bioactivity-guided isolation required
Toxicology/safety	Limited preparation-specific data	Insufficient	Chronic, reproductive and genotoxicity data lacking	Comprehensive safety studies required
Pharmacokinetics	Very limited	Insufficient	ADME and systemic exposure poorly characterized	Major translational gap
Human clinical efficacy	No established evidence in current dataset	Very low/insufficient	Clinical trials unavailable	Not established

The integrated evidence indicates that *C. reflexa* possesses a chemically diverse phytochemical profile associated with multiple preclinical pharmacological activities. The strongest evidence concerned anticancer, antioxidant, anti-inflammatory, and hepatoprotective effects. Anticancer activity was

supported by concentration-dependent inhibition of cancer-cell growth, experimental tumor-growth suppression, modulation of apoptosis-related proteins, inhibition of NF-κB-associated inflammatory signalling, and ROS-dependent apoptosis.

Hepatoprotective findings demonstrated improvement in biochemical and histopathological manifestations of experimental liver injury, particularly in toxicant-associated hepatic damage. These observations suggest a mechanistic convergence involving phytochemical-mediated regulation of oxidative stress, inflammation, apoptosis, and tissue integrity.

Nevertheless, heterogeneity of plant material, host species, extraction methods, doses, and experimental models substantially limits direct comparison between studies. The available findings therefore support *C. reflexa* as a promising preclinical natural-product candidate but do not establish it as a clinically validated anticancer or hepatoprotective treatment.

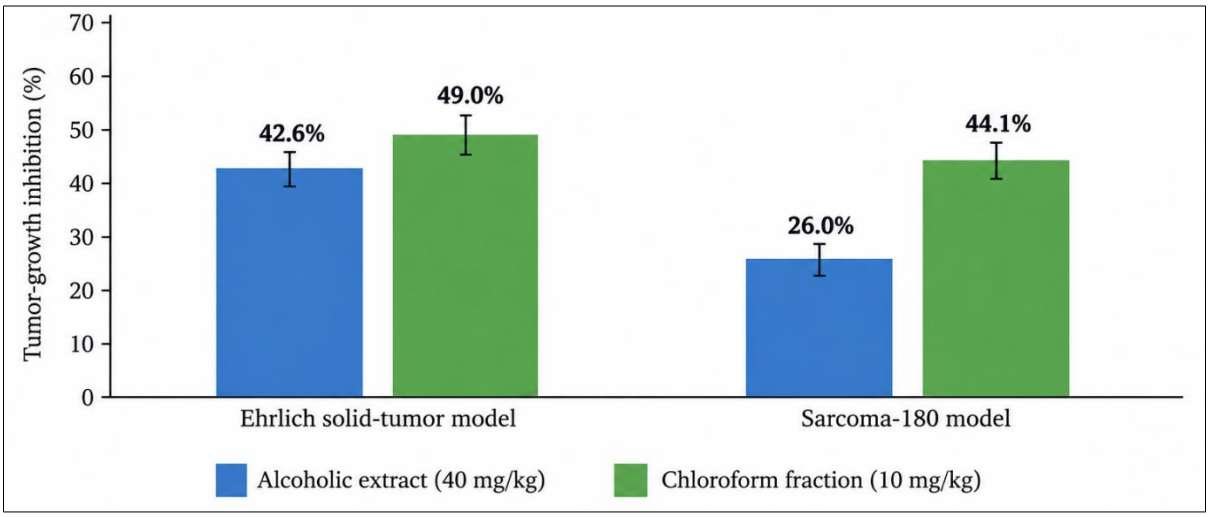

Figure 1: Tumor-growth inhibition produced by alcoholic extract and chloroform fraction of *Cuscuta reflexa* in Ehrlich solid-tumor and Sarcoma-180 experimental models

Figure 1 the alcoholic extract at 40 mg/kg produced approximately 42.6% and 26.0% tumor-growth inhibition in the Ehrlich and Sarcoma-180 models, respectively, whereas the chloroform fraction at 10 mg/kg produced approximately 49.0% and 44.1% inhibition.

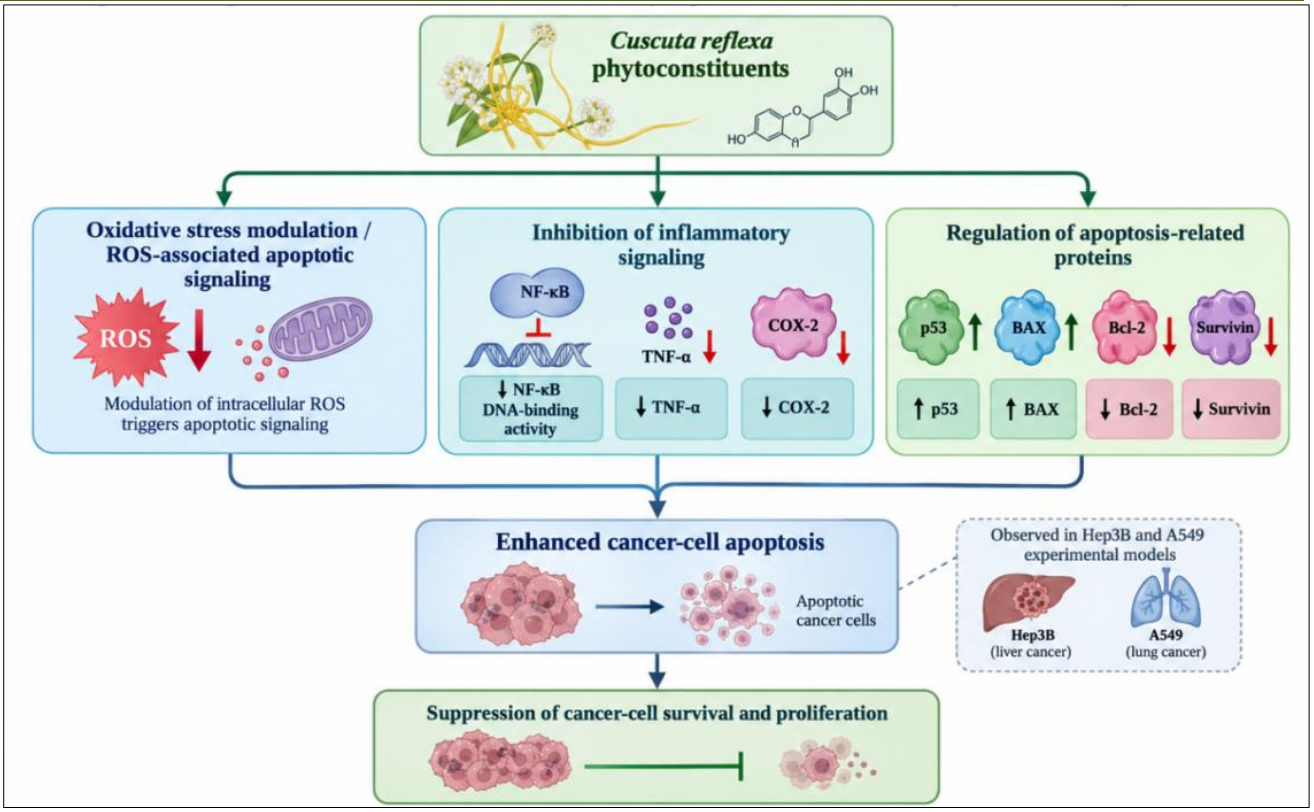

Figure 2: Proposed molecular mechanisms underlying the anticancer activity of Cuscuta reflexa

Figure 2 the proposed pathway illustrates modulation of oxidative stress, inhibition of NF-κB-associated inflammatory signalling, suppression of TNF-α and COX-2, increased BAX and p53, decreased Bcl-2 and survivin, and consequent enhancement of cancer-cell apoptosis.

DISCUSSION

The present review demonstrates that *Cuscuta reflexa* Roxb. (Tukhme Kasoos/Aftimoon) possesses a broad and chemically heterogeneous pharmacological profile, with the most compelling contemporary evidence relating to anticancer, antioxidant, anti-inflammatory, and hepatoprotective activities. The findings are particularly noteworthy because experimental observations extend beyond nonspecific phytochemical screening and increasingly provide mechanistic evidence involving apoptosis, oxidative-stress regulation, inflammatory signalling, and preservation of cellular integrity. Nevertheless, the available evidence remains predominantly preclinical, and considerable heterogeneity in plant material, extraction procedures, chemical standardization, experimental models, and pharmacological endpoints continues to limit direct translation into clinical practice.

One of the principal findings of the present synthesis was the considerable phytochemical diversity of *C. reflexa*. Constituents identified from different preparations include phenolic compounds, flavonoids, coumarin-related compounds, sterols, glycosides, and phenylpropanoid derivatives. This chemical diversity is consistent with the broader phytochemical profile reported for the genus *Cuscuta*, in which flavonoids and glycosidic constituents represent prominent classes associated with antioxidant, antiproliferative, anti-inflammatory, and hepatoprotective activities [10]. A more recent comprehensive review of *C. reflexa* similarly documented numerous isolated bioactive compounds and emphasized the pharmacological breadth of the species while highlighting the need for stronger experimental and toxicological validation [11]. These observations support the interpretation that the pharmacological activity of *C. reflexa* is unlikely to be attributable to a single molecule and may instead arise from multiple constituents acting through complementary pathways.

The importance of chemical standardization is reinforced by recent pharmacognostic work. Khan *et al*., characterized *C. reflexa* using physicochemical assessment, preliminary phytochemical screening, and HPLC profiling and demonstrated the presence of carbohydrates, phenolic compounds, alkaloids, glycosides, flavonoids, steroids, tannins, and saponins [12]. Such studies are valuable because they provide analytical parameters that may ultimately contribute to crude-drug authentication and batch-level quality control. However, a general HPLC fingerprint alone does not establish which constituents are responsible for anticancer or hepatoprotective activity. Future studies

therefore need to progress toward quantitative marker analysis, LC-MS-based metabolomic profiling, and bioactivity-guided fractionation linking individual chemical features with reproducible pharmacological effects.

The anticancer evidence synthesized in the present review is particularly encouraging. The experimental results demonstrated concentration-dependent growth inhibition in several cancer-cell models and measurable tumor-growth suppression in murine systems. The observation that the chloroform fraction produced approximately 49.0% inhibition in the Ehrlich solid-tumor model and 44.1% inhibition in the Sarcoma-180 model, compared with approximately 42.6% and 26.0%, respectively, with the alcoholic extract, suggests that fractionation may enrich constituents possessing stronger antitumor activity. However, this apparent difference must be interpreted cautiously because the preparations were administered at different doses and were not standardized according to an identical concentration of chemically defined active compounds. Consequently, the results should not be interpreted as definitive evidence that the chloroform fraction is intrinsically more potent.

The current findings also indicate that the anticancer effects of *C. reflexa* cannot be explained solely by nonspecific cytotoxicity. Mechanistic evidence indicates modulation of several proteins involved in the balance between cancer-cell survival and programmed cell death. Increased p53 and BAX expression together with reduced Bcl-2 and survivin expression suggests activation of a pro-apoptotic cellular environment. This is biologically relevant because the BAX/Bcl-2 balance is closely associated with mitochondrial apoptotic regulation, whereas survivin contributes to inhibition of apoptosis and maintenance of malignant-cell survival. The observed changes therefore provide a plausible molecular explanation for the growth-inhibitory effects identified in cellular and animal models.

More recent experimental evidence strengthens this interpretation. Elasbali *et al.,* examined *C. reflexa* stem extract in A549 human lung adenocarcinoma cells using antioxidant assays, cytotoxicity testing, Annexin V/PI evaluation, mitochondrial membrane-potential analysis, ROS assessment, and network pharmacology [8]. Their findings supported ROS-dependent apoptosis and indicated interactions between phytochemical constituents and multiple proteins associated with lung-cancer progression. This is consistent with the mechanistic model proposed in the present review, in which modulation of intracellular redox status contributes to mitochondrial dysfunction and apoptotic cancer-cell death. Importantly, ROS biology in cancer is context-dependent: moderate ROS concentrations may support malignant signalling, whereas excessive intracellular ROS can exceed cellular antioxidant capacity and trigger apoptosis. Therefore, the apparent coexistence of antioxidant capacity in cell-free assays and pro-oxidant apoptotic activity in malignant cells is not necessarily contradictory.

Network pharmacology and computational analyses provide useful additional information but require careful interpretation. Adnan *et al*., demonstrated that previously identified *C. reflexa* constituents showed predicted interactions with several biological targets and also evaluated ADME/T characteristics using in-silico approaches [9]. Such approaches are valuable for identifying candidate compounds and pathways, particularly in chemically complex natural products. However, docking affinity or network connectivity alone cannot establish pharmacodynamic activity. Experimental confirmation using target-specific inhibition, gene silencing, protein-expression studies, or pathway-rescue experiments is required before predicted interactions can be considered validated molecular mechanisms.

Another important finding of the present synthesis was the interaction between anticancer and anti-inflammatory mechanisms. Suppression of NF-κB DNA-binding activity, TNF-α, and COX-2 suggests that *C. reflexa* may interfere with signalling pathways that connect chronic inflammation to malignant-cell survival and proliferation. NF-κB activation is particularly relevant because it can promote transcription of genes involved in inflammation, cell survival, proliferation, invasion, and resistance to apoptosis. Therefore, simultaneous inhibition of inflammatory signalling and enhancement of apoptosis represents a pharmacologically plausible dual mechanism.

This interpretation is supported by newer evidence. Ahmed *et al*., investigated the anti-inflammatory potential of *C. reflexa* using chromatographic, in-silico, and in-vitro approaches and identified multiple candidate phytochemicals in an ethyl acetate extract [13]. Their study demonstrated anti-inflammatory activity in protein-denaturation and human red-blood-cell membrane-stabilization assays and implicated COX-2- and interleukin-associated targets through computational analyses. Although these findings do not directly establish anticancer efficacy, they strengthen the evidence that anti-inflammatory pathways form an important component of the overall pharmacological profile of *C. reflexa*.

The hepatoprotective findings are similarly significant. Experimental studies summarized in the present review demonstrated improvement in hepatic biochemical and histopathological abnormalities in animal models, including chemically induced hepatic injury. These effects appear to involve attenuation of oxidative stress, maintenance of endogenous antioxidant defences, reduction of lipid peroxidation, and preservation of hepatocellular structure. Such

mechanisms are biologically plausible because oxidative stress is a major contributor to hepatocyte membrane damage, mitochondrial dysfunction, inflammatory signalling, and progression of toxicant-induced liver injury.

The growing hepatoprotective literature has recently been critically reviewed by Gull *et al.,* who concluded that *C. reflexa* preparations can ameliorate experimentally induced liver damage through modulation of hepatic enzymes, oxidative stress, and liver histoarchitecture [14]. Their review also emphasized that, despite promising animal and in-vitro data, clinical trials and more rigorous mechanistic investigations remain necessary. This conclusion closely agrees with the present findings. The currently available evidence justifies describing *C. reflexa* as a promising hepatoprotective candidate but does not support describing it as an established treatment for human hepatic disease.

The relationship between antioxidant activity and hepatoprotection warrants particular attention. Several *C. reflexa* preparations have demonstrated antioxidant capacity in chemical assays, while experimental liver studies suggest improvement in oxidative-stress-related endpoints. However, antioxidant activity measured using DPPH, ABTS, FRAP, hydroxyl-radical, or related chemical assays should not be directly equated with therapeutic antioxidant activity in vivo. These assays primarily measure chemical reducing or radical-scavenging capacity under controlled experimental conditions. Demonstration of biological antioxidant action requires assessment of intracellular ROS, glutathione status, superoxide dismutase, catalase, glutathione peroxidase, lipid-peroxidation products, redox-sensitive signalling pathways, and corresponding histopathological outcomes.

An additional aspect that distinguishes *C. reflexa* from many conventional medicinal plants is its parasitic biology. As a holoparasitic species, its nutritional and metabolic interaction with the host plant raises the possibility that host identity influences secondary-metabolite composition and therefore pharmacological activity. Recent comprehensive evaluations of *C. reflexa* have highlighted the need for improved botanical and phytochemical characterization before its biological activities can be interpreted reliably [11]. Future publications should therefore report host species, collection site, collection season, plant part, voucher specimen, developmental stage, extraction yield, and chemical fingerprint. Failure to document these parameters may partly explain discrepancies between pharmacological studies.

The traditional Unani profile of Tukhme Kasoos/Aftimoon provides an additional ethnopharmacological dimension. Contemporary reviews of Aftimoon document its longstanding use in Unani practice and describe numerous traditional therapeutic applications and compound formulations [15]. The historical association of the drug with hepatobiliary conditions is especially interesting in light of modern hepatoprotective findings. Similarly, historical references to Sartan provide a basis for hypothesis generation concerning anticancer activity. Nevertheless, traditional indications should not be retrospectively interpreted as equivalent to experimentally proven modern disease entities. Traditional evidence is most appropriately used to prioritize pharmacological hypotheses rather than to demonstrate efficacy.

Several limitations of the current evidence base require emphasis. First, the majority of anticancer evidence derives from cell culture or experimental tumor models, and no established human anticancer efficacy was identified. Second, extract concentrations and animal doses cannot currently be translated into rational clinical doses because pharmacokinetic data are inadequate. Third, crude extracts and fractions contain multiple constituents, making it difficult to determine whether observed effects result from a single active molecule, additive actions, synergistic interactions, or nonspecific cytotoxicity. Fourth, normal-cell controls have not been consistently incorporated into anticancer studies, limiting assessment of tumor selectivity and therapeutic index. Fifth, comprehensive repeated-dose, chronic, reproductive, genotoxic, and herb–drug interaction studies remain insufficient.

The safety issue is especially important because anticancer activity alone is not adequate for drug-development relevance. A phytochemical or extract that kills malignant cells at concentrations similar to those toxic to normal tissues has limited therapeutic value. Future studies should therefore determine selectivity indices using matched normal-cell controls, compare active preparations with established anticancer agents, characterize concentration-response relationships, and establish maximum tolerated doses in appropriate animal models. Parallel pharmacokinetic studies should determine whether concentrations associated with in-vitro activity are achievable in vivo.

From a translational perspective, the findings support a staged research pathway. Initial work should focus on authenticated botanical material and chemically standardized extracts. Bioactivity-guided fractionation should then identify the constituents responsible for anticancer and hepatoprotective effects. Candidate molecules should undergo target-specific mechanistic validation followed by pharmacokinetic, toxicological, and efficacy studies in multiple validated animal models. Only after reproducible activity and an acceptable safety margin have been established should standardized

formulations progress to early-phase human investigation.

Overall, the present evidence places *C. reflexa* at an important but still preclinical stage of natural-product drug development. Its phytochemical diversity, experimentally demonstrated tumor-growth inhibition, modulation of apoptosis-related proteins, suppression of inflammatory signalling, ROS-associated anticancer activity, and hepatoprotective effects collectively justify continued investigation. At the same time, the lack of standardized preparations, limited compound-specific evidence, inadequate pharmacokinetic characterization, incomplete toxicological assessment, and absence of established clinical efficacy necessitate cautious interpretation. The greatest future value of *C. reflexa* research will therefore depend not on accumulating additional nonspecific screening studies, but on developing chemically defined preparations, validating molecular targets, establishing reproducibility and safety, and systematically progressing the strongest candidates toward translational and clinical investigation.

CONCLUSION

Cuscuta reflexa Roxb. (Tukhme Kasoos/Aftimoon) represents a pharmacologically promising medicinal plant with a long history of traditional use and an expanding body of contemporary experimental evidence. The available findings demonstrate substantial phytochemical diversity, including phenolic compounds, flavonoids, coumarin-related constituents, sterols, glycosides, and other secondary metabolites that may contribute collectively to its biological activities.

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