



Research Article:

HERB-DRUG INTERACTIONS: A REVIEW OF CURRENT EVIDENCE

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Abstract:

Herb–drug interactions (HDIs) occur when herbal medicines alter the pharmacokinetic or pharmacodynamic properties of conventional drugs, potentially leading to adverse reactions or reduced therapeutic efficacy. With the increasing integration of Ayurvedic herbs into contemporary healthcare, understanding these interactions has become essential for patient safety. The present review aimed to evaluate commonly reported herb–drug interactions associated with widely used Ayurvedic medicinal plants and to explore the role of emerging technologies in predicting and managing these risks. A systematic literature search was conducted using PubMed, Scopus, and Google Scholar, focusing on peer-reviewed articles published within the last decade, including clinical studies, experimental research, and review papers. Data were analyzed regarding frequently used herbs, mechanisms of interaction, and clinical implications. Commonly consumed herbs such as *Allium sativum*, *Zingiber officinale*, and *Curcuma longa* were found to interact with anticoagulants, antidiabetic agents, antihypertensives, and drugs metabolized through cytochrome P450 enzymes, resulting in increased bleeding



risk, altered glycemic control, and modified drug bioavailability. Additionally, advancements in pharmacogenomics, artificial intelligence-based interaction screening tools, and pharmacovigilance systems demonstrate potential in improving early detection and prevention of HDIs. The study highlights the necessity of systematic evaluation, clinician awareness, and technological integration to ensure the safe and evidence-based use of Ayurvedic and modern medicines in combination.

Keywords: Herb–drug interactions, Ayurveda, Pharmacokinetics, Pharmacodynamics, Cytochrome P450, Pharmacovigilance

Introduction

Herbal medicines constitute a significant component of primary healthcare systems worldwide. According to the World Health Organization, nearly 80% of the global population relies on herbal preparations for disease prevention and treatment. (1,2) The widespread acceptance and accessibility of these remedies have resulted in their frequent concomitant use with allopathic medicines, particularly among patients with chronic disorders such as diabetes mellitus, hypertension, and arthritis, where prolonged pharmacotherapy is common. (3–5)

Although herbal products are often perceived as natural and inherently safe, accumulating scientific evidence demonstrates that their bioactive phytoconstituents can significantly influence the absorption, metabolism, distribution, or pharmacological activity of conventional drugs. Such interactions, termed herb–drug interactions (HDIs), may potentiate or diminish therapeutic efficacy and, in certain circumstances, precipitate adverse or toxic effects. (3–5) Consequently, a comprehensive understanding of HDIs from both Ayurvedic and modern pharmacological perspectives is essential to ensure patient safety and therapeutic effectiveness in integrative healthcare practice.

Principles governing drug safety, compatibility, and adverse reactions were systematically articulated in Ayurveda several millennia ago. Classical treatises such as the *Charaka Samhita* and *Sushruta Samhita* emphasize that improper combinations of foods, drugs, or therapeutic



procedures—collectively described as *Viruddha Ahara* (incompatible intake)—can produce deleterious outcomes, even when the individual components are wholesome. (6,7)

Related doctrines, including *Matra Viruddha* (incompatible dosage), *Kala Viruddha* (inappropriate timing), and *Anupana Viruddha* (incorrect adjuvant or vehicle), describe mechanisms closely analogous to modern pharmacological concepts such as overdose toxicity, chronopharmacological variability, and excipient incompatibility.⁸ Ayurveda also recognizes *Vyapada*, a term denoting adverse effects or toxic manifestations arising from such incompatibilities. (9) Collectively, these principles constitute an early risk-assessment and safety-monitoring framework comparable to contemporary pharmacovigilance and drug–drug interaction surveillance systems. (10) Interpreting these classical Sanskrit doctrines through the lens of modern pharmacology enhances the conceptual understanding of HDIs and supports safer integrative therapeutic strategies.

The present review aimed to evaluate commonly reported herb–drug interactions associated with widely used Ayurvedic medicinal plants and to explore the role of emerging technologies in predicting and managing these risks.

Methodology

A systematic literature search was conducted to identify and evaluate evidence regarding herb–drug interactions and their clinical significance. The search strategy included three major electronic databases: PubMed, Scopus, and Google Scholar. Peer-reviewed articles published within the last ten years were considered to ensure the inclusion of current and relevant scientific evidence. The search terms comprised combinations of keywords such as “herbal medicine,” “medicinal plants,” “herb–drug interactions,” “pharmacokinetics,” “pharmacodynamics,” “drug metabolism,” “clinical outcomes,” and “adverse effects.”

Studies eligible for inclusion encompassed clinical trials, observational studies, case reports, experimental investigations (in vitro and in vivo), and review articles that addressed the mechanisms, prevalence, or clinical consequences of herb–drug interactions. Articles not published in English, conference abstracts without full-text availability, duplicate records, and studies lacking sufficient methodological details were excluded. The titles and abstracts of



retrieved articles were screened for relevance, followed by a full-text assessment of potentially eligible studies.

Data extraction focused on commonly used medicinal herbs, interacting pharmaceutical agents, underlying mechanisms of interaction, and reported clinical outcomes. Attention was given to pharmacokinetic interactions involving drug-metabolizing enzymes, especially cytochrome P450 isoenzymes, and drug transporters such as P-glycoprotein, as well as pharmacodynamic interactions that could alter therapeutic efficacy or increase the risk of adverse reactions.

The collected evidence was synthesized qualitatively to identify patterns and trends in herb–drug interactions. Frequently reported herbs, including those associated with significant alterations in drug absorption, distribution, metabolism, and excretion, were highlighted. Furthermore, the clinical implications of these interactions were evaluated in terms of patient safety, therapeutic effectiveness, and healthcare management. This comprehensive approach enabled a critical assessment of current knowledge and provided insights into the importance of monitoring herbal medicine use in patients receiving conventional pharmacotherapy.

Literature Review

Parallels Between Ayurvedic Concepts and Modern Pharmacology

Ayurvedic descriptions of drug incompatibility and adverse reactions demonstrate striking parallels with contemporary pharmacological mechanisms underlying HDIs. The concept of *Viruddha Ahara*, which encompasses incompatibility related to substance, timing, or processing, aligns with pharmacokinetic alterations such as enzyme induction or inhibition and changes in drug absorption. (11) Similarly, *Matra Viruddha* (incorrect dosage) and *Ati-Matra Prayoga* (over-administration) correspond to dose-dependent toxicity recognized in biomedical science. (12)

The doctrine of *Karma Viruddha*, wherein the inherent actions (*Karma*) of two substances oppose one another, mirrors pharmacodynamic antagonism, in which opposing receptor-mediated effects negate therapeutic benefit. (13) Furthermore, Ayurvedic determinants of drug response such as *Agnibala* (metabolic capacity) and *Deha Prakruti* (constitutional type) parallel modern concepts of genetic polymorphisms and metabolic phenotypes influencing



cytochrome P450 (CYP450) enzyme activity. (14,15) These classical insights anticipate the principles of personalized medicine and pharmacogenomics, underscoring Ayurveda's sophisticated understanding of drug safety long before the advent of molecular pharmacology.

Mechanistic Classification of Herb–Drug Interactions

Herb–drug interactions can be broadly classified into pharmacokinetic and pharmacodynamic mechanisms, both of which have clear analogues within Ayurvedic pharmacology. Pharmacokinetic interactions involve alterations in absorption, distribution, metabolism, or excretion (ADME), whereas pharmacodynamic interactions arise from additive, synergistic, or antagonistic effects at shared physiological targets. (16) Ayurveda conceptualizes these processes through doctrines such as *Ahara-paka* (digestive transformation), *Vyavaya* (systemic diffusion), *Paka Prakriya* (metabolic conversion), and *Mala Vikruti* (altered excretion).

Pharmacokinetic Interactions: Ahara-Paka and Paka Prakriya

Alterations in drug concentrations frequently result from modulation of metabolic enzymes, particularly CYP450 isoenzymes, and efflux transporters such as P-glycoprotein (P-gp). (17) For example, *Hypericum perforatum* (St. John's Wort) induces CYP3A4 and P-gp, accelerating the metabolism of drugs such as cyclosporine, oral contraceptives, and statins. (18) Conversely, *Hydrastis canadensis* (Goldenseal) inhibits CYP3A4, thereby increasing plasma concentrations of co-administered medications. (19)

Ayurvedic herbs such as *Piper nigrum* (Maricha) and *Curcuma longa* (Haridra) inhibit intestinal P-gp, enhancing the bioavailability of concomitant drugs—an effect consistent with the Ayurvedic concept of *Yogavahi Guna*, whereby a substance acts as a carrier or amplifier of another's action. (20)

Ayurvedic correlates include:

- *Grahani Vyapada* (impaired assimilation) → reduced absorption
- *Agnibala Hani* (diminished metabolic strength) → slowed biotransformation
- *Mala Vikruti* (disturbed excretion) → prolonged drug half-life

Pharmacodynamic Interactions: Guna–Virya Samyoga and Viruddha Prabhava



Pharmacodynamic HDIs occur when a herb modifies a drug’s effect without altering its systemic concentration. Ayurvedic concepts such as *Samyoga Guṇa* (additive or synergistic effects) and *Viruddha Prabhava* (opposing actions) describe these interactions. (21) For instance, *Terminalia arjuna* potentiates the hypotensive effects of beta-blockers, whereas *Glycyrrhiza glabra* antagonizes diuretics by promoting sodium retention. (22) Similarly, *Allium sativum* (Lasuna) and *Ginkgo biloba* increase bleeding risk when combined with anticoagulants through additive platelet inhibition. (23) Such combinations are classified in Ayurveda as *Karma Viruddha* (functional incompatibility).

Enzyme and Transporter Modulation: Ayurvedic Analogies

Enzyme/Transporter	Modulating Herbs	Effect	Ayurvedic Analogy
CYP3A4	St. John’s Wort (induction), Goldenseal (inhibition)	Alters metabolism of >50% of drugs	<i>Agnibala Vaikruti</i>
CYP2C9	<i>Curcuma longa</i> , Milk Thistle	Affects warfarin, phenytoin	<i>Karma Viruddha</i>
CYP1A2	<i>Withania somnifera</i> , Echinacea	Alters clearance of caffeine, antidepressants	<i>Prakruti Vaishamya</i>
P-glycoprotein	Piper nigrum, Curcumin	Increases bioavailability	<i>Yogavahi Guṇa</i>

Ayurvedic Perspective on Drug Compatibility (Viruddha Aushadha)

Ayurveda presents an elaborate framework for assessing drug compatibility and incompatibility under the concept of *Viruddha Aushadha*, encompassing many principles now central to modern pharmacology. (25–27) Classical authorities such as Charaka and Sushruta cautioned that inappropriate combinations of drugs, foods, or vehicles may convert otherwise beneficial substances into toxic agents. (28,29)

Ayurvedic Category	Definition	Modern Parallel
<i>Dravya Viruddha</i>	Substance incompatibility	Chemical antagonism



Karma Viruddha	Opposing therapeutic action	Pharmacodynamic antagonism
Kala Viruddha	Improper timing	Chronopharmacology
Matra Viruddha	Incorrect dosage	Toxic overdose or underdosing
Anupana Viruddha	Inappropriate vehicle	Drug–excipient incompatibility

These doctrines collectively represent an early and sophisticated pharmacovigilance model in which substance, dose, timing, and vehicle determine therapeutic compatibility and safety. (33)

Herb–Drug Interaction Case Examples

Ayurvedic drug	Interacting drug(s)	Nature of interaction
<i>Allium sativum</i> L. (Rason)	Warfarin	Enhances anticoagulant activity, increasing bleeding risk and prolonging clotting time. (34)
<i>Aloe vera</i> (L.) Burm.f. (Kumari)	Cardiac glycosides, quinidine, thiazide diuretics, corticosteroids, liquorice	Laxative action reduces drug absorption; hypokalemia enhances cardiotoxic and antiarrhythmic effects. (35,36)
<i>Cinnamomum cassia</i> Blume (Dalchini)	Tetracycline hydrochloride	Reduces solubility and in vitro bioavailability of tetracycline; clinical significance uncertain. (37,38)
<i>Ephedra sinica</i> Stapf (Soma)	Cardiac glycosides, halothane, guanethidine, MAO inhibitors, ergot alkaloids, oxytocin	May precipitate arrhythmias, potentiate sympathomimetic effects, and induce severe hypertension. (39,40)
<i>Glycyrrhiza glabra</i> L. (Yashtimadhu)	Diuretics, cardiac glycosides, spironolactone, amiloride	Causes potassium depletion, increasing glycoside toxicity and reducing efficacy of potassium-sparing diuretics. (41,42)



<i>Rauvolfia serpentina</i> (L.) Benth. ex Kurz (Sarpagandha)	CNS depressants, antihypertensives, diuretics, digitalis, quinidine, MAOIs, TCAs, anaesthetics	Potentiates sedative and hypotensive effects; may cause marked blood pressure reduction. (43-45)
<i>Cassia senna</i> L. (Sonamukhi)	Cardiac glycosides, quinidine	Increased intestinal motility reduces drug absorption; hypokalemia enhances cardiotoxicity. (46,47)
<i>Zingiber officinale</i> Roscoe (Adrak)	Anticoagulants, antiplatelet agents	Inhibits platelet aggregation; high doses may potentiate anticoagulant effects. (48,49)
<i>Azadirachta indica</i> A. Juss. (Nimba)	Insulin, oral antidiabetic drugs	Potentiates hypoglycemic activity, increasing the risk of hypoglycemia. (50)
<i>Carthamus tinctorius</i> L. (Kusumbha)	Anticoagulants	Exhibits antiplatelet activity, increasing the risk of bleeding. (51)
<i>Crocus sativus</i> L. (Kumkuma)	Anticoagulants	Inhibits platelet aggregation and may enhance anticoagulant effects. (52)
<i>Plantago ovata</i> Forsk. (Isabgol)	Lithium, Minerals, Vitamin B ₁₂	Reduces absorption of drugs and nutrients; decreases lithium bioavailability. (53,54)
<i>Taraxacum officinale</i> Weber ex Wiggers (Dugdhapheni)	Ciprofloxacin	Decreases plasma ciprofloxacin concentration in animal studies. (55)



<i>Trigonella foenum-graecum</i> L. (Methika)	Insulin, hypoglycemics oral	Alters glucose metabolism and enhances the effects of hypoglycemic drugs. (56)
<i>Althaea officinalis</i> L. (Khatmi)	Orally administered drugs	Delays drug absorption due to high mucilage content. (57)
<i>Andrographis paniculata</i> (Burm.f.) Nees (Kalmegh)	Isoniazid	Demonstrates synergistic interaction, potentially enhancing therapeutic efficacy. (58)
<i>Eucalyptus globulus</i> Labill. (Tailaparna)	Hepatically metabolized drugs	Induces drug-metabolizing enzymes, potentially reducing drug efficacy. (59)
<i>Hypericum perforatum</i> L. (Naagratha, Vasant)	Digoxin, warfarin, cyclosporine, theophylline, SSRIs, antiretrovirals	Induces CYP450 enzymes, reducing plasma drug levels and increasing the risk of serotonin syndrome. (60-65)
<i>Ocimum sanctum</i> L. (Tulasi)	Paracetamol	May exacerbate hepatotoxicity through glutathione depletion. (66)
<i>Vitex agnus-castus</i> L. (Sindhuvarya)	Dopamine antagonists, hormonal contraceptives	Reduces the efficacy of dopamine antagonists and alters hormonal drug effects. (67,68)
<i>Berberis vulgaris</i> L. (Daruharidra)	Chemotherapeutic agents, Cyclosporine	May induce chemoresistance via P-glycoprotein and increase cyclosporine levels. (69,70)
<i>Momordica charantia</i> L. (Karavella)	Antidiabetic drugs	Potentiates hypoglycemic activity, increasing risk of hypoglycemia. (71)



<i>Ricinus communis</i> L. (Eranda)	Cardiac glycosides, Diuretics, Fat-soluble Vitamins	Alters drug absorption and electrolyte balance, increasing toxicity risk. (72)
<i>Rosmarinus officinalis</i> L. (Rusmari)	CYP450-substrate drugs	Induces hepatic enzymes, altering the pharmacokinetics of co-administered drugs. (73,74)
<i>Trifolium pratense</i> L. (Tripatra)	Tamoxifen	Isoflavones may modulate the anti-estrogenic effect of tamoxifen. (75,76)
<i>Withania somnifera</i> (L.) Dunal (Ashwagandha)	Barbiturates, Benzodiazepines	Potentiates barbiturates and reduces the efficacy of certain benzodiazepines. (77)
<i>Carica papaya</i> L. (Erandakarkati)	Oral Antidiabetics, Certain Antibiotics, P-gp substrates	Alters drug absorption and transport mechanisms. (78)
<i>Convolvulus pluricaulis</i> Choisy (Shankhapushpi)	Phenytoin	Reduces plasma concentration and anticonvulsant efficacy of phenytoin. (79)
<i>Tamarindus indica</i> L. (Amlika)	Aspirin	Enhances the bioavailability of aspirin when co-administered with food. (80)
<i>Mentha piperita</i> L. (Putiha)	Felodipine, Nifedipine	Inhibits drug metabolism, increasing plasma drug concentrations. (81)
<i>Asparagus racemosus</i> Willd. (Shatavari)	Tolbutamide	Potentiates the insulin- stimulating action of tolbutamide. (82)



<i>Glycine max</i> (L.) Merr. (<i>Raja Shimbi</i>)	Thyroid hormones	Reduces absorption of thyroid medications. (83)
<i>Camellia sinensis</i> (L.) Kuntze (<i>Syamaparni</i>)	Antihypertensive drugs	Mildly elevates blood pressure, potentially reducing drug efficacy. (84)
<i>Curcuma longa</i> L. (<i>Haridra</i>)	P-glycoprotein substrate drugs	Modulates drug absorption via P-glycoprotein interaction. (85)
<i>Valeriana officinalis</i> L. (<i>Tagara</i>)	Sedative-hypnotics	Potentiates the sedative and hypnotic effects of barbiturates. (86)
<i>Sida cordifolia</i> L. (<i>Bala</i>)	Caffeine, Antihypertensives	Concomitant use may reduce antihypertensive drug potency. (87)

Discussion

The concurrent use of herbal medicines and conventional pharmaceutical drugs has increased significantly worldwide due to the growing acceptance of Ayurveda and other traditional systems of medicine. Although Ayurvedic medicines are generally regarded as safe when used according to classical principles and under professional supervision, their combined use with modern medications may lead to herb–drug interactions (HDIs). These interactions can influence the pharmacokinetics or pharmacodynamics of drugs, potentially altering therapeutic efficacy and safety.

Pharmacokinetic interactions are among the most commonly reported mechanisms of HDIs. Herbal constituents may affect drug absorption, distribution, metabolism, and excretion by modulating drug-metabolizing enzymes such as cytochrome P450 (CYP450) and transport proteins including P-glycoprotein. Such modifications can result in increased or decreased drug bioavailability, leading either to toxicity or reduced therapeutic effectiveness. Pharmacodynamic interactions may also occur when herbal medicines and conventional drugs



exert similar or opposing pharmacological effects. These interactions can produce additive, synergistic, or antagonistic responses, thereby influencing clinical outcomes.

Several commonly used Ayurvedic herbs have demonstrated the potential to interact with pharmaceutical agents. *Withania somnifera* (Ashwagandha), widely used for its adaptogenic and anxiolytic properties, may enhance the sedative effects of central nervous system depressants. *Glycyrrhiza glabra* (Licorice) has been reported to influence corticosteroid metabolism and electrolyte balance, potentially increasing the risk of hypertension and hypokalemia. Similarly, *Allium sativum* (Garlic), known for its cardiovascular benefits, possesses antiplatelet activity and may potentiate the effects of anticoagulant drugs such as warfarin, thereby increasing the risk of bleeding.

Despite growing interest in HDIs, most available evidence is derived from in vitro studies, animal experiments, and case reports. Well-designed clinical trials remain limited, making it difficult to accurately determine the frequency and clinical significance of many reported interactions. Furthermore, variability in herbal product composition, dosage, and patient-specific factors contributes to the complexity of assessing HDIs.

To minimize risks, healthcare professionals should obtain comprehensive medication histories that include herbal products and dietary supplements. Patient education, regular monitoring, use of validated interaction databases, and adherence to recommended dosages are essential preventive strategies. Emerging technologies such as artificial intelligence, pharmacovigilance systems, genomic profiling, and clinical decision-support tools offer promising approaches for improving the detection and prediction of herb–drug interactions. Continued clinical research and interdisciplinary collaboration are necessary to ensure the safe integration of Ayurvedic medicines with modern pharmacotherapy and to optimize patient outcomes.

Conclusion

Herb–drug interactions (HDIs) represent an important and increasingly relevant aspect of healthcare due to the widespread use of herbal medicines alongside conventional pharmaceutical therapies. The growing popularity of Ayurveda and other traditional systems of medicine has highlighted the need for a better understanding of the potential interactions between herbal products and modern drugs. While many medicinal herbs provide significant



therapeutic benefits and have a long history of traditional use, their biologically active constituents may alter the pharmacokinetic or pharmacodynamic properties of conventional medications, potentially affecting treatment efficacy and patient safety.

This review demonstrates that HDIs can occur through multiple mechanisms, including alterations in drug absorption, metabolism, distribution, excretion, and pharmacological action. Several commonly used Ayurvedic herbs have shown the potential to interact with conventional medications, emphasizing the importance of careful assessment and monitoring in clinical practice. However, the current body of evidence is largely based on experimental studies, case reports, and limited clinical investigations, highlighting the need for more robust and well-designed clinical research to establish the true significance of these interactions.

Ayurvedic principles of individualized treatment, appropriate dosage, and rational drug selection provide valuable insights that align with modern concepts of personalized medicine. Integrating these traditional principles with contemporary pharmacological knowledge may contribute to safer and more effective therapeutic approaches. Furthermore, advancements in artificial intelligence, bioinformatics, pharmacogenomics, and pharmacovigilance systems offer promising opportunities for improving the detection, prediction, and management of HDIs.

To ensure patient safety, healthcare professionals should encourage open communication regarding herbal medicine use, provide appropriate counselling, and utilize evidence-based resources when evaluating potential interactions. Increased awareness among practitioners and patients, combined with continuous monitoring and reporting of adverse events, is essential for minimizing risks. Future research should focus on bridging traditional knowledge with scientific evidence, thereby supporting the development of safe, effective, and evidence-based integrative healthcare practices that maximize therapeutic benefits while reducing the likelihood of adverse outcomes.

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