ISSN: 2322 - 0902 (P)
ISSN: 2322 - 0910 (O)

International Journal of Ayurveda and Pharma Research

Review Article

NATURAL FOOD TOXINS AND ITS AYURVEDIC PURVIEW

C. Naisha[1]*, M.C. Shobhana[2]

*[1]PG Scholar, [2]Professor & HOD, Dept. of Swasthavritta, VPSV Ayurveda College, Kottakkal, Kerala, India.

Article info

Article History:
Received: 18-12-2024
Accepted: 11-01-2025
Published: 07-02-2025

KEYWORDS:
Natural food toxin, Phytotoxin, Marine toxin, *Visha, Sthavara visha, Jangama visha.*

ABSTRACT

Toxins are poison of plant or animal origin, produced by or derived from microorganisms and acting as an antigen in the body. These are formed in dietary articles as a part of the defence mechanism of the plant against predators or insects or in response to extreme climatic conditions. These secondary metabolites are chemical compounds having diverse structure and differ in biological function and toxicity, will have adverse impact on human or animal health when consumed in excess. Natural food toxins can be categorized under phytotoxins and marine toxins. Cyanogenic glycosides, glycoalkaloids, lectins, glucosinolates and pyrrolizidine alkaloids are the major phytotoxins. Marine or aquatic toxicity occurs by the ingestion of shellfish that have consumed toxin producing algae, or by bacterial growth in fish due to improper handling. Some of these toxins are extremely potent and harmful when consumed in large quantities. These toxins possess a serious health threat to both humans and livestock. It may result in acute and chronic toxicity ranging from gastrointestinal upset to fatality. So, detoxification of dietary articles containing natural food toxins is crucial as it helps to eliminate these chemicals and thereby ensuring food safety. So, this article intends to highlight the Ayurvedic viewpoint of natural food toxins and the importance of precautionary measures for preventing toxicity of natural food toxin from daily consuming dietary article.

INTRODUCTION

Food borne illness refers to any illness caused by the contamination of food with harmful bacteria, viruses, parasites, or toxins. According to the WHO, approximately 1 in 10 people globally experience a foodborne illness annually, resulting in an estimated 600 million cases and 420,000 deaths each year.[1] So, toxins are a significant cause of foodborne illnesses. These poisonous substances, which can be of plant or animal origin, are produced by or derived from microorganisms and can act as antigens in the body. While toxins may have beneficial medicinal properties in small doses, they become harmful and potentially lethal in larger amounts.

Natural toxins are harmful secondary metabolites produced by living organisms. Secondary metabolites are organic compounds that do not play a direct role in the normal growth or development of the plant, but instead help in ecological interactions. While these substances typically do not harm the organisms that produce them, they can pose health risks to humans or animals when consumed in large quantities or inappropriately, especially when present in food. These substances serve as a defence mechanism for plants and generally do not harm the organisms that produce them. However, plant secondary metabolites can negatively affect the health of consumers, causing both acute and chronic toxicity. Acute toxicity may result in symptoms such as nausea, dizziness, stomach pain, vomiting, and skin reactions, while long-term exposure can lead to irreversible damage to vital organs like the immune system, kidneys, and reproductive system. In severe cases, they can be carcinogenic and even fatal.[2]

According to the literature, various traditional and emerging food-processing techniques such as including drying, boiling/cooking, fermentation, germination, microwave heating, have been identified as effective strategies for reducing toxicants. Therefore, this review article provides an overview of natural food toxicants from plant sources and explores how various traditional and innovative food

Access this article online

Quick Response Code

https://doi.org/10.47070/ijapr.v13i1.3514

processing techniques contribute to their detoxification.

MATERIALS AND METHODS

Published articles are used to acquire material on the topic

DISCUSSION

Natural toxins can be divided into phytotoxins and marine toxins. Commonly seen groups of phytotoxins are cyanogenic glycosides, glycoalkaloids, lectins, glucosinolates and pyrrolizidine alkaloids.

Phytotoxins

1. Cyanogenic Glycosides

These are amino acid-derived compounds produced as secondary metabolites in plants. Known as phytotoxins (toxins produced by plants), they are found in approximately 2,500 species, including those in families like Compositae and Fabaceae. Cyanogenic glycosides, while not toxic in their intact form, release hydrogen cyanide (HCN), which is responsible for their toxic effects in the body. The lethal dose of HCN for humans ranges from 0.5 to 3.5mg/kg. These compounds are particularly present in plants such as cassava (*Manihot esculenta* Crantz)- linamarin, 900–2000mg HCN/kg dry matter, flaxseed (*Linum usitatissimum*)- linustatin, 264–354mg/kg, sorghum (Sorghum bicolor L.) - dhurrin, 30% dry weight, apricot kernal (*Prunus armeniaca*)– amygdalin, 49–4000mg/ kg, apple seeds (*Malus domestica*)- amygdalin, 1–4mg/g, cocoyam (*Colocasia esculenta*)- linamarin, 21.0–171.3mg/kg dry matter, and bamboo shoots - (taxiphyllin, 1000–8000mg HCN/kg.[3]

Mechanism of Toxicity

Cyanide inhibits cytochrome oxidase, which prevents oxygen use and results in cytotoxic anoxia. This reduces the use of oxygen in the tissues.[4]

Cyanogenic Glycoside Detoxification Methods

1. **Peeling:** Peeling is the initial step in processing cassava roots. The peel of cassava contains a higher concentration of cyanide compared to the pulp. By removing the peel, up to 50% of the cyanogenic glycosides in the root can be reduced. For bitter cassava, the peel contains 650 ppm of total cyanide, while the pulp has 310 ppm. In contrast, sweet cassava varieties have a cyanide concentration of 200 ppm in the peel and 38 ppm in the pulp. In sweet varieties, the pulp can be safely consumed after boiling once the peel is removed, while bitter varieties require additional detoxification processes before consumption.[5]
2. **Grating:** Grating is a size reduction method that increases the surface area, facilitating more efficient contact between linamarin and linamarase, which aids in the detoxification process. The smaller particle size enables the release of intracellular linamarin, allowing it to react with external linamarase enzymes to produce volatile hydrogen cyanide (HCN). The cyanide content in grated cassava roots is influenced by the duration of contact between the glucoside and glucosidase in water. While grating alone is not enough for complete detoxification, it can be combined with other methods to enhance HCN evaporation or reduction, such as fermentation. For instance, fermented grated-mash cassava used in tofu production has been shown to reduce cyanogenic glycoside levels by 85.5% within 72 hours.[5,6]
3. **Drying:** Drying is a highly effective technique for reducing cyanogenic glycosides in plant foods. Various drying methods, such as sun drying, oven drying, freeze drying, and superheated steam, can be used to lower cyanogen levels in food products. Specifically, superheated steam drying at temperatures between 120–160°C significantly breaks down taxiphyllin, which causes bitterness in bamboo shoots and oven drying at 60°C after grinding for 8 hours leads to a 95% reduction in cyanogen content.[6,7]
4. **Boiling/Cooking:** Cooking and boiling are some of the most effective methods for reducing cyanogenic compounds in plant foods. These processes appear to promote the rupture of cell walls, facilitating the release of cell contents, including harmful chemicals and anti-nutrients. An early study on the impact of boiling on cassava's cyanogenic glycoside content found that boiling for 25 minutes reduced bound cyanogenic glycosides by 45 to 50%. Other studies also showed significant reductions in cyanogenic glycosides through boiling. For example, boiling Bambusa vulgaris shoots for 10 minutes led to a decrease of 67.84–76.92% in cyanogenic glycosides, and an additional 10 minutes of boiling further reduced the cyanogen concentration by up to 87%. Additionally, steaming was shown to significantly decrease the cyanide content in cassava flour (raw material) by 72.6%, with further reductions during saccharification and fermentation, ultimately lowering the cyanide content by 81.5%.[8]
5. **Soaking/Wetting:** Soaking or wetting, similar to other food processing methods, helps improve the shelf life, safety, and quality of products. A comparison of different soaking techniques showed that soaking peeled cassava roots was more effective at reducing cyanogen levels than soaking unpeeled roots. Retaining the peel during processing led to significant cyanogen retention in the pulp. In a similar process, cassava flour mixed with water and left in the shade for 5 hours at approximately 30°C allowed HCN gas to escape,

resulting in a reduction of total cyanide levels by a factor of three to six.[9]

6. **Fermentation:** Fermentation is an ancient food preservation technique that has become increasingly popular across cultures, valued for its nutritional benefits and unique sensory properties. It enhances the nutritional profile of food by producing essential vitamins, amino acids, and breaking down anti-nutrients. In one study, an optimized enzymatic fermentation of flaxseed for 48 hours using 12.5% glucosidase and 8.9% cyanide hydratase reduced the cyanide concentration in flaxseed powder by 99.3%. Similarly, the fermentation of cassava flour was found to eliminate 81.5% of the cyanide content in the sample.[8,10]
7. **Germination:** Germination is an ancient food processing method known to effectively reduce cyanogenic glycoside levels. The germination of flaxseed leads to a significant reduction in its cyanogenic glycoside content. [11]

2. Glycoalkaloids

Glycoalkaloids (GAs) are naturally occurring toxic compounds found in plants of the Solanaceae family, where they play a role in insect resistance. Potatoes (*Solanum tuberosum*) contain α-solanine and α-chaconine, tomatoes (*Solanum lycopersicum*) produce α-tomatine and dehrdrotomatine, and eggplants (*Solanum melongena*) contain solasonine and α-solamargine as glycoalkaloids. These plants, along with other members of the Solanaceae family, are known for producing significant amounts of glycoalkaloids. The toxin is primarily concentrated in the roots, leaves, flowers, and edible parts of the plants, including the skin and sprouts.[12]

Green tomatoes can contain as much as 500mg/kg of tomatine, but this level decreases to approximately 5mg/kg as the fruit ripens. In ripe red tomatoes, the tomatine content consumed by humans is typically between 10–30mg /kg, whereas in green tomatoes, it can range from 200 to 500mg/kg. In eggplants, the glycoalkaloid concentration varies between 0.625 and 20.5mg/kg across 21 different varieties. For potatoes, glycoalkaloid levels vary depending on the variety and growing conditions, with damaged plants showing higher concentrations. Normal potato tubers contain between 12–20mg/kg of glycoalkaloids, while green tubers can have 250–280mg /kg, and green skins may contain as much as 1500–2200mg/kg.[13]

In humans, the toxic dose of total glycoalkaloid is 2 to 5mg/kg body weight (BW), and the lethal dose is 3 to 6mg/kg BW. [14]

Mechanism of Toxicity

α-Chaconine is considered the most toxic of the potato alkaloids due to its overall toxicity. It inhibits acetylcholinesterase, leading to cell damage and organ dysfunction, and is also teratogenic, affecting embryonic development. In contrast, α-solanine is somewhat less harmful.[15] Their intake has been linked to diarrhoea, fever, vomiting, gastrointestinal discomfort, gastroenteritis, neurological disorders, a high pulse rate, low blood pressure, and mortality in humans and farm animals. [16]

Glycoalkaloids Detoxification Methods

Peeling the outer 3-4mm of potato tissue effectively removes almost all of the glycoalkaloids.[17] Boiling is said to reduce the levels of major glycoalkaloids by about 3.5%, while microwaving cuts them by approximately 15%. Temperatures exceeding 170°C lead to significant degradation of glycoalkaloids, while deep-frying at 150°C has minimal impact on their concentrations. Heating potatoes at 210°C for 10 minutes reduces the levels of α-chaconine and α-solanine by roughly 40%.[16]

3. Lectin

Lectins, also referred to as phytohaemagglutinins due to their ability to agglutinate red blood cells, are protein-based toxic compounds found in legumes. These toxic proteins are present in many plants, especially in seeds such as cereals, beans, wheat, peas, kidney beans, lentils, soybeans, bananas, and mushrooms. The lethal dose of lectins is approximately 50mg/kg.[19]

Mechanism of toxicity

Lectins bind to the surface of intestinal epithelial cells, disrupting the normal functioning of the digestive system and reducing protein digestibility in vitro. They damage the lining of the small intestine and can spread throughout the body, leading to various health issues like diarrhoea, nausea, and vomiting.[20]

A high lectin concentration in food causes nutritional deficits, gastrointestinal distress, immunological allergic reactions, and food poisoning.[21]

Lectin Detoxification Methods

Heating plant-based foods during cooking can significantly reduce their lectin levels[22] Fermenting lentils (Lens culinaris) for 72 hours at an optimal temperature has been shown to completely eliminate the lectin content in the seeds. Soaking the seeds in distilled water also leads to a substantial reduction in their lectin levels, with a decrease ranging from 0.11% to 5.18%.[23]

4. Glucosinolates

Glucosinolates (GSLs) are a class of chemicals found in plants such as broccoli, cauliflower, and cabbage that belong to the goitrogen family. Elevated

levels of GSLs have been associated with several negative effects, such as thyroid enlargement, reduced plasma thyroid hormone levels, organ dysfunction (particularly in the liver and kidneys), stunted growth, impaired reproductive health, and in severe cases, mortality.[24]

Mechanism of toxicity

The enzyme myrosinase breaks down glucosinolates into various derivatives, including thiocyanates, isothiocyanates, and epithionitriles, during chewing. These compounds interfere with the thyroid's ability to absorb iodide, leading to iodine deficiency and, consequently, the inhibition of T4 production.[25] GSLs and similar chemicals have long been associated with adverse effects on the human body, with their consumption linked to changes in thyroid function and a higher risk of various thyroid disorders.[26]

Glucosinolates Detoxification Methods

Cooking is considered an effective method for reducing glucosinolate levels in food.[27] Traditional boiling of cruciferous vegetables leads to significant losses of glucosinolates (up to 90%), as the toxins leach into the cooking water.[28] Glucosinolate reductions vary with different cooking methods: steaming results in a decrease of 18 to 22%, blanching reduces them by 30-52%, and boiling lowers them by 46-61%.[29] Likewise, boiling and high-pressure cooking have been shown to reduce total glucosinolate content by 64%. In broccoli, steaming for 5 and 10 minutes reduced glucosinolate levels by 57.5% and 72.3%, respectively. Blanching for 5 and 10 minutes, in contrast, decreased glucosinolate content by 62.0% and 67.7%, respectively.[30]

5. Pyrrolizidine Alkaloids (PA)

Pyrrolizidine alkaloids (PAs) are a group of heterocyclic compounds produced by plants, believed to serve as a defence mechanism against herbivores.[31] These alkaloids are primarily found in plants from the Asteraceae (also known as Compositae) family, including genera such as Senecio, Eupatoria, and Tussilago; the Boraginaceae family, with genera like Heliotropium, Symphytum, and Trichodesma; and the Fabaceae (also called Leguminosae) family.[32]

Mechanism of toxicity

Pyrrolizidine alkaloids (PAs) are quickly absorbed through the gastrointestinal tract and metabolized in the liver, where they are converted into highly reactive pyrroles. These compounds can cause cytotoxic damage to liver cells, potentially leading to liver failure and death. The symptoms associated with PA toxicity include vomiting, bloody diarrhoea, and liver enlargement.[33]

Pyrrolizidine alkaloids Detoxification Methods

Methods Soaking and boiling with peeling are indicated as effective methods for minimizing the PA content of food.[34]

6. Marine Toxins

Marine biotoxins are toxic chemicals naturally produced by specific algae species and other microorganisms, such as bacteria. These toxins can enter the food chain primarily through the consumption of fish and other seafood, including mollusks and crustaceans. Their occurrence in marine and freshwater environments is heavily influenced by factors like climate and temperature.[35]

These compounds have varied chemical structures and are typically secondary metabolites known for their high toxicity to a broad spectrum of cells and organisms. They can accumulate in large quantities in filter-feeding primary consumers like bivalve mollusks and may bioaccumulate up the food chain. This accumulation can lead to significant mortality events in fish, seabirds, marine mammals, and even human poisoning through the consumption of contaminated seafood.

High levels of toxins in seafood, particularly in shellfish and finfish, can lead to various forms of human poisoning. Many of the algal toxins linked to seafood poisoning are heat-resistant and remain active even after cooking. Furthermore, it is not possible to visually differentiate between toxic and non-toxic fish and shellfish.[36]

On the basis of their poisoning symptoms, they are also classified as toxins causing paralytic shellfish poisoning (PSP), amnesic shellfish poisoning (ASP), diarrheic shellfish poisoning (DSP), neurotoxic shellfish poisoning (NSP), and ciguatera fish poisoning (CFP).[37]

7. Paralytic Shellfish Poisoning (PSP)

Paralytic shellfish poisoning occurs when individuals consume bivalve mollusks (such as mussels, clams, oysters, and scallops) that have ingested toxic dinoflagellates. The toxins are absorbed and temporarily stored within the shellfish.[38]

Mechanism of toxicity

Shellfish accumulate toxins, such as saxitoxins, after ingesting toxin-producing algae. These saxitoxins are neurotoxins that work by blocking sodium (Na+) ion flow through nerve sodium channels, disrupting signal transmission. The lethal dose for humans ranges from 1 to 4mg, expressed as saxitoxin equivalents, while the FDA's action limit is set at 80 micrograms (µg) of toxin per 100 grams of shellfish tissue.[39]

Neurological symptoms typically emerge within an hour of consuming toxic shellfish, and in non-lethal cases, they generally resolve within a few days. These symptoms include tingling, numbness, and

a burning sensation in the lips and fingertips, along with ataxia, dizziness, staggering, drowsiness, dry throat and skin, confusion, aphasia, rash, and fever. In severe cases, respiratory paralysis may occur, potentially leading to death within the first 24 hours. However, those who survive this critical period usually have a favourable prognosis. There is no known antidote, but respiratory support is provided if paralysis develops. No lasting effects are reported, and patients typically make a full recovery.[40]

8. Amnesic Shellfish Poisoning (ASP)

Amnesic shellfish poisoning has been proposed by Todd (1989) as a name for the syndrome caused by domoic acid produced by marine diatoms.[41]

Mechanism of toxicity

DA interacts with glutamate receptors in the central nervous system, leading to excessive activation of these receptors, which results in the generation of reactive oxygen species and, in some cases, cell death. The symptoms can vary, including gastrointestinal issues such as nausea, vomiting, diarrhoea, or abdominal pain, as well as neurological symptoms like confusion, lethargy, disorientation, numbness, and short-term memory loss. In severe instances, this may progress to coma or even be fatal.[42]

Diarrheic Shellfish Poisoning (DSP)

Diarrhetic shellfish poisoning is caused by ingestion of mussels, scallops, or clams that have been feeding on Dinophysis fortii or D. acuminata and other species of Dinophysis and possibly Prorocentrum.[43]

These dinoflagellates are found globally, meaning the illness can potentially occur in various regions around the world. Several toxins have been identified, including okadaic acid (OA) and related compounds.

Symptoms typically appear within 30 minutes to a few hours after consuming shellfish that have ingested toxic algae. Gastrointestinal issues such as diarrhoea, vomiting, and abdominal pain are common, and recovery usually occurs within 3-4 days, with or without treatment. There have been no reported fatalities associated with the condition.[44]

9. Neurotoxic Shellfish Poisoning (NSP)

Neurotoxic shellfish poisoning (also known as brevetoxic shellfish poisoning or BSP) occurs when shellfish consume the red tide organism *Gymnodinium breve*. [45]

The blooms of this dinoflagellate are often visible as a red discoloration in the water, and the organisms can be identified under a microscope. Red tides typically result in large fish die-offs, with the carcasses washing up on the shore.

Neurotoxic shellfish poisoning presents with both neurological and gastrointestinal symptoms, including nausea, vomiting, diarrhoea, paresthesia, cramps, bronchoconstriction, paralysis, seizures, and coma. In severe cases, it can be fatal.[46]

10. Ciguatera Fish Poisoning (CFP)

This condition results from consuming fish that have become toxic after feeding on harmful dinoflagellates or toxic herbivorous fish. The main source of toxicity is the benthic dinoflagellate *Gambierdiscus toxicus*, which is typically found in tropical regions, where it associates with macroalgae and is often attached to dead corals.[47]

Symptoms primarily involve gastrointestinal distress, including nausea, vomiting, abdominal pain, and diarrhoea. However, cardiovascular issues such as bradycardia and hypertension, along with neurological symptoms like paresthesias, dysesthesias, and hyperesthesias, can develop within hours to two weeks following exposure.[48]

Ayurvedic Purview

In Ayurveda the term *Visha* encompasses various forms of toxins, including *Sthavara Visha, Jangama Visha* and *Kritrima visha* and in general which will disrupts the digestive fire, aggravate *Tridoshas* and deplete the *Ojas.*[49]

देहं प्रविश्य यद् द्रव्यं दूषयित्वा रसादिकान् । स्वास्थ्यप्राणहरं च स्यात् तद् द्रव्यं विषमुच्यते ॥

After entering into the body, *Visha* immediately causes vitiation of *Rasadi dhatus* and hampers both health as well as life. Even though there is no explicit mentioning of natural food toxins such as Ayurveda, *Sthavara* and *Jangama visha*, can be aligned with the concept of phytotoxins and aquatic biotoxins. In this article the concept of *Visha* and its mode of action has been highlighted with its similarity with that of the natural food toxins.

According to Ayurveda, *Hithahara* contributes to the healthy state of body and *Ahithahara* to unhealthy state. Dietary articles having natural toxins belongs to *Ahithahara* and results in diseases. By taking sufficient precautionary measures it can be turned into *Hithahara.*

Sthavara visha adhishtanas are mentioned in classics such as *Mula, Patra, Phala, Pushpa, Twak, Ksheera, Sara, Niryasa, Dhatu* and *Kanda.*[50] All the items mentioned in the *Sthavara visha adhishtanas* are inherently toxic and after detoxifying procedures it can be used for medicinal purposes. In case of phytotoxins of edible food articles, *Mula visha* can be attributed to linamarin in cassava and cocoyam, *Kanda visha* to α-solanine and α-chaconine in potato, *Patra visha* to glucosinolates in cabbage, *Phala visha* to α-tomatine and dehrdrotomatine in tomato, solasonine, and α-solamargine in eggplant and *Pushpa visha* to glucosinolates in cauliflower and broccoli. After proper processing, these can be consumed as *Ahara dravya*.

Concept of *Jangama visha* is entirely different from that of the aquatic biotoxins. Toxins are inherently present in 16 *Jangama visha adhishtanas,* but in case of aquatic biotoxins when shellfish ingests the toxin producing algae, then only it becomes toxic.

Natural food toxins consumed in excess quantity may result in death indicating similar properties with that of *Visha*. Lot of cattle death are reported by consuming unprocessed cassava along with peel as it contains the phytotoxin cyanide as natural food toxin.[51]

The concept of *Gara visha* and *Dushi visha* is entirely different from that of the natural food toxins. But there is similarity of action of natural food toxins with that of the *Gara visha* and *Dushi visha*., in *Gara visha, 'Vishanam cha alpaviryanam'* is mentioned**,** so due to *Alpa viryatva* it is resulting in diseases after long period of consumption. In *Dushi visha 'viryaalpabhavath'* is mentioned, due to mild potency it will remain in the body for very long years and during favourable circumstances result in diseases in the same way as chronic Natural toxins are acting.

CONCLUSION

Natural food toxins are the naturally derived contents in the edible food items. Phytotoxins, occurring in plants, possess a significant concern for human and animal health when consumed in excess. Though plants have evolved these toxins as defence mechanisms, they can be detrimental to humans causing a range of health issues from mild gastrointestinal symptoms to chronic diseases like cancer and even death. Toxicity due to aquatic biotoxins are acquired toxicity by ingestion of toxin producing algae by the shellfish. Mild gastrointestinal symptoms to diverse health impacts such as drop in blood pressure and cyanosis are the drastic outcome of this toxin which may ultimately lead to death. Different traditional and emerging food processing techniques are proposed by researchers that could significantly reduce most of the phytotoxins in food to the safest level.

Ayurvedic concept of *Sthavara* and *Jangama visha* go in parlance with the concept of natural toxins in food, emphasizing the importance of proper food selection, preparation, and processing for preventing the forthcoming impact. *Sthavara* or plant-based toxin and *Jangama* or animal-based toxin as present as natural food toxins can have debilitating effects on human health if consumed in excess or improperly.

REFERENCES

1. Lee H, Yoon Y. Etiological agents implicated in foodborne illness worldwide. Food science of animal resources. 2021 Jan; 41(1): 1.
2. Sachchan TK, Sharma A, Bangar A, Titoria M. Natural toxicants as potential health hazards: an overview.
3. Urugo MM, Tringo TT. Naturally occurring plant food toxicants and the role of food processing methods in their detoxification. International Journal of Food Science. 2023; 2023(1): 9947841
4. Chongtham N, Bisht MS, Premlata T, Bajwa HK, Sharma V, Santosh O. Quality improvement of bamboo shoots by removal of antinutrients using different processing techniques: A review. Journal of Food Science and Technology. 2022 Jan 1: 1-1.
5. Panghal A, Munezero C, Sharma P, Chhikara N. Cassava toxicity, detoxification and its food applications: a review. Toxin Reviews. 2021 Jan 2.
6. Montagnac JA, Davis CR, Tanumihardjo SA. Nutritional value of cassava for use as a staple food and recent advances for improvement. Comprehensive reviews in food science and food safety. 2009 Jul; 8(3): 181-94.
7. Qin Y, Duan B, Shin JA, So HJ, Hong ES, Jeong HG, Lee JH, Lee KT. Effect of fermentation on cyanide and ethyl carbamate contents in cassava flour and evaluation of their mass balance during lab-scale continuous distillation. Foods. 2021 May 14; 10(5): 1089.
8. Qin Y, Duan B, Shin JA, So HJ, Hong ES, Jeong HG, Lee JH, Lee KT. Effect of fermentation on cyanide and ethyl carbamate contents in cassava flour and evaluation of their mass balance during lab-scale continuous distillation. Foods. 2021 May 14; 10(5): 1089.
9. Chiwona-Karltun L, Afoakwa EO, Nyirenda D, Mwansa CN, Kongor JE, Brimer L. Varietal diversity and processing effects on the biochemical composition, cyanogenic glucoside potential (HCNp) and appearance of cassava flours from South-Eastern African region
10. Wu CF, Xu XM, Huang SH, Deng MC, Feng AJ, Peng J, Yuan JP, Wang JH. An efficient fermentation method for the degradation of cyanogenic glycosides in flaxseed. Food Additives & Contaminants: Part A. 2012 Jul 1; 29(7): 1085-91.
11. Li X, Li J, Dong S, Li Y, Wei L, Zhao C, Li J, Liu X, Wang Y. Effects of germination on tocopherol, secoisolarlciresinol diglucoside, cyanogenic glycosides and antioxidant activities in flaxseed (Linum usitatissimum L.). International Journal of Food Science & Technology. 2019 Jul; 54(7): 2346-54.
12. Schilter B, Constable A, Perrin I. Naturally occurring toxicants of plant origin. In Food safety management 2014 Jan 1 (pp. 45-57). Academic Press.
13. Nepal B, J. Stine K. Glycoalkaloids: Structure, properties, and interactions with model membrane systems. Processes. 2019 Aug 5; 7(8): 513

14. Morris SC, Lee TH. The toxicity and teratogenicity of Solanaceae glycoalkaloids, particularly those of the potato (Solanum tuberosum): A Review.
15. Bejarano L, Mignolet E, Devaux A, Espinola N, Carrasco E, Larondelle Y. Glycoalkaloids in potato tubers: the effect of variety and drought stress on the α-solanine and α-chaconine contents of potatoes. Journal of the Science of Food and Agriculture. 2000 Nov; 80(14): 2096-100.
16. Langkilde S, Mandimika T, Schrøder M, Meyer O, Slob W, Peijnenburg A, Poulsen M. A 28-day repeat dose toxicity study of steroidal glycoalkaloids, α-solanine and α-chaconine in the Syrian Golden hamster. Food and Chemical Toxicology. 2009 Jun 1; 47(6): 1099-108.
17. Friedman M. Potato glycoalkaloids and metabolites: roles in the plant and in the diet. Journal of agricultural and food chemistry. 2006 Nov 15; 54(23): 8655-81.
18. Barceloux DG. Potatoes, tomatoes, and solanine toxicity (Solanum tuberosum L., Solanum lycopersicum L.). Disease-a-month. 2009 Jun 1; 55(6): 391-402.
19. De Mejía EG, Prisecaru VI. Lectins as bioactive plant proteins: a potential in cancer treatment. Critical reviews in food science and nutrition. 2005 Sep 1; 45(6): 425-45.
20. Kumar Y, Basu S, Goswami D, Devi M, Shivhare US, Vishwakarma RK. Anti-nutritional compounds in pulses: Implications and alleviation methods. Legume Science. 2022 Jun;4(2):e111.
21. Xu Y, Shrestha N, Préat V, Beloqui A. Overcoming the intestinal barrier: a look into targeting approaches for improved oral drug delivery systems. Journal of controlled release. 2020 Jun 10; 322: 486-508.
22. Popova A, Mihaylova D. Antinutrients in plant-based foods: A review. The Open Biotechnology Journal. 2019 Jul 29; 13(1).
23. Shi L, Arntfield SD, Nickerson M. Changes in levels of phytic acid, lectins and oxalates during soaking and cooking of Canadian pulses. Food Research International. 2018 May 1; 107: 660-8.
24. Baenas N, Marhuenda J, García-Viguera C, Zafrilla P, Moreno DA. Influence of cooking methods on glucosinolates and isothiocyanates content in novel cruciferous foods. Foods. 2019 Jul 12; 8(7): 257.
25. Sikorska-Zimny K, Beneduce L. The metabolism of glucosinolates by gut microbiota. Nutrients. 2021 Aug 10; 13(8): 2750.
26. Miękus N, Marszałek K, Podlacha M, Iqbal A, Puchalski C, Świergiel AH. Health benefits of plant-derived sulfur compounds, glucosinolates, and organosulfur compounds. Molecules. 2020 Aug 21; 25(17): 3804.
27. Jones RB, Frisina CL, Winkler S, Imsic M, Tomkins RB. Cooking method significantly effects glucosinolate content and sulforaphane production in broccoli florets. Food chemistry. 2010 Nov 15; 123(2): 237-42.
28. Song L, Thornalley PJ. Effect of storage, processing and cooking on glucosinolate content of Brassica vegetables. Food and chemical toxicology. 2007 Feb 1; 45(2): 216-24.
29. Francisco M, Velasco P, Moreno DA, García-Viguera C, Cartea ME. Cooking methods of Brassica rapa affect the preservation of glucosinolates, phenolics and vitamin C. Food Research International. 2010 Jun 1; 43(5): 1455-63.
30. Hwang ES, Kim GH. Effects of various heating methods on glucosinolate, carotenoid and tocopherol concentrations in broccoli. International Journal of Food Sciences and Nutrition. 2013 Feb 1; 64(1): 103-11.
31. Giera DS, Preisitsch M, Brevard H, Nemetz J. Quantitative removal of pyrrolizidine alkaloids from essential oils by the hydrodistillation step in their manufacturing process. Planta Medica. 2022 Jun; 88(07): 538-47.
32. Ma C, Liu Y, Zhu L, Ji H, Song X, Guo H, Yi T. Determination and regulation of hepatotoxic pyrrolizidine alkaloids in food: A critical review of recent research. Food and Chemical Toxicology. 2018 Sep 1; 119: 50-60.
33. Chen T, Mei N, Fu PP. Genotoxicity of pyrrolizidine alkaloids. Journal of Applied Toxicology: An International Journal. 2010 Apr; 30(3): 183-96.
34. Takenaka M, Miyake N, Kimura T, Todoriki S, Urushiyama T. Reduction of pyrrolizidine alkaloids by cooking pre-treatment for the petioles and the young spikes of Petasites japonicus. Food Science and Technology Research. 2022; 28(3): 245-55.
35. Mafra Jr LL, de Souza DA, Menezes M, Schramm MA, Hoff R. Marine biotoxins: Latest advances and challenges toward seafood safety, using Brazil as a case study. Current Opinion in Food Science. 2023 Oct 1; 53: 101078.
36. Mafra Jr LL, de Souza DA, Menezes M, Schramm MA, Hoff R. Marine biotoxins: Latest advances and challenges toward seafood safety, using Brazil as a case study. Current Opinion in Food Science. 2023 Oct 1; 53: 101078.
37. Poletti R, Milandri A, Pompei M. Algal biotoxins of marine origin: new indications from the European Union. Veterinary research communications. 2003 Jan; 27: 173-82.
38. Halstead BW, Schantz EJ, World Health Organization. Paralytic shellfish poisoning. World Health Organization; 1984.

39. Schantz EJ. Chemistry and biology of saxitoxin and related toxins. Annals of the New York academy of sciences. 1986 Dec; 479(1): 15-23.
40. Joint FA, Intergovernmental Oceanographic Commission, World Health Organization. Report of the Joint FAO/IOC/WHO ad hoc Expert Consultation on Biotoxins in Bivalve Molluscs: short summary.
41. Teitelbaum JS, Zatorre RJ, Carpenter S, Gendron D, Evans AC, Gjedde A, Cashman NR. Neurologic sequelae of domoic acid intoxication due to the ingestion of contaminated mussels. New England Journal of Medicine. 1990 Jun 21; 322(25): 1781-7.
42. Schwarz M, Jandová K, Struk I, Maresova D, Pokorný J, Riljak V. Low dose domoic acid influences spontaneous behavior in adult rats. Physiological Research. 2014 May 1; 63(3): 369.
43. Ahmed FE. Naturally occurring fish and shellfish poisons. In Seafood safety 1991. National Academies Press (US).
44. Trainer VL, Moore L, Bill BD, Adams NG, Harrington N, Borchert J, Da Silva DA, Eberhart BT. Diarrhetic shellfish toxins and other lipophilic toxins of human health concern in Washington State. Marine Drugs. 2013 May 28; 11(6): 1815-35.
45. Baden DG, Mende TJ, Poli MA, Block RE. Toxins from Florida's red tide dinoflagellate Ptychodiscus brevis.
46. Watkins SM, Reich A, Fleming LE, Hammond R. Neurotoxic shellfish poisoning. Marine drugs. 2008 Jul 12; 6(3): 431-55.
47. Bagnis R. Ciguatera fish poisoning. Algal toxins in seafood and drinking water. 1993 Jan 1; 1.
48. Silva M, Rodriguez I, Barreiro A, Kaufmann M, Neto AI, Hassouani M, Sabour B, Alfonso A, Botana LM, Vasconcelos V. First report of ciguatoxins in two starfish species: Ophidiaster ophidianus and Marthasterias glacialis. Toxins. 2015 Sep 21; 7(9): 3740-57.
49. Srikantha Murthy K.R.Ashtang Hrudayam, uttarsthana, Varanasi, choukhamba Krishnadas Academy, 2021, P.N.329
50. Shastri Ambikadutta, editor, Sushruta Samhita, Varanasi; Chaukhambha Sanskrit Sansthana, Kalpasthana- 2/4, reprint 2014. pp 21
51. Obua BE, Ukweni IA, Uchegbu MC. Deaths among pigs fed sole fresh cassava by-products in humid southeastern Nigeria. Animal Production Research Advances. 2008; 4(2): 152-6.

Cite this article as:
C. Naisha, M.C. Shobhana. Natural Food Toxins and its Ayurvedic Purview. International Journal of Ayurveda and Pharma Research. 2025;13(1):127-134.
https://doi.org/10.47070/ijapr.v13i1.3514
Source of support: Nil, Conflict of interest: None Declared

***Address for correspondence**
Dr. C. Naisha
PG Scholar,
Dept. of Swasthavritta,
VPSV Ayurveda College,
Kottakkal, Kerala, India.
Email: mvineetha001@gmail.com