

Solanine Poisoning: Effects, Risks, and Management Strategies

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Plants produce toxic substances to protect themselves against external factors such as fungi and insects. Alkaloids naturally produced by plants can serve as examples of these toxic substances. Errors in the harvesting and storage of plants can lead to an increase in these toxic substances, posing a danger to human health. Alkaloids are chemical compounds typically containing one or more nitrogen atoms in a heterocyclic ring system.^[1] One of these chemical compounds is solanine, a glycoalkaloid poison found in plants of the *Solanum* genus, such as potatoes, tomatoes, and eggplants.^[2]

Solanine can naturally form in any part of the plant. In potato plants, it tends to concentrate in the leaves, stems, sprouts, and fruits. In normal potatoes, the concentration of solanine is typically at levels that do not pose a threat to human health. However, exposure to light and physical damage can lead to an increase in solanine concentration in potatoes.^[3] Although methods such as frying, boiling, and cooking can reduce the solanine content in potatoes to some extent, they often may not be sufficient to prevent poisoning.^[4]

Some countries have established specific limits for solanine content to prevent potential poisonings.^[1]

ABSTRACT

The plants naturally produce alkaloids as a defense mechanism against certain insects and fungi to protect themselves from external factors. Plants of the *Solanum* genus, such as potatoes, tomatoes, and eggplants, produce the substance solanine through natural processes. The amount of solanine produced by the plant for self-protection can increase due to genetic and environmental factors. This situation is often caused by improper storage conditions after harvest. Consuming potatoes with increased solanine concentration can be highly risky for health and can lead to solanine poisoning. This review discusses solanine poisoning and its mechanism.

Keywords: Alkaloids, potatoes, solanine, solanine poisoning, solanine toxicity.

Since people and chefs generally avoid using sprouted potatoes, solanine poisonings are not common, and spontaneous recovery is often observed in cases of poisoning. However, if large amounts of solanine are ingested, the situation can become serious and may result in death. Common symptoms include nausea, diarrhea, vomiting, throat-burning sensation, headache, and dizziness. In more severe cases, hallucinations, sensory loss, paralysis, fever, jaundice, and dilated pupils may occur. The mechanism of action of solanine is suggested to involve interaction with mitochondrial membranes.^[3]

ALKALOIDS

Alkaloids are complex chemical substances, mostly produced by plants and sometimes by animals. The basic building blocks of alkaloids are amino acids. They typically function as amines containing one or more nitrogen atoms, often found in a heterocyclic ring system.^[1]

When chemically named, alkaloids are generally referred to by adding the suffix '-ine' to the word. Alkaloids are found in almost all parts of the plant

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kingdom. They are observed in some fungi and most commonly in dicotyledons (dicotyledonous plants). The families Papaveraceae, Solanaceae, Fabaceae, Apocynaceae, Solanaceae, and Rubiaceae serve as examples of plant families containing highly rich alkaloid plants. Alkaloids accumulate in specific organs of plants; for instance, in some species, they accumulate in leaves, while in others, they accumulate in fruits and seeds, or in rhizomes and roots. Additionally, while synthesized in one tissue of the plant, they tend to accumulate in other tissues. Alkaloid-containing plants are more commonly observed in warm regions. Most alkaloids consumed by animals or humans exert their effects through the central nervous system and the autonomic nervous system. Alkaloids also possess potent pharmacological effects and can be used in epilepsy treatment due to their narcotic properties.^[5,6]

Some animals and humans utilize alkaloids that pose risks for protection from other animals. Certain insects exhibit immunity to toxic effects and perform their own defense by employing alkaloids as a poison against predatory animals.^[1] For plants, alkaloids are secondary metabolites. Secondary metabolites do not directly contribute to the plant's growth, development, or reproduction. The absence of secondary metabolites does not directly result in death.^[7]

The functions of alkaloids in most plants remain not fully understood. However, it is known that they participate in nitrogen metabolism as a nitrogen source and are generally secreted by plants after nitrogen uptake.^[5,8] The most significant known function of secondary metabolites is their role as a defense mechanism for plants when they encounter any stress factor.^[9]

Alkaloids can be found in foods for various reasons. They naturally occur in some important food plants commonly consumed by humans, such as potatoes. In certain foods, although alkaloids are not naturally present, certain fungi growing on food crops produce toxic alkaloids during plant growth or storage.^[1]

Solanine

Solanine is a type of steroid alkaloid known as a glycoalkaloid produced by plants belonging to the Solanaceae family as a defense mechanism. Among the main plants that produce solanine are potatoes, tomatoes, and eggplants.^[10,11]

Solanine is a toxic compound with fungicidal and insecticidal properties. It can naturally occur in any

part of the plant, including the leaves, fruits, and tubers.^[12]

The plant's different parts contain unequal amounts of the compound. For instance, in the henbane plant, all parts are toxic, but the seeds contain a higher amount of poison. In potatoes, the above-ground fruits and green tubers are toxic.^[10] This serves as a natural defense mechanism against consumption by insects, and even small quantities can be highly poisonous.^[13]

When the human body starts to metabolize solanine, sugar is separated, and solanidine is released. Although solanidines ingested through Solanaceae may not be immediately toxic, they can be stored in our bodies and may cause harm during periods of stress. Solanine is particularly present in potatoes, with its counterpart in tomatoes being tomatidine. Both steroid alkaloids are produced similarly to chlorophyll, indicating that they will be more abundant in the green parts of the plant.^[10]

SOLANINE POISONING

While solanine poisoning is not commonly observed today, recorded cases of poisoning exist. Normally, potatoes contain non-toxic levels of solanine in their tubers. However, in green or stressed potatoes, the concentration of solanine can be high, leading to toxicity.^[14] Potatoes showing signs of greening, sprouting, rotting, or physical damage should not be consumed as they may contain high levels of solanine. The presence of greenish discoloration underneath the potato skin indicates the concentration of solanine. In such cases, the potato should not be eaten as high levels can also be present in the rest of the potato.^[15]

The primary glycoalkaloids in edible Solanum species are alpha (α)-chaconine and α -solanine.^[1] Alpha-chaconine and α -solanine represent approximately 95% of the glycoalkaloid content in potato tubers. The concentration of glycoalkaloids can vary depending on environmental and genetic factors. The amount of solanine is minimal in ripe fruits. Factors that increase solanine concentrations include physical injury to the plant, certain potato varieties, physiological stress, immaturity, low storage temperature, and exposure to light. The greening of potatoes after harvest is evidence of improper storage under suitable conditions.^[16,17]

Commercially available potatoes have been examined for solanine content and found to contain

less than 0.2 mg/g. Most commercial potatoes have a solanine content of less than 0.2 mg/g. In some countries, the glycoalkaloid limit in potatoes is set at 0.2 mg/g.^[6,13]

When exposed to light and beginning to green, potatoes can contain 1 mg/g or more. Potatoes containing solanine at these levels can be dangerous.^[6]

The glycoalkaloid content is higher in the skin compared to the inner part of the potato. Peeling the potato by 3-4 mm can remove toxic solanine content.^[1] Peeled potatoes contain 30-80% less solanine compared to unpeeled potatoes.^[6,18] Therefore, peeling potatoes before cooking reduces solanine intake. Tasting a small piece of the peeled skin helps determine the solanine content in the potato. A bitter taste indicates high glycoalkaloid content. If there is a sudden burning sensation in the mouth, it can be said that the potato contains more than 0.2 mg/g of solanine.^[11] It is said that doses of 200-400 mg can cause toxic symptoms in adults. For children, doses of 20-40 mg can lead to toxicity. Doses of 600 mg are considered to be at lethal levels.^[3,13]

It has been observed that methods such as boiling, cooking, and frying potatoes have minimal effect on the level of solanine. Studies have shown that boiling potatoes only reduces the solanine level by approximately 1.2%.^[4] At 150 °C, deep frying also did not result in a significant change. It has been observed that decomposition and degradation begin at 170 °C, and with 10 minutes of frying at 210 °C, a 40% reduction in solanine content in potatoes is observed. Potatoes cooked in a microwave oven show a 15% reduction in solanine content.^[18,19]

Solanine poisoning primarily manifests itself with gastrointestinal and neurological disorders. Symptoms typically appear within the first 2-24 hours. If ingested in very high concentrations, symptoms may appear within 30 minutes. These symptoms include nausea, diarrhea, vomiting, stomach cramps, throat irritation, headache, dizziness, and irregular heart rhythms. In more severe cases, hallucinations, loss of consciousness, paralysis, fever, jaundice, dilated pupils, and hypothermia have been reported.^[6,13]

The initial treatment for solanine poisoning aims to reduce the release of the toxin from the intestine. For this purpose, the stomach is washed with a weak potassium permanganate solution. Subsequently, treatment should continue with activated charcoal or other drugs from the sorbent group, and drinking plenty of water is recommended.^[6]

In cases that are not very serious, spontaneous recovery is usually observed within 24 hours.^[20]

Mechanism of Action/Toxicity

There are several mechanisms proposed to explain how solanine induces toxicity in humans, but the exact mechanism of action is not fully understood. Glycoalkaloid toxicity has been shown to inhibit acetylcholinesterase activity in the central nervous system, disrupt membrane integrity affect the digestive system and overall body metabolism, and cause birth defects.^[21,22]

In vitro experiments have observed that α -solanine and α -chaconine are potent cytotoxins that rapidly induce cell lysis. When these two compounds act together, their cytotoxic potency significantly increases. The synergistic effect of these two molecules reveals their impact on disrupting the cell membrane. Poisonings caused by such glycoalkaloids are often difficult to diagnose because the observed symptoms resemble those of common gastrointestinal disorders.^[22] In another study, it was demonstrated that solanine can facilitate the opening of potassium channels in mitochondria, leading to the release of Ca^{2+} from these organelles. This results in an increase in Ca^{2+} concentration within the cell. The rapid and continuous increase in Ca^{2+} concentration is the earliest biochemical change observed in cells undergoing apoptosis. Therefore, exposure to solanine increases Ca^{2+} concentration in cells, triggering the apoptosis mechanism and formation, leading to cell damage. Further research is needed to understand how potassium channels are opened and which enzymes and genes are involved in this process.^[23] Symptoms observed in solanine poisoning, such as a burning sensation in the mouth, nausea, vomiting, stomach cramps, diarrhea, and internal bleeding, are likely to result from membrane disruptions occurring in cells.^[24]

Today, high-performance liquid chromatography (HPLC) is preferred for the determination of glycoside toxins.^[1] HPLC is an analytical method used for the separation, identification, and quantification of components present in a mixture.^[25] Laboratories can identify solanine from blood samples using the HPLC method.^[20]

Another method suggested is the use of a commercially available enzyme-linked immunosorbent assay (ELISA) kit that allows for the rapid analysis of glycoalkaloid content. Analysis results show that examinations conducted with both HPLC and ELISA kit are consistent with each other.^[21]

Recorded Poisoning Cases

Since people generally tend to avoid green potatoes, solanine poisonings are rarely observed.^[1]

In documented poisoning cases, after consuming potatoes containing 0.41 mg of solanine per gram in Scotland in 1918, 61 cases of solanine poisoning were reported, resulting in the death of a five-year-old child.^[26] Subsequently, in a case report dated 1925, it is documented that seven family members who consumed green potatoes fell ill due to solanine poisoning, resulting in the deaths of a 45-year-old mother and her 16-year-old daughter. However, the other family members fully recovered.^[27] In a report from 1979, it is noted that in a boarding school in the United Kingdom, 78 students fell ill after consuming potatoes for lunch, with 17 children requiring hospitalization. Gastrointestinal, circulatory, neurological, and dermatological findings were consistent with solanine poisoning according to laboratory investigations.^[28] In Canada, 61 children and teachers showed symptoms of solanine toxicity after consuming baked potatoes. Another mass poisoning case was reported in Canada in 1984. A similar incident occurred in Canada, where 61 out of 109 schoolchildren (along with their teachers) fell ill after consuming baked potatoes containing approximately 50mg of solanine per 100g (determined by an unspecified method). Symptoms such as nausea, abdominal cramps, headache, vomiting, fever, and diarrhea were believed to be a response to a solanine dose of approximately 2.5 mg/kg body weight.^[29]

Recorded poisoning cases are not limited to humans. Especially in cattle, sheep, and some ruminant animals, symptoms of solanine poisoning are observed. It is stated that there has been a significant increase in cases of poisoning and acidosis in animals such as potatoes and apples during harvest time.^[30,31]

In conclusion, solanine is a chemical compound produced by plants as a defense mechanism. Since this chemical compound is found in foods we commonly consume in our daily lives, we need to be extremely cautious when consuming them. Research has shown that doses of solanine found in potatoes ranging from 2-5 mg/kg-1 body weight can cause toxic symptoms, while doses of 6 mg/kg-1 body weight and above can lead to fatal outcomes. Potatoes should be stored in cool, dark environments to prevent an increase in solanine content due to environmental conditions. Green and sprouted potatoes should never be consumed as

they contain high concentrations of solanine. It has been concluded that solanine toxicity disrupts the digestive system, general body metabolism, and membrane integrity, and can cause birth defects. When an individual experiences solanine poisoning, practices such as removing the toxic substance from the stomach, administering mucosal protective agents, and analeptic drugs are employed. Potato-related solanine poisoning has made many people ill and resulted in some deaths according to statistics.

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REFERENCES

1. Bhambhani S, Kondhare KR, Giri AP. Diversity in Chemical Structures and Biological Properties of Plant Alkaloids. *Molecules*. 2021 Jun 3;26:3374.
2. Slanina P. Solanine (glycoalkaloids) in potatoes: toxicological evaluation. *Food Chem Toxicol*. 1990 Nov;28:759-61.
3. Liu JM, Wang SS, Zheng X, Jin N, Lu J, Huang YT, et al. Antimicrobial Activity Against Phytopathogens and Inhibitory Activity on Solanine in Potatoes of the Endophytic Bacteria Isolated From Potato Tubers. *Front Microbiol*. 2020 Nov 17;11:570926.
4. Phillips BJ, Hughes JA, Phillips JC, Walters DG, Anderson D, Tahourdin CS. A study of the toxic hazard that might be associated with the consumption of green potato tops. *Food Chem Toxicol*. 1996 May;34:439-48.
5. Nair JJ, van Staden J. Antiviral alkaloid principles of the plant family Amaryllidaceae. *Phytomedicine*. 2023 Jan;108:154480.
6. Vezikov LV, Simpson M. Plant Alkaloids Toxicity. 2023 Apr 29. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan--.
7. Hughes EH, Shanks JV. Metabolic engineering of plants for alkaloid production. *Metab Eng*. 2002 Jan;4:41-8.
8. Saito H, Miyairi S. Recent advances in cinchona alkaloid catalysis for enantioselective carbon-nitrogen bond formation reactions. *Curr Top Med Chem*. 2014;14:224-8.
9. Jain P. Secondary metabolites for antiulcer activity. *Nat Prod Res*. 2016;30:640-56.
10. Hassan SH, Gul S, Zahra HS, Maryam A, Shakir HA, Khan M, Irfan M. Alpha Solanine: A Novel Natural Bioactive Molecule with Anticancer Effects in Multiple Human Malignancies. *Nutr Cancer*. 2021;73:1541-52.
11. Lee MR. The Solanaceae: foods and poisons. *J R Coll Physicians Edinb*. 2006 Jun;36:162-9.
12. Shin M, Umezawa C, Shin T. NATURAL ANTI-MICROBIAL SYSTEMS | Antimicrobial Compounds in Plants. In:

- Batt CA, Tortorello ML, editors. *Encyclopedia of Food Microbiology* (Second Edition). Oxford: Academic Press; 2014. p. 920-9.
13. Izawa K, Amino Y, Kohmura M, Ueda Y, Kuroda M. 4.16-human–environment interactions—Taste. *Comprehensive Natural Products II*. 2010;631-71.
 14. Hodgson E. Toxins and venoms. *Progress in molecular biology and translational science*. 2012;112:373-415.
 15. Bock JH, Norris DO. Chapter 1 - Introduction to Forensic Plant Science. In: Bock JH, Norris DO, editors. *Forensic Plant Science*. San Diego: Academic Press; 2016. p. 1-22.
 16. Barceloux DG. Potatoes, tomatoes, and solanine toxicity (*Solanum tuberosum* L., *Solanum lycopersicum* L.). *Dis Mon*. 2009 Jun;55:391-402.
 17. Dalvi RR, Bowie WC. Toxicology of solanine: an overview. *Vet Hum Toxicol*. 1983 Feb;25:13-5.
 18. Maga JA. Potato glycoalkaloids. *Crit Rev Food Sci Nutr*. 1980;12:371-405.
 19. Friedman M. Potato glycoalkaloids and metabolites: roles in the plant and in the diet. *J Agric Food Chem*. 2006 Nov 15;54:8655-81.
 20. Chen Z, Li C, Yuan A, Gu T, Zhang F, Fan X, et al. α -Solanine Causes Cellular Dysfunction of Human Trophoblast Cells via Apoptosis and Autophagy. *Toxins (Basel)*. 2021 Jan 18;13:67.
 21. Friedman M, McDonald GM. Postharvest changes in glycoalkaloid content of potatoes. *Adv Exp Med Biol*. 1999;459:121-43.
 22. Koffi GY, Remaud-Simeon M, Due AE, Combes D. Isolation and chemoenzymatic treatment of glycoalkaloids from green, sprouting and rotting *Solanum tuberosum* potatoes for solanidine recovery. *Food Chem*. 2017 Apr 1;220:257-65.
 23. Gao SY, Wang QJ, Ji YB. Effect of solanine on the membrane potential of mitochondria in HepG2 cells and $[Ca^{2+}]_i$ in the cells. *World J Gastroenterol*. 2006 Jun 7;12:3359-67.
 24. Bianco G, Schmitt-Kopplin P, Crescenzi A, Comes S, Kettrup A, Cataldi TR. Evaluation of glycoalkaloids in tubers of genetically modified virus Y-resistant potato plants (var. Désirée) by non-aqueous capillary electrophoresis coupled with electrospray ionization mass spectrometry (NACE-ESI-MS). *Anal Bioanal Chem*. 2003 Mar;375:799-804. Epub 2003 Feb 27. Erratum in: *Anal Bioanal Chem*. 2003 Jul;376:763-4.
 25. Vogeser M, Seger C. A decade of HPLC-MS/MS in the routine clinical laboratory--goals for further developments. *Clin Biochem*. 2008 Jun;41:649-62.
 26. Willimott SG. An investigation of solanine poisoning. *Analyst*. 1933;58:431-9.
 27. Jadhav SJ, Sharma RP, Salunkhe DK. Naturally occurring toxic alkaloids in foods. *Crit Rev Toxicol*. 1981 Apr;9:21-104.
 28. McMillan M, Thompson JC. An outbreak of suspected solanine poisoning in schoolboys: Examinations of criteria of solanine poisoning. *Q J Med*. 1979 Apr;48:227-43.
 29. Hopkins J. The glycoalkaloids: naturally of interest (but a hot potato?). *Food Chem Toxicol*. 1995 Apr;33:323-8.
 30. Wang S, Panter KE, Gaffield W, Evans RC, Bunch TD. Effects of steroidal glycoalkaloids from potatoes (*Solanum tuberosum*) on in vitro bovine embryo development. *Anim Reprod Sci*. 2005 Feb;85:243-50.
 31. Solmaz V, Tekatas A, Erdoğan MA, Erbaş O. Exenatide, a GLP-1 analog, has healing effects on LPS-induced autism model: Inflammation, oxidative stress, gliosis, cerebral GABA, and serotonin interactions. *Int J Dev Neurosci*. 2020 Nov;80:601-12.