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Interplay of medicinal plants, toxicants, and chronic diseases: An integrative review

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ABSTRACT

Chronic diseases result from complex interactions among genetic factors, environmental toxins like metals and air pollutants, lifestyle choices, and our overall exposure. At the same time, medicinal plants and their active components have various effects that may counteract or sometimes worsen these toxin-related processes. Many phytochemicals influence oxidative stress, inflammation, metabolism of foreign substances, hormonal signaling, genetic programming, mitochondrial function, and the gut-microbiome-liver connection—all of which can be disrupted by toxins. The most substantial evidence suggests that polyphenols, alkaloids, terpenoids, and sulfur compounds, such as curcumin, quercetin, resveratrol, berberine, silymarin, EGCG, and sulforaphane, are effective, although their clinical applications vary. These interactions can include benefits such as activating Nrf2, chelating metals, and reshaping the microbiome, as well as risks including inhibiting CYP/P-gp, producing oxidative effects at high doses, and contamination. Plant-based interventions show potential for countering the harmful effects of toxins in chronic conditions like heart disease, liver disease, kidney issues, neurodegenerative disorders, immune problems, and cancer. Future research should focus on trials that consider exposure, validated biomarkers, and integrated methods that connect exposomics and pharmacology.

Keywords: Exposome, Endocrine disruptors, Epigenetics, Oxidative stress, Xenobiotic metabolism, Gut microbiome, Polyphenols, Nrf2, NF-κB, Herb–drug interaction.
Article type: Review Article.

INTRODUCTION

Chronic diseases are the leading cause of global illness and death (heart disease, type 2 diabetes, chronic kidney disease, neurodegenerative diseases, autoimmune diseases, and cancer). Contrastingly, infectious diseases usually have a single cause. Chronic diseases vary significantly and are attributable to combinations of many different factors, such as genetics, lifestyle choices, and environmental factors. Some of the factors that contribute to chronic diseases, namely toxic substances (heavy metals, persistent organic pollutants (POPs), pesticides, endocrine-disrupting chemicals (EDCs), and particulate matter (PM)), have been increasingly seen to play a role in the development and progression of ill health (Cosmin Stan & Paul 2024). The exposome framework describes the total lifetime of environmental exposures, and using this framework allows a better understanding of how ubiquitous toxicants overlap and interact with human biology to influence chronic disease trajectories. These exposures are also often meant to be understood as interacting synergistically and in combination with a poor diet, limited exercise, and psychosocial stress, contributing to complex disease trajectories (Bolognesi *et al*. 2022). Many of the phytochemicals activate antioxidant response elements (ARE), integrate inflammatory mediation (Nuclear factor Kappa beta (NFκβ)), facilitate mitochondrial dynamics (mitophagy), influence epigenetic regulation, and support the development of the gut microbiome community (Bahmani *et al*. 2014; Widoyo *et al*. 2022). We maintain that the relationship between medicinal plants and toxicants must be understood as network pharmacology and exposure biology. Instead of acting independently, both phytochemicals and toxicants modify interrelated molecular networks that include other substances and exposures, creating some level of resilience or vulnerability to chronic disease.

The toxicant landscape relevant to chronic disease

Chronic diseases arise from long-term dynamic interactions between human biology and the environment. Indeed, the environment includes toxicants—as chemical or physical agents that, through acute or chronic exposure, undermine cellular homeostasis, resulting in long-term health impacts. While acute poisoning occurs from episodic acute exposure at a high dose, chronic low-dose exposure typically does not produce immediate symptoms; instead, chronic poisoning modifies metabolic, inflammatory, endocrine, and epigenetic networks creating a susceptibility to noncommunicable disease (NCD). In this section, major classes of toxicants relevant to chronic disease will be introduced, highlighting important information regarding sources of exposure, molecular mechanisms, and epidemiological associations.

Metals and Metalloids

Lead (Pb)

Lead is a worldwide public health issue, even after decades of regulatory limitations. Lead accumulates in bone, serving as a long-term depot; however, lead exerts toxic effects through calcium mimicry, inhibition of heme synthesis, oxidative stress, and disruption of synaptic signaling. Chronic lead exposure is strongly correlated to hypertension, chronic kidney disease (CKD), and neurocognitive decline (Ali *et al*. 2019).

Cadmium (Cd)

Cadmium exposure primarily is from tobacco smoke, rice, and seafood. These foods reflect where we see cadmium bring primarily present - namely phosphate fertilizers and industrial emissions. Cadmium binds to metallothioneins and accumulates in renal tubular cells, causing mitochondrial dysfunction and oxidative stress (Amanpour *et al*. 2024).

Mercury (Hg)

Methylmercury, the organic form bioaccumulated in fish and seafood, is the most serious public health concern when it comes to mercury exposure. Methylmercury easily crosses the blood–brain and placental barriers and its neurotoxic effects are attributed to glutamate excitotoxicity, microtubule disruption, and oxidative damage. Chronic low-level mercury exposure has been linked to neurodevelopmental deficits, cardiovascular disease and immune dysfunction (Rattan *et al*. 2017; Manouchehri *et al*. 2022).

Arsenic (As)

Widespread arsenic exposure occurs from naturally occurring sources in aquifers throughout South Asia, Latin America, and parts of the Middle East (including some US locations). Arsenic is a problem for millions of people around the world through both contaminated water and food (for example, rice). Arsenic affects mitochondrial respiration, produces oxidative damage to DNA, and changes DNA methylation patterns. Chronic arsenic exposure is causally linked to skin lesions, cancers of the lung and bladder, cardiovascular disease, and diabetes mellitus (Abu Bakar *et al.* 2025).

Persistent Organic Pollutants (POPs) and PFAS

POPs (polychlorinated biphenyls [PCBs], dioxins, organochlorine pesticides)

POPs are extremely lipophilic, bioaccumulate in fat tissue, and can persist for decades. POPs exert action mainly through activating the aryl hydrocarbon receptor (AhR), which alters xenobiotic metabolism and signaling in immune cells. Epidemiological evidence suggests that POPs may be related to metabolic syndrome, diabetes, dyslipidemia, and immune dysregulation. Dioxins (i.e., TCDD) have been associated with increased risk of certain cancers and reproductive toxicity (Gheibi *et al*. 2020, 2023).

Per- and polyfluoroalkyl substances (PFAS)

PFAS, often referred to as "forever chemicals," can be found in drinking water, food packaging, textiles, and firefighting foams. They disrupt peroxisome proliferator-activated receptors (PPARs), thyroid hormone signaling, and bile acid metabolism (Briassoulis *et al.* 2025).

Air pollution and combustion products

Particulate matter ($PM_{2.5}$ and ultrafine particles)

Air pollution is now recognized as the leading risk factor for environment-related mortality globally. Fine and ultrafine particles are inhaled deeply into the lungs and then translocated into the circulation, causing systemic oxidative stress, vascular inflammation, endothelial dysfunction, and autonomic imbalance. Chronic exposure increases the risk of cardiovascular disease, type 2 diabetes, neurodegeneration, and certain cancers (Noshadirad *et al*. 2023).

Ozone, nitrogen oxides, and polycyclic aromatic hydrocarbons (PAHs)

These combustion-related pollutants amplify oxidative stress and DNA adduct formation. PAHs, generated by incomplete combustion of fossil fuels and biomass, are potent mutagens and carcinogens. Ozone exposure aggravates asthma and chronic obstructive pulmonary disease (COPD), and may accelerate atherosclerosis (Paget-Bailly *et al.* 2012).

Pesticides and Solvents

Organophosphates (OPs)

Widely used as insecticides, OPs irreversibly inhibit acetylcholinesterase, leading to acute cholinergic toxicity. Chronic low-dose exposure is associated with neurobehavioral impairment, endocrine disruption, and possibly Parkinson’s disease (Barzi *et al*. 2020).

Pyrethroids and glyphosate

Pyrethroids, though considered safer alternatives, are linked to neurotoxicity and oxidative stress in animal studies. Glyphosate, the most widely used herbicide, has raised concern due to potential carcinogenicity, gut microbiome disruption, and endocrine effects, although human evidence remains debated (Rattan *et al*. 2017).

Industrial solvents

Organic solvents, such as trichloroethylene, benzene, and toluene, are associated with hepatotoxicity, nephrotoxicity, hematologic malignancies, and autoimmune diseases. Mechanisms include DNA adduct formation, oxidative injury, and immune dysregulation (Barzi *et al*. 2020).

Food-contact chemicals and additives

Bisphenols (BPA, BPS, and BPF)

These endocrine-disrupting chemicals are used in plastics, resins, and food packaging. They mimic estrogen, antagonize androgen receptors, and interfere with thyroid signaling. Prenatal and early-life exposure is linked to obesity, insulin resistance, neurodevelopmental issues, and reproductive disorders (Tuzimski *et al*. 2025).

Phthalates

Used as plasticizers and in personal care products, phthalates are ubiquitous in human urine. They alter steroidogenesis and activate peroxisome proliferator-activated receptors. Epidemiological studies connect phthalates to metabolic syndrome, reduced fertility, and adverse developmental outcomes (Hlisníková *et al*. 2020).

Emerging Toxicants

Micro- and nanoplastics

Plastic particles are now detected in water, air, soil, and food. They can act as carriers for other toxicants and induce oxidative stress, inflammation, and barrier dysfunction in animal models. Human health implications are under investigation, but ingestion and inhalation are widespread (Yan *et al*. 2019).

Nanomaterials

Engineered nanomaterials (e.g., carbon nanotubes, silver nanoparticles) offer technological benefits but pose risks of pulmonary inflammation, fibrosis, and genotoxicity. Long-term effects are poorly characterized (Mishra *et al.* 2018).

Mixture toxicity and the exposome perspective

Exposure to a single toxicant in the environment is rare. Consumers are more likely to encounter multiple toxicants that could interact additively, synergistically, or antagonistically. For example, the effects of metals on persistent organic pollutants make oxidative stress worse and vice versa. Air pollution could also alter the pharmacokinetics or bioavailability of pesticides.

Medicinal plants and bioactive classes (Table 1)

Medicinal plants have an extreme variety of secondary metabolites, and their specific life functions include the ecological roles of plant defense against pathogens, herbivores, and invading stressors. For human health, these represent chemically diverse scaffolds with pleotropic biological activities. Phytochemicals are dissimilar to synthetic drugs that often target only a specific molecular pathway (Widoyo *et al*. 2022).

Polyphenols

Flavonoids

Flavonoids are the largest subclass of polyphenols and are prevalent in fruits, vegetables, tea, and cocoa. Some of the most extensively studied flavonoids include quercetin, kaempferol, catechins (e.g., epigallocatechin gallate, or EGCG), and anthocyanins. Flavonoids exert biological effects through mechanisms that involve scavenging reactive oxygen species (ROS), chelating transition metals, and modulating important transcription factors such as nuclear factor erythroid 2–related factor 2 (Nrf2) and nuclear factor kappa B (NF-κB; Samanta *et al.* 2011).

Stilbenes

Resveratrol, which is commonly found in grapes, blueberries, and blackberries, stimulates sirtuin-1 (SIRT1) and AMP-activated protein kinase (AMPK). This action improves mitochondrial energy production and insulin sensitivity. Resveratrol also shows chemo-preventative effects and boosts the effectiveness and tolerability of chemotherapy by affecting p53, NF-κB, histones, and various epigenetic regulators like histone deacetylases (HDACs). Animal studies indicate it can reduce oxidative damage caused by cadmium or arsenic.

Curcuminoids

Curcumin from turmeric, *Curcuma longa*, is one the most studied plant compounds to date. It affects a number of signaling pathways, such as Nrf2/Keap1, NF-κB, JAK/STAT, and Wnt/β-catenin. Curcumin has protective effects in models of toxin-induced liver damage, for example, and improves insulin sensitivity in individuals with metabolic syndrome. The clinical and health benefits of curcumin have not been fully realized due to its insolubility and rapid metabolism (Liew *et al*. 2020).

Alkaloids

Berberine

Berberine, an ingredient derived from *Berberis* species and *Coptis chinensis*, is a protoberberine alkaloid that has well-documented effects on metabolism. Berberine has demonstrated beneficial protective effects against nephrotoxocity from heavy metals and oxidative injury from pesticides. Berberine is a substrate for P-glycoprotein (P-gp) or P-glycoprotein (P-gp) modulator, which suggests the potential for herb–drug interactions (Javed Iqbal *et al*. 2020).

Capsaicin

Capsaicin is found in chili peppers and is a strong activator of transient receptor potential vanilloid 1 (TRPV1) channels. It helps with energy use, pain control, and blood vessel function. It may also reduce inflammation caused by toxins by increasing the production of nitric oxide and blocking NF-κB signaling. However, its use in treatment is limited by how well people can tolerate it and the irritation it can cause in the gut (Jiao *et al*. 2015).

Indole alkaloids

Compounds like vincristine and reserpine show the clinical power of alkaloid scaffolds, but they also reveal risks of toxicity. Milder examples, such as harmine, show potential for improving mitochondrial function and neuroplasticity in models of pesticide-induced neurotoxicity (Panahi *et al*. 2018).

Terpenoids

Silymarin complex

Extracted from milk thistle, *Silybum marianum*, silymarin contains compounds called flavonolignans, including silybin, silychristin, and silydianin. Silymarin stabilizes cell membranes, removes free radicals, and boosts Nrf2-mediated antioxidant enzymes. Clinically, it has been used for liver injuries caused by toxins, such as mushroom poisoning and exposure to solvents. Trials in nonalcoholic steatohepatitis (NASH) and viral hepatitis show slight improvements in liver enzymes and tissue structure (Pickova *et al.* 2020).

Boswellic acids

Derived from *Boswellia serrata* (frankincense), boswellic acids inhibit 5-lipoxygenase, reducing leukotriene-mediated inflammation. They show benefit in osteoarthritis and inflammatory bowel disease and may counteract toxicant-induced immune activation. Safety is generally favorable, though gastrointestinal side effects can occur (Majeed *et al*. 2021).

Monoterpenes (limonene, and carvacrol)

These compounds, sourced from citrus peels and oregano, show favorable effects in fighting microbes, reducing oxidation, and preventing cancer. Limonene increases the levels of phase II detoxification enzymes. Carvacrol, based on animal studies, has protective effects against toxicity from pesticides (Sharifnia *et al.* 2023).

Organosulfur compounds

Sulforaphane

Sulforaphane is a strong Nrf2 activator and histone deacetylase (HDAC) inhibitor found in cruciferous vegetables. It boosts the production of glutathione, helps detoxify harmful substances, and changes the way our body responds to inflammation. Studies involving humans show that sulforaphane improves markers of oxidative stress and detoxification. This includes higher urinary excretion of benzene metabolites and compounds from air pollutants (Treasure *et al.* 2023).

Allicin and related thiosulfinates

Allicin is produced when garlic, *Allium sativum* is crushed, and serves as an antimicrobial, antioxidant, and cardioprotective agent. Clinical studies suggest that garlic supplementation decreases blood pressure, lowers cholesterol, and increases detoxification enzymes. The sulfur compounds in garlic may interact with drug metabolism and platelet aggregation, therefore caution should be exercised with patients on anticoagulants (Akter *et al.* 2022).

Polysaccharides and fibers

Beta-glucans

Found in oats, barley, and medicinal mushrooms (e.g., Ganoderma, and Lentinula), beta-glucans modulate innate immunity through dectin-1 and complement receptor 3. They improve lipid and glucose metabolism, and may enhance resilience against toxicant-induced immunosuppression.

Inulin and fructooligosaccharides

Prebiotic fibers provide food for beneficial gut microbiota, stimulate a short-chain fatty acid (SCFA) response, and improve gut barrier functioning. Since the microbiome metabolizes both toxicants and phytochemicals, prebiotic fibers impact exposure–response relationships indirectly.

Lignans, carotenoids, and other classes

Lignans (silybin, and schisandrin)

These compounds modulate estrogen receptors and antioxidant pathways. Schisandrin from *Schisandra chinensis* protects against solvent-induced hepatotoxicity in animal models, while silybin is a key component of silymarin's hepatoprotective properties (Huang *et al*. 2025).

Carotenoids (lycopene, and astaxanthin)

Carotenoids are potent singlet oxygen quenchers with membrane-stabilizing properties. Lycopene intake is associated with reduced risk of prostate cancer and cardiovascular disease. Astaxanthin demonstrates neuroprotective and anti-inflammatory effects in heavy-metal exposure models (Lalehgani *et al.* 2025; Mardani-Nafchi *et al*. 2025).

Mechanistic crosstalk: Shared nodes between toxicants and phytochemicals. (Baird & Yamamoto 2020)**.**

Redox–inflammation axis. Nrf2/Keap1 activation; NF-κB, AP-1 inhibition; NLRP3 inflammasome.

Mitochondrial homeostasis. Biogenesis (PGC-1α), dynamics (DRP1/MFN), mitophagy (PINK1/Parkin).

Xenobiotic metabolism & transport. Phase I/II (CYPs, UGTs, SULTs), GSTs, NQO1; transporters (P-gp/ABCB1, BCRP/ABCG2, OATPs).

Endocrine & metabolic signaling. PPARs, insulin signaling, AMPK–mTOR, ER/AR/thyroid receptors.

Epigenetic Programming. DNA methylation, histone acetylation/acetyltransferases (HDAC/HAT), sirtuins; microRNA regulation.

Barrier function & immunity. gut epithelial tight junctions, mucosal immunity, Treg/Th17 balance.

Microbiome–metabolome. microbial biotransformation of phytochemicals; SCFAs; bile-acid signaling (FXR/TGR5); enterohepatic cycling of toxicants.

Table 1. Medicinal plants/Phytochemicals → Targets → Evidence → Dosing → Cautions

Plant/Compound	Key Targets/Pathways	Evidence Level	Typical Clinical Dosing	Key Safety Notes
Curcumin (standardized)	Nrf2↑, NF-κB↓, HDAC	RCTs (mixed), meta-analyses	500–2,000 mg/day (with enhancer)	GI upset; CYP interactions (piperine)
Berberine	AMPK activation; P-gp substrate	RCTs in T2D/dyslipidemia	500 mg bid–tid	CYP2D6/3A4 interactions; GI
Silymarin (silybin-rich)	Antioxidant; Nrf2; hepatoprotection	Trials in liver disease	140–420 mg/day	Rare GI; allergenic Asteraceae
EGCG (green tea extract)	Antioxidant; AMPK; catechol-O-methyltransferase	Mixed clinical	300–800 mg/day EGCG	Hepatotoxicity at high doses
Quercetin	Nrf2; mast cell stabilization	Emerging	500–1,000 mg/day	CYP2C8; drug interactions
Sulforaphane (broccoli sprout)	Nrf2; HDAC inhibition	Human mechanistic trials	50–150 μmol/day	Thyroid caution in iodine deficiency

Disease-focused evidence maps (Table 2)

Cardiometabolic disease (CVD, T2D, and Metabolic Syndrome; Thangavel *et al.* 2022; Bejenaru *et al*. 2024)

Toxicant links. Metals, POPs, air pollution cause insulin resistance, endothelial dysfunction.

Plant evidence. Berberine (AMPK), resveratrol (SIRT1), quercetin/EGCG (endothelial NO), curcumin (Nrf2/NF-κB), garlic/sulforaphane (Nrf2, H_2S signaling).

Clinical considerations. Lipid and glycemic endpoints; exposure stratification; medication co-use (statins, and metformin).

Liver disease (NAFLD/NASH, toxicant-associated fatty liver disease; Liu *et al*. 2022)

POPs/PFAS, solvents, microplastics; mitochondrial and ER stress.

Plant evidence. silymarin, curcumin, anthocyanins; bile acid signaling and microbiome.

Kidney disease (CKD) & Nephrotoxicity, (Amanpour *et al*. 2024)

Cadmium/lead, solvents; tubular transporters.

Plant evidence. silymarin, curcumin, astragaloside; caution with aristolochic acids.

Neurodegenerative disorders (Luthra & Roy 2022)

Air pollution, metals, pesticides; neuroinflammation and proteostasis.

Plant evidence. EGCG, curcumin, ginsenosides; BBB transport issues.

Cancer. (Srivastava *et al*. 2011)

Genotoxic and epigenetic toxicants; promotion/progression pathways.

Plant evidence: sulforaphane (HDAC inhibition, Nrf2), resveratrol, quercetin; synergy with conventional therapy; potential antagonism or PK interactions.

Immune/autoimmune & inflammatory conditions

EDCs and immune skewing; AhR/Treg/Th17 balance.

Plant evidence. Boswellic acids, curcumin, omega-3-rich botanicals; infection risk and immunotherapy cautions.

Table 2. Toxicants → Sources → Mechanisms → Linked chronic diseases.

Toxicant/Class	Major Sources & Routes	Primary Mechanisms	Biomarkers of Exposure/effect	Chronic diseases
Lead	Water, old paint, soil, spices	Oxidative stress; renal tubular injury; hypertension	Blood lead, δ-ALAD inhibition	CVD, CKD, neurocognitive
Cadmium	Tobacco, rice, shellfish	Mitochondrial dysfunction; metallothionein binding	Blood/urine Cd, β2-microglobulin	CKD, CVD
Mercury (MeHg)	Fish/seafood	Neurotoxicity; oxidative stress	Hair/whole blood Hg	Neurodevelopmental, CVD
Arsenic	Groundwater	DNA damage; epigenetics; diabetes risk	Urinary arsenicals	Skin, CVD, T2D
PFAS	Water, food packaging	PPAR disruption; lipid metabolism	Serum PFOS/PFOA	Dyslipidemia, NAFLD
POPs (PCBs/dioxins)	Food chain	AhR; endocrine disruption	Serum POPs	MetS, cancer
Air pollution (PM2.5)	Ambient/indoor air	Oxidative stress; endothelial dysfunction	Personal monitors; CRP	CVD, neuro
Pesticides	Occupation, diet	Cholinergic/mitochondrial	Urinary metabolites	Neuro, cancer

Interaction space: Herb, toxicant, drug interplay

The use of medicinal plants for therapeutic purposes occurs in a complicated biological landscape, composed of exposure to environmental toxins, pharmaceutical drugs, and natural metabolic activity.

Pharmacokinetic interactions

"Pharmacokinetics" is defined as the absorption, distribution, metabolism, and excretion (ADME) of exogenous substances. Medicinal plants can influence pharmacokinetics by modulating drug-metabolizing enzymes, transporters, and enterohepatic recirculation that affect systemic exposure to co-administered drugs or toxins.

Pharmacodynamic interactions

Pharmacodynamics focuses on the biological effects of foreign substances and how they interact at molecular targets. Medicinal plants can create additive, synergistic, or opposing effects with drugs and toxins through similar or contrasting mechanisms.

Shared molecular targets

Nrf2/Keap1 pathway. Many toxins, like metals, persistent organic pollutants (POPs), and pesticides, induce oxidative stress. Polyphenols (curcumin, resveratrol) and organosulfur compounds (sulforaphane, allicin) activate Nrf2, boosting antioxidant defenses. Co-exposure to toxins may result in additive protective effects, while certain drugs (e.g., chemotherapy drugs) may be weakened by strong antioxidant activity (Baird & Yamamoto 2020).

Inflammatory pathways. COX-2, NF-κB, and MAPK signaling are common targets where herbal compounds (boswellic acids, and flavonoids) and drugs (NSAIDs, and corticosteroids) interact. Synergistic inhibition may reduce inflammation, while antagonism may happen if antioxidants weaken reactive oxygen species (ROS)-dependent drug signaling (de Paula Porto *et al*. 2014).

Endocrine targets. ACE, DPP-4, PPARs, and estrogen/androgen receptors are influenced by both plant compounds and pharmaceutical drugs. For example, berberine and metformin work through AMPK activation, potentially providing extra metabolic benefits. In contrast, botanicals with estrogenic effects (phytoestrogens, lignans) may disrupt endocrine therapies or hormone-related pathways driven by toxins (Zhang *et al*. 2020).

Network-level effects

Because plant compounds often act on multiple signaling pathways at once, their PD interactions can also indirectly influence toxicity. For instance, Nrf2 activation by sulforaphane may reduce liver injury from acetaminophen or environmental metals, while NF-κB inhibition may limit blood vessel inflammation caused by air pollution.

Exposure modifiers

In addition to PK and PD, medicinal plants can change the internal dose of toxins or drugs through processes like chelation, adsorption, or increased excretion.

Chelation

Polyphenols, organosulfur compounds, and flavonoids can bind to transition metals (lead, cadmium, mercury), lowering their availability and cellular entry. For example, quercetin and catechins create stable complexes with cadmium and lead, lessening oxidative damage in liver and kidney cells(Rudrapal *et al*. 2024).

Adsorption and sequestration

Dietary fibers, inulin, pectins, and certain polysaccharides can bind to fat-soluble toxins (PCBs, dioxins) in the digestive system, reducing systemic absorption. This effect may also impact drug availability, which should be tracked (Beigh 2024).

Enhanced excretion

Herbs that stimulate bile flow (like silymarin and artichoke leaf extract) or renal clearance (like dandelion and celery) can help expel conjugated foreign substances. Inducing phase II enzymes and conjugation processes also promotes the removal of harmful and fat-soluble toxins.

Risk scenarios

Hepatotoxic adulterants

Pyrrolizidine alkaloids (Senecio spp., Comfrey), aristolochic acids (Aristolochia spp.), and unregulated extracts can cause both acute and chronic liver damage and kidney toxicity. Being exposed to other hepatotoxic substances (like acetaminophen, solvents, metals) can worsen this damage (Wang *et al*. 2021).

Contamination and mislabeling

Heavy metals, pesticide residues, and microbial contamination are common in unregulated herbal products. Misidentification or adulteration (replacing plants with toxic varieties) can lead to negative outcomes, particularly in vulnerable groups.

Herb–drug–toxicant convergence

Taking drugs, plant compounds, and environmental toxins at the same time can lead to unexpected results. For instance, St. John's wort can activate CYP3A4, speeding up the metabolism of a toxic drug, which may lead to either ineffective treatment or the buildup of harmful metabolites. Similarly, grapefruit-like inhibition of CYP3A4 by flavonoids may increase drug or toxin toxicity (Schäfer *et al.* 2024).

Conclusion

Medicinal plants interact with toxin-driven biology through overlapping molecular and ecological pathways. When rigorously standardized and clinically evaluated while considering exposure and safety, botanical products can provide a practical addition to reduce risk and influence disease progression in chronic conditions.

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