

Plant-Derived Toxins: A Comprehensive Systematic Review of Their Pharmacological and Toxicological Implications

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ABSTRACT

Naturally occurring toxins are chemical substances synthesized by living organisms. While these compounds generally do not harm the host that produces them, they can be toxic to other organisms and serve as a protective strategy against

environmental pressures. In plants, such compounds function as defense mechanisms against insects, herbivores, and microbial invasion, while also helping the plant adapt to stresses such as salinity, drought, or heavy metal exposure. Some of these substances, though harmless to aquatic species like fish and shellfish, may be highly toxic to humans. Examples of naturally occurring toxins include aquatic biotoxins, cyanogenic glycosides, furocoumarins, lectins, mycotoxins, solanine and chaconine, poisonous mushroom toxins, and pyrrolizidine alkaloids. While certain compounds hold therapeutic potential and are even used in drug development, they may also disrupt pharmaceutical manufacturing processes and, in some cases, cause serious toxicity or disease in humans. The purpose of this review is to outline the nature of these naturally derived toxic compounds and to assess their potential impact on human health.

Keywords: Natural Toxin, Microorganisms, Food contaminate, Biotoxic, Micotoxin

Background

The principle posits that all substances are inherently toxic, with the distinction between toxicity and non-toxicity determined solely by the dosage. Consuming large quantities of any substance, such as excessive water intake within a short period, can lead to health risks, such as sodium imbalances in the body¹. Natural products are classified as primary and secondary metabolites, which are produced in small quantities by living organisms. These metabolites can be simple or complex in composition, making their external synthesis in laboratories challenging.²

Primary metabolites are compounds derived from essential metabolic pathways necessary for life. They play a crucial role in the efficiency of vital cells, fulfilling their primary needs and providing the required energy pathways. Examples of primary metabolites include carbohydrates, amino acids, and nucleic acids.³

In contrast, secondary metabolites are non-essential for life and serve various functions, such as transmitting vital signals between cells and aiding in cellular protection against external organisms. Secondary metabolites include alkaloids, saponins, phenols, and terpenes.⁴

Natural Toxins

Natural organisms including flora, algae, and fauna synthesize bioactive compounds

that possess inherent toxicity. These substances are commonly present in botanical food sources and frequently serve as protective mechanisms against herbivory and arthropod invasion. The fundamental toxicological principle established by Paracelsus demonstrates that toxicity is dose-dependent rather than substance-specific. Water exemplifies this concept, as consumption of excessive volumes (4–5 litres) within brief timeframes (2–3 hours) results in water intoxication through hyponatremia, progressing to convulsions, cerebral oedema, and potential mortality. Similarly, vitamin A, despite its antioxidant properties, exhibits hepatotoxicity at excessive dosages and may demonstrate pro-oxidant effects during chronic elevation.⁵ Even *Glycyrrhiza glabra* (licorice), considered benign, poses health risks when consumed excessively. Bannister et al documented a case of hypokalaemia-induced cardiac arrest in a 58-year-old female following chronic consumption of approximately 1.8kg of licorice weekly. Contemporary scientific research has increasingly focused on naturally occurring biotoxins within food and feed systems. The prevalence of these deleterious compounds of biological origin presents escalating public health concerns. Certain natural toxins demonstrate extreme acute toxicity (e.g., botulinum toxin) whilst others exhibit potent carcinogenic properties (e.g., aflatoxins).⁶

To detoxify plants with toxic compounds⁷, one can remove immature green parts in tubers (indicating fungal infection), discard damaged sections, and soak legumes for five hours before boiling them for at least 10 minutes to minimize toxicity.⁸

Naturally occurring toxins in food arise from both plant and animal sources. In plants, well-recognized examples include glycoalkaloids found in potatoes, cyanogenic glycosides present in apricot kernels and bamboo shoots, and lectins and enzyme inhibitors commonly detected in green beans and other legumes.⁹

From the animal kingdom, approximately 1,200 species are known to produce toxic compounds, which can accumulate and pose significant risks to human health when ingested. Consumption of certain fish, such as grass carp, illustrates the importance of careful food handling to prevent toxin-related poisoning.¹⁰

Biologically derived toxins are broadly classified into five principal categories, based on their origin and mechanism of action. Fig.1:

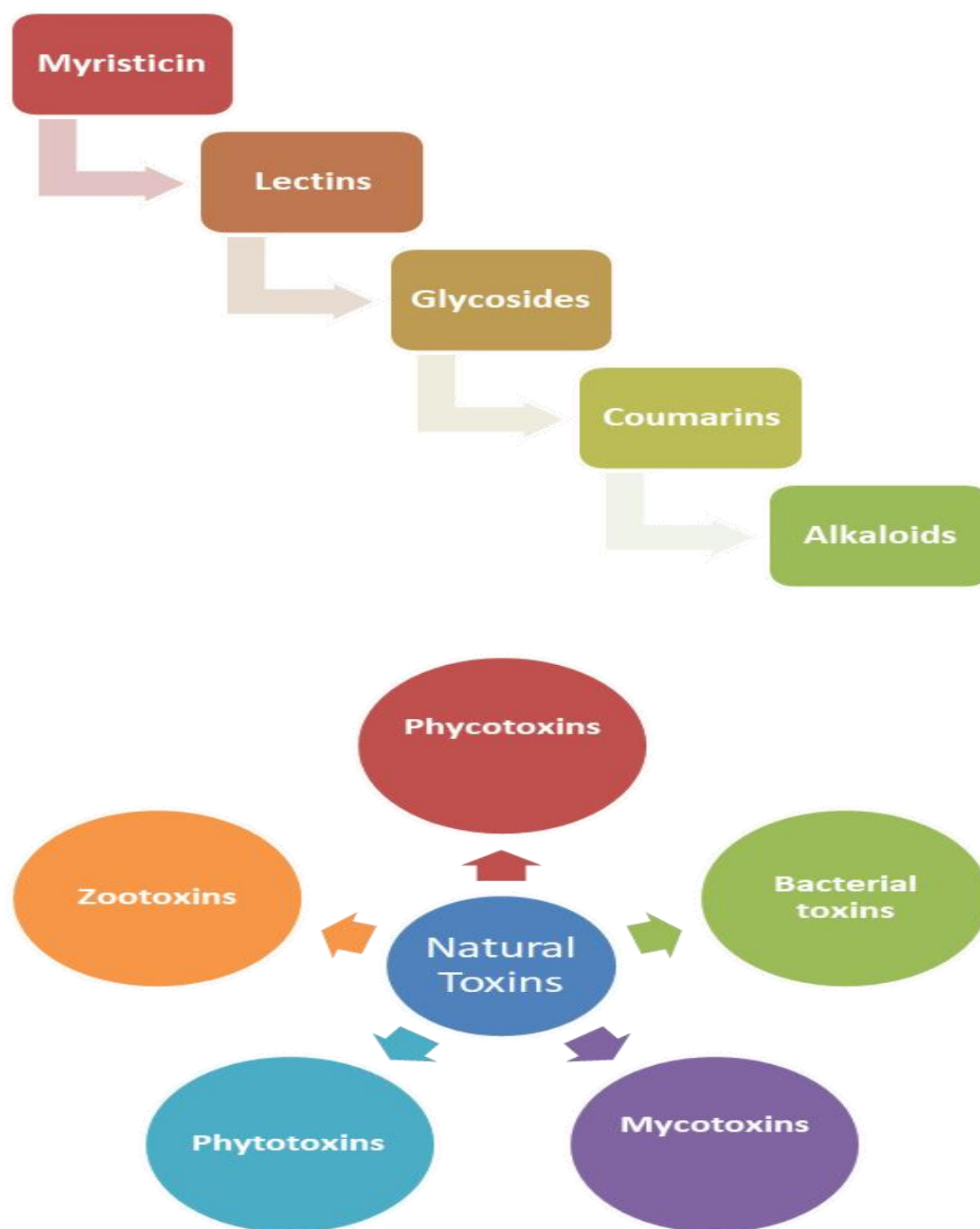


Fig.1: Diagram of Natural Toxins

Mycotoxins

Mycotoxins are secondary metabolites produced by fungi, which can cause diseases in humans and animals. Examples include aflatoxin and ergotamine. Mold can generate various types of mycotoxins, with multiple types of the same mycotoxin

potentially being produced. These are structurally diverse, low molecular weight complexes synthesized through the metabolic pathways of molds or fungi under suitable temperature and humidity conditions. Mycotoxins can contaminate a wide range of food products and pose significant risks to both animal and human health. Fungi demonstrate remarkable metabolic versatility, exhibiting the capacity to utilise diverse substrate profiles across extensive environmental gradients. These organisms synthesize mycotoxins exclusively under aerobic conditions.⁹ Approximately 25% of global crops are affected by fungal growth, leading to the production of mycotoxins, which have poisonous effects on both animal and human health, causing a condition known as mycotoxicosis. The intensity of toxicity varies depending on factors such as toxin concentration, animal maturity, exposure duration, and species-specific differences. Mycotoxins are commonly found in extremely dry, barren, temperate regions and moist areas. These toxins can contaminate human food at various stages in the food chain, and prolonged exposure has been linked to health ailments in both animals and humans.¹² Delayed storage of crops in scorching and moist conditions has been identified to promote the growth of aflatoxin-producing fungi and the accumulation of the toxin.¹³

Bacterial Toxins

Bacterial toxins represent a distinct group of biologically active compounds with dual roles. On one hand, they are capable of invading and damaging host tissues while disrupting immune homeostasis, thereby contributing to disease pathogenesis. Conversely, several bacterial toxins have been harnessed in medicine, where purified forms are applied therapeutically, including in oncology and other disease treatments. Toxicity can also arise following bacterial cell lysis, which results in the release of potent toxins into the host system. Among the most clinically significant are the toxins produced by *Staphylococcus aureus*, a major cause of food poisoning and systemic infections.¹⁴

Phycotoxins

Algal-derived toxins bioaccumulate through trophic chains, ultimately contaminate seafood products, particularly bivalve molluscs. Notable phycotoxins of significant public health concern include paralytic shellfish poisoning toxins, diarrhetic shellfish

poisoning toxins, and amnesic shellfish poisoning toxins. Endogenous phytotoxins represent naturally occurring plant constituents that exhibit toxicity and adversely affect nutrient bioavailability. Many botanical toxins contribute to plant defence mechanisms against fungal pathogens, bacterial infections, arthropod herbivory, and other environmental stressors.¹⁵ These phytotoxins occur naturally in commonly consumed plant foods, including vegetables and fruits, functioning as secondary metabolites that protect against diverse threats, including pathogenic microorganisms, insect predation, and mammalian herbivory. Plants frequently cause clinical complications due to their bioactive chemical constituents. Numerous phytochemicals and plant-derived secondary metabolites possess toxicological properties comparable to bacterial exotoxins and may induce adverse effects in humans. Plant toxins can enter the human body through inhalation, ingestion, or dermal contact. Toxicological activity depends primarily on specific plant constituents, including glycosides, alkaloids, tannins, terpenes, proteins, volatile oils, and steroids. These compounds exert biological effects through distinct mechanisms involving cellular transporters, receptors, enzymatic systems, and genetic material within specific tissues and organs. In certain plant species, toxic constituents are distributed systemically throughout the entire organism occurring.¹⁶

Classification of Phytotoxins

Alkaloids

Alkaloid-rich plants exhibit a wide spectrum of pharmacological properties, including antimicrobial, analgesic, anticancer, and antidepressant activities.¹⁷ These nitrogen-containing compounds are biosynthesized by plants as part of their adaptive strategies. However, several alkaloids, such as hepatotoxic pyrrolizidines, indolizidines, piperidines, and tropanes, have been documented to exert toxic effects in both humans and animals¹⁸. Toxic alkaloids interfere with normal physiological functions through interactions with specific receptors, transporters, enzymes, and genetic material within target cells and tissues.¹⁹ Acute exposure may lead to pruritus, nausea, vomiting, and mild gastrointestinal disturbances, whereas chronic exposure has been associated with severe outcomes, including psychosis, paralysis, teratogenicity, arrhythmias, and even sudden death.²⁰

- **Tropane Alkaloids:** Tropane alkaloids (TAs) represent ubiquitous alkaloid compounds distributed throughout plant tissues, with highest concentrations occurring in seeds and roots of the Erythroxylacea, Brassicaceae, and Solanaceae families.²¹ Historically, tropane alkaloid-containing plants have been employed by indigenous populations for traditional medicinal and ceremonial purposes due to their anticholinergic and psychoactive properties. Clinical manifestations of tropane alkaloid toxicity include constipation, photophobia, mydriasis, visual disturbances, and desiccation of upper gastrointestinal and respiratory tract mucosae. Mydriasis and neurobehavioural alterations constitute the predominant toxicological effects observed in experimental animal models.²²
- **Piperidine Alkaloids:** Piperidine alkaloids induce acute toxicity in adult livestock, manifesting as musculoskeletal abnormalities. Clinical signs of acute intoxication include polyuria, diarrhoea, muscular weakness, tachyarrhythmias, ataxia, progressive debilitation, collapse, and mortality secondary to respiratory failure. The teratogenic potential of piperidine alkaloids results in multiple congenital arthrogryposis syndromes and cleft palate in caprine, porcine, bovine, and ovine species.²³
- **Pyrrolizidine Alkaloids:** Pyrrolizidine alkaloids are predominantly found in plant families such as *Boraginaceae*, *Asteraceae*, and *Fabaceae*²⁴. Severe outbreaks of illness have been reported among populations consuming cereal grains contaminated with seeds from plants containing these alkaloids. In addition, pyrrolizidine alkaloids have been detected in secondary food sources, including milk from cows and goats, as well as in honey. Compounds with unsaturated necine ring structures are particularly hazardous, exhibiting teratogenic, mutagenic, carcinogenic, and hepatotoxic effects. The underlying mechanism of toxicity involves enzymatic conversion of pyrrolizidine alkaloids into highly reactive pyrroles, which act as alkylating agents. Once formed in the liver, these metabolites can enter the circulation and reach the lungs, where they contribute to pulmonary vascular remodeling and pulmonary hypertension.²⁵

Coumarins

Coumarin is found in plant teas prepared from melilot, tonka beans and woodruff the

savoring oil of spice cassia and the bergamot In the United States, the addition of coumarin to food was banned in 1954 due to reports of liver toxicity in rodents. Prolonged exposure to high doses of coumarin has been linked to hepatic tumors in mice and hepatic and pulmonary tumors in rats. Fungi present in blemished sweet clover and other hay products can metabolize coumarin into dicoumarol, a compound structurally similar to vitamin K. By reducing vitamin K levels, dicoumarol stimulates hemorrhage. Fodder concentrations of dicoumarol exceeding 10 parts per million have been responsible for cattle deaths due to bleeding. ..²⁶Coumarins and their derivatives exhibit a broad spectrum of biological activities that underlie their therapeutic potential across metabolic and non-metabolic disorders. This review considers their cellular, molecular, and biochemical mechanisms of action, surveys natural and synthetic sources and routes of production, and summarizes current knowledge of toxicity, pharmacokinetics, and bioavailability — with particular attention to scopoletin as an illustrative compound²⁷.

Myristicin

Myristicin, the primary component of *Myristica fragrans*, has been linked to several adverse effects, including dissociation, hypotension, confabulation, tachycardia, numbness, sedation, vomiting, flushing, gagging, painful intestinal obstruction, paresthesia, blurred vision, hypothermia, and excessive sweating²⁸. These toxicities can occur both accidentally and intentionally, often in pursuit of a psychoactive experience, as myristicin and potentially other compounds in nutmeg are known to induce hallucinations. Since the middle Ages, *Myristica* has been employed for various purposes, including as a stimulant, abortifacient, remedy for flatulence, narcotic, and emmenagogue.²⁸Myristicin, present in low concentrations in culinary herbs and spices, and does not pose a significant health risk through dietary exposure. At the same time, its bioactive properties highlight potential applications within traditional medicinal practices²⁹.

Glycosides

A wide range of toxic glycosides is found in higher plants, with the most prominent groups including glucosinolates (thioglycosides), cyanogenic glycosides, aliphatic nitrotoxins, and cardiac glycosides.³⁰ the concentration of these compounds within

plants can vary significantly under physiological stress, such as drought or nutrient deficiency. Cyanogenic glycosides release hydrogen cyanide, which exerts its toxic effects by binding to mitochondrial cytochrome oxidase and thereby halting electron transport. Severe cyanide intoxication can manifest with neurological and systemic symptoms, including muscle twitching, dizziness, rapid respiration, hypotension, tachycardia, headache, stupor, gastrointestinal pain, vomiting, diarrhea, cyanosis, confusion, convulsions, and in extreme cases, irreversible coma.³¹ Toxicity is often reported after ingestion of herbal teas or plant parts such as flowers, leaves, or seeds containing cardiac glycosides, with serious poisoning most frequently associated with intentional ingestion or inappropriate therapeutic use. *Nerium oleander*, though native to the Mediterranean, is cultivated globally as an ornamental plant.³² All parts of the plant are poisonous, predominantly due to its cardiac glycosides—oleandrin, nerin, olinerin, and digitoxigenin—of which oleandrin is the principal toxin. The bark also contains rosagenin, a compound capable of producing strychnine-like effects.³³

➤ Lectins

Lectins constitute a diverse group of glycoproteins present in high concentrations within leguminous plants, including lima beans, black beans, kidney beans, soybeans, and lentils, as well as cereal grains.³⁴ Various legume species contain lectins as natural toxins, with kidney beans exhibiting the highest concentrations, particularly red varieties. Consumption of uncooked beans induces severe gastric distress, diarrhoea, and emesis. Lectins demonstrate high affinity for mucosal epithelial cells, thereby disrupting intestinal nutrient absorption.³⁵ Since lectin-induced intestinal malabsorption depends upon the presence of indigenous microflora, and it has been hypothesised that lectins may exert toxicity through promotion of pathogenic bacteria proliferation within the gastrointestinal tract. Clinical manifestations of phytohaemagglutinin toxicity in humans, including emesis, nausea, and diarrhoea, typically develop within three hours of ingestion. Recovery generally occurs within four to five hours following symptom onset.

- ³⁶ Plant lectins are known to confer protection against various abiotic stressors,

including drought, salinity, and temperature extremes. While they play important physiological roles in plants, their ingestion can exert toxic effects in animals and humans. Owing to their functional attributes—such as antimicrobial, anticancer, immunomodulatory, anti-inflammatory, and antiviral activities—lectins have also emerged as compounds of considerable interest in medical research and therapeutic development.³⁷

Phytotoxins

Plant-derived toxins, known as phytochemicals, often serve as a defense mechanism against fungal and insect attacks³⁸. Examples include glycoalkaloids found in potatoes and toxic compounds present in herbs such as pyrrolizidine alkaloids and anisatin in certain star anise varieties. Algae, or phototrophic eukaryotic microorganisms, produce toxins known as phycotoxins. A significant portion of aquatic flora contains algae; including macroalgae (seaweeds) and microalgae.³⁹ Dinoflagellates, a group of aquatic algae, are notorious for producing potent, highly toxic compounds. The toxicity of food, particularly seafood like clams, mussels, fish, and oysters, has been reported globally. Many fish are naturally non-toxic but become toxic after consuming toxic algae. As a result, humans may experience toxicity through the consumption of fish that eat toxic algae or by consuming flesh-eating fish that have ingested these plant-eating fish.⁴⁰

Zootoxins

Animal-derived toxins, including those produced by scorpions, serpents, and certain amphibians, constitute zootoxins. These compounds typically pose minimal risk to human health via oral exposure; however, notable exceptions exist, such as bufotoxin secreted by *Bufo marinus*. Dermal contact with the dorsal surface of this toad induces hallucinogenic effects. Venomous or toxic animals possess specialised poison glands and delivery mechanisms that facilitate toxin inoculation through stings, bites, or contact that breach dermal barriers, representing significant global public health concerns.⁴¹ Toxicity of venoms from toads within the genus *Bufo* varies considerably among species, with some demonstrating greater potency than others; for example, *B. marinus* exhibits higher toxicity than *Bufo vulgaris*. The toxin profile comprises bufotoxins, bufadienolides, catecholamines, bufotenins, and non-cardiac steroidal compounds.⁴² Clinical manifestations range from mild oral mucosal irritation and

hypersalivation to moderate symptoms including weakness, ataxia, depression, cardiac arrhythmias, and involuntary urination and defecation

.⁴³. Aptamers development with nanoconjugation can link with zootoxin, providing directly to infected organ without damaging other sides⁴⁴.

Conclusion

Contemporary research on natural toxins encompasses dual therapeutic paradigms: the development of treatments for toxin-induced pathologies and the exploitation of these compounds as pharmaceutical agents for diverse medical conditions. Natural toxins represent a heterogeneous array of bioactive molecules, including marine biotoxins, cyanogenic glycosides, furocoumarins, lectins, mycotoxins, glycoalkaloids such as solanine and chaconine, toxic metabolites derived from poisonous fungi, and pyrrolizidine alkaloids. The advancement of nanotechnological applications, particularly aptamer-based therapeutic platforms, has substantially enhanced the efficacy of natural toxin-derived treatment modalities.

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