Potatoes, Tomatoes, and Solanine Toxicity (*Solanum tuberosum* L., *Solanum lycopersicum* L.)

Donald G. Barceloux, MD

History

The Inca people first cultivated the potato in the Altiplano of the High Andes of Chile during prehistoric times.[1] Spanish explorers brought the potato to Europe near the end of the 16th century. Although initially considered "peasant food," the cultivation of the potato spread across Europe including continental Europe, Britain, and Ireland. By 1780, the potato became a staple crop in Ireland. Although cultivation of the potato contributed to the rapid expansion of the Irish population between 1750 and 1850, a famine began in the late 1840s as a result of a fungal-induced potato blight [*Phytophthora infestans* (Mont.) de Bary]. During The Great Hunger in Ireland between 1845 and 1852, approximately one million deaths occurred along with the migration of 1.5 million people.[1] The creation of new transgenic potato cultivars with increased resistance against phytopathogens and improved composition of nutrients is an area of active research.

Although the toxicity of plants (eg, deadly nightshade, henbane) from the nightshade (Solanaceae) family has been known since ancient times, toxicity from members of the *Solanum* genus (eg, potato, tomato) has been documented only in modern times. Solanine intoxication is relatively rare considering the frequent use of potatoes and tomatoes as food staples. Outbreaks of solanine poisoning occur primarily when toxic glycoalkaloid concentrations increase substantially in commercial potatoes. Most sporadic cases of solanine toxicity involve children ingesting poisonous wild plant parts.[2]

This article was published in: Barceloux DG. Medical Toxicology of Natural Substances: Foods, Fungi, Medicinal Herbs, Toxic Plants, and Venomous Animals. Hoboken, NJ: John Wiley & Sons, 2008. pp. 77-83.
Dis Mon 2009;55:391-402
0011-5029/2009 $36.00 + 0
doi:10.1016/j.disamonth.2009.03.009

Botanical Description

Sweet potatoes [*Ipomoea batatas* (L.) Lam.] are a seasonal crop grown in tropical and subtropical regions, primarily for human consumption of the fresh or dried tubers. This South American native is a member of the morning glory family (Convolvulaceae) rather than the potato family. Sweet potatoes are widely cultivated in warm areas throughout the world as annual herbaceous plants. Three closely related, cultivated species of eggplant include *Solanum macrocarpon* L. (gboma eggplant), *Solanum aethiopicum* L. (scarlet eggplant, Ethiopian nightshade), and *Solanum melongena* L. (brinjal or aubergine eggplant).[3]

Common Name: Garden tomato

Scientific Name: *Solanum lycopersicum* L.

Botanical Family: Solanaceae (potato)

Physical Description: This herbaceous perennial plant produces a large berry with 2-12 locules that contain many small seeds. Most varieties of tomatoes are red when mature.

Distribution and Ecology: The tomato plant was originally cultivated in Mexico and Peru. European explorers brought seeds to Europe, and the tomato is now widely grown in temperate climates throughout the world.

Common Name: Eggplant, aubergine, brinjal

Scientific Name: *Solanum melongena* L.

Botanical Family: Solanaceae (potato)

Physical Description: This hairy, erect, annual, herbaceous plant reaches up to 1 m (about 3 ft) in height. The branched stems contain alternate, petiolate leaves with an ovate-elliptic shape. The flowers have a lobed calyx and a tubular, violet corolla. The fruit is a large ellipsoid berry that contains many brown seeds and turns purple when mature.

Distribution and Ecology: This perennial, frost-tender plant requires sunny and moist conditions. This species of eggplant is cultivated worldwide. Major areas of cultivation include China, Turkey, Japan, Mexico, Greece, Egypt, and Syria.

Common Name: Irish potato

Scientific Name: *Solanum tuberosum* L.

Botanical Family: Solanaceae (potato)

Physical Description: These annual, sprawling plants produce lavender to white flowers with yellow stamen. The relatively weak stems reach up to about 1 m (about 3 ft) in length with long pinnate leaves and ovate leaflets.

Distribution and Ecology: The potato is widely cultivated in temperate regions for the edible underground tubers.

TABLE 1. Glycoalkaloid structure of food products from genus *Solanum*

Plant	Scientific Name	Aglycone Skeleton	Glycoalkaloid
Potato	*Solanum tuberosum*	Solanidine	α-Chaconine, α-solanine
Tomato	*Solanum lycopersicum*	Tomatidine	α-Tomatine, dehydrotomatine
Eggplant	*Solanum melongena*	Solasodine	Solamargine, solasonine

Exposure

The genus *Solanum* contains a number of food-producing species including eggplant (*S. melongena)*, potatoes (*S. tuberosum*), and tomatoes (*S. lycopersicum*). African societies use the cholinesterase-lowering activity of solanine and chaconine in potatoes for the traditional treatment of human immunodeficiency virus (HIV) infections. Some communities (eg, Bangladesh immigrants in the UK) use tomato leaves as a food source without obvious toxicity.

Principal Toxins

Structure and Properties

Glycoalkaloids are natural plant glycosides that contain nitrogen in a steroidal structure (aglycone) and a carbohydrate side chain at the 3-OH position. The hexacyclic alkaloid aglycones are derived from cholesterol. Species of the genus *Solanum* that contain glycoalkaloids include the potato (α-solanine, α-chaconine), the tomato (α-tomatine, dehydrotomatine), and the eggplant (solasonine, solamargine).[4] Table 1 lists the basic glycoalkaloids and the corresponding steroid skeleton (aglycone) for potatoes, tomatoes, and eggplants. The glycoalkaloid content varies substantially between different cultivars depending on the strain and postharvest conditions (light, mechanical injury, storage). Potatoes may also contain small amounts of the glycoalkaloid hydrolysis products (β- and γ-chaconines, β- and γ-solanines) and solanidine.[5] Food processing (cooking, baking, frying) does not significantly alter the glycoalkaloid content. Boiling removed $<3.5\%$ of the main glycoalkaloids in potatoes, whereas microwaving decreases the concentrations of these compounds about 15%.[5] Significant degradation of these glycoalkaloids begins at temperatures above 170°C (338°F), and deep-frying at 150°C (302°F) does not significantly alter the concentrations of these glycoalkaloids. Heating potatoes to 210°C (410°F) for 10 minutes reduces the α-chaconine and α-solanine concentrations about 40%.[5] Potatoes and eggplants also contain water-soluble nortropane alkaloids, such as calystegine A_3

TABLE 2. Some physical properties of solanine

Physical Property	Value
Melting point	285°C[a]
Log P (octanol-water)	2.000
Water solubility	1.380 mg/L (25°C)
Vapor pressure	1.67E−34 mmHg (25°C)
Henry's Law Constant	1.22E−31 atm m^3/mole (25°C)
Atmospheric OH rate constant	3.72E−10 cm^3 molecule/s (25°C)

[a]Decomposes at this temperature.

and calystegine B_2.[6] There are few data on the human toxicity of these nortropane alkaloids.

Potatoes. Glycoalkaloids occur in all parts of potato plants including tubers, roots, sprouts, and leaves. In general, glycoalkaloids are α-compounds, whereas enzymatic or acid hydrolysis of the trisaccharide side chains of these glycoalkaloids produces β- and γ-compounds. The two major glycoalkaloids, α-solanine and α-chaconine, share the same aglycone (solanidine), but these two glycoalkaloids differ with respect to the composition of the sugar side-chain. α-Solanine (CAS RN: 20562-02-1, $C_{45}H_{73}NO_{15}$) is a tri-glycoside compound composed of galactose, glucose, and rhamnose, whereas α-chaconine is composed of glucose and two rhamnose moieties. Accumulation of glycoalkaloids stops after processing, but home processing (baking, boiling, frying, microwaving) does not eliminate significant amounts of glycoalkaloids from the potato.[7] Solanine is practically insoluble in water. Table 2 lists some of the physical properties of solanine.

Tomatoes. Tomatidine is the basic aglycone for the glycoalkaloids, α-tomatine and dehydrotomatine, in the tomato plant. These glycoalkaloids probably aid the defense of the tomato plant against bacteria, fungi, viruses, and insects.

Eggplant. Eggplant fruit contains a relatively large amount of antioxidant phenolic compounds, primarily hydroxycinnamic acid conjugates such as chlorogenic and dicaffeoylquinic acids.[8]

Poisonous Parts

Although the sweet potatoes contain low concentrations of a potentially toxic furanoterpenoid (ipomearone),[9] consumption of sweet potatoes has not been associated with human toxicity.

Potato. Although glycoalkaloids are present throughout the potato plant, the highest concentrations occur in the foliage, blossoms, periderm, cortex, and areas of high metabolic activity (eg, eyes, green skin, stems,

sprouts) followed by the peel and the tuber. α-Chaconine and α-solanine represent up to 95% of the glycoalkaloid content in potato tubers, and the general term solanine typically describes the glycoalkaloids present in *Solanum tuberosum*.[10] The glycoalkaloid concentration varies due to environmental and genetic factors. Ripe fruits contain the least amount of solanine. Factors that increase the solanine concentrations include physical injury to the plant, specific species (eg, potato cultivar Lenape), physiological stress, immaturity (green potato), low storage temperature, and storage in bright lighting.[11]

The typical ratio of α-chaconine to α-solanine in the tuber is about 60 : 40. The glycoalkaloid content of peels is substantially higher than tubers, particularly α-chaconine. In a study of Mexican potatoes, the peel always contained higher total glycoalkaloid concentrations than the tuber, and α-chaconine accounted for 65-71% of the total content of glycoalkaloids.[12] Potato leaves also contain higher concentrations of glycoalkaloids than the tubers, particularly α-chaconine. In a study of UK potatoes, the leaves contained 0.06-55.7 mg α-chaconine/100 g and 0.64-22.6 mg α-solanine/100 g.[13] Unprocessed tubers contained 0.3-0.63 mg α-chaconine/100 g and 0.05-0.65 mg α-solanine/100 g.

Normally, potato tubers (*Solanum tuberosum*) contain low concentrations of toxic glycoalkaloids (eg, solanine) unless adverse storage conditions or cultivation methods increase the solanine content. In a study of Mexican potatoes, the glycoalkaloid content of boiled peeled potatoes ranged from nondetectable to 9 mg/100 g fresh weight.[12] Because of the bitter taste of the alkaloids, solanine poisoning is rare except in times of food shortages when stressed or green potatoes are consumed. Normal food processing methods (baking, frying, broiling, microwaving) do not remove substantial amounts of glycoalkaloids from potatoes.[14] In a study of 20 commercial potato products, the α-chaconine and α-solanine content was lowest in canned boiled potatoes (0.04-0.08 mg/100 g and 0.04-0.06 mg/100 g, respectively) and highest in deep fat fried peels (93.1-97.9 mg/100 g and 46.1-48.0 mg/100 g, respectively).[15] The α-chaconine and α-solanine concentrations of individual commercial potato products were similar. The boiling of sprouted potatoes allows the diffusion of glycoalkaloids from the sprouts to the tuber and therefore increases the glycoalkaloid content of the tuber.[16]

Tomato. Tomato leaves and vines contain glycoalkaloids similar to solanine, but toxic glycoalkaloids are not usually detectable in the fruit. As listed in Table 3, the glycoalkaloid content varies between plant part and the type of glycoalkaloid. Approximately 72% of the original amount of α-tomatine remained after the preparation of Southern fried green

TABLE 3. Glycoalkaloid content in various parts of the tomato plant

Tomato Part	Dehydrotomatine[a]	α-Tomatine[a]
Fruit (green)	1498 ± 49	16,285 ± 112
Fruit (red)	ND	ND
Flowers	1023 ± 3	4825 ± 191
Calyxes	370 ± 17	2870 ± 129
Leaves	304 ± 15	1847 ± 112
Stems	331 ± 4	1547 ± 32
Roots	Trace	Trace

Source: Data from Ref. 18.

[a]Mean ± standard deviation of three samples expressed as μg/g fresh weight; ND, nondetectable.

tomatoes using traditional cooking methods.[17] In a study of three tomato plants, the ratio of mean concentrations of α-tomatine to dehydrotomatine ranged from 4.7 in the stems and flowers to 10.9 in green fruit as measured by high performance liquid chromatography (HPLC) with UV detection.[18] There were trace amounts of glycoalkaloid compounds in the roots and nondetectable concentrations in mature fruit.

Mechanism of Toxicity

In vitro studies indicate that both α-solanine and α-chaconine are reversible inhibitors of human plasma cholinesterase (butyrylcholinesterase).[19] In a study of rabbits, intraperitoneal doses of solanine caused mild to moderate inhibition of both specific and nonspecific cholinesterases.[20] The relevance of plasma cholinesterase inhibition to human solanine toxicity is unclear because solanine toxicity is not classically associated with a cholinergic syndrome. In experimental animals, α-solanine and α-chaconine also demonstrate cytotoxic properties.[13,21]

Dose Response

In animal models, the toxicity of solanine depends on the dose, species, and route of administration with the parenteral route much more toxic than the oral route of exposure.[22] Volunteer studies using taste panels suggest that a bitter taste and burning sensation in the mouth occurs when the glycoalkaloid content of potatoes exceeds 14 mg/100 g and 22 mg/100 g fresh weight, respectively.[23,24] The presence of total glycoalkaloid content exceeding 60 mg/100 g fresh weight produces strong bitterness and burning.[25] The total glycoalkaloid content of most commercial potatoes does not usually exceed 10 mg/100 g. The US Food & Drug Administration (FDA) limits solanine content in potatoes to ≤20 mg/100 g potatoes.

Toxicokinetics

Animal studies indicate that the absorption of solanine from the gastrointestinal tract is poor, and solanine distributes in the highest concentration to the spleen with progressively lower concentrations in the kidney, liver, lung, fat, heart, brain, and blood.[26] In a study of seven healthy volunteers, the ingestion of mashed potatoes containing glycoalkaloid concentrations of 1 mg/kg (α-solanine:α-chaconine ratio = 41 : 59) produced peak serum α-solanine and α-chaconine concentrations at approximately 5 $\pm$ 1 hours and 6 $\pm$ 1 hours, respectively.[27] The biological half-lives of α-solanine and α-chaconine in serum samples were about 11 hours and 19 hours, respectively. The stomach hydrolyzes solanine to the less toxic glycoalkaloid, solanidine. Elimination occurs rapidly in the feces and to a lesser extent in urine. Animal studies indicate that the gastrointestinal tract also poorly absorbs α-chaconine with excretion occurring rapidly in the feces, probably by biliary excretion.[28] The elimination of α-chaconine and α-solanine is similar with the major metabolite being the aglycone, solanidine. Fig 1 demonstrates the degradation of major glycoalkaloids in the potato.

Clinical Response

Clinical features of solanine poisoning include gastrointestinal and neurologic symptoms, particularly vomiting, headache, and flushing. The glycoalkaloid content of young leaves is substantially higher than the tubers[13]; therefore, ingestion of the above-ground portion of these plants can cause gastrointestinal distress. The largest series of solanine poisoning involved an English day school where 78 schoolboys developed diarrhea and vomiting after eating potatoes stored since the summer term.[29] Symptoms began 7-19 hours after ingestion with vomiting, diarrhea, anorexia, and malaise. Of the 78 boys, 17 were admitted to the hospital. Other symptoms included fever (88%), altered mental status (drowsiness, confusion, delirium) (82%), restlessness (47%), headache (29%), and hallucinations (23%). Three boys were seriously ill with hypotension, tachycardia, and stupor out of proportion to fluid and electrolyte imbalance. These boys were discharged 6-11 days after admission, and they had nonspecific symptoms and visual blurring for several weeks after release from the hospital.

Fatalities from solanine poisoning are not well documented in the modern medical literature. Deaths have been associated with consumption of toxic potatoes, but those reports involved malnourished patients who may not have received adequate care.[30] Headache,

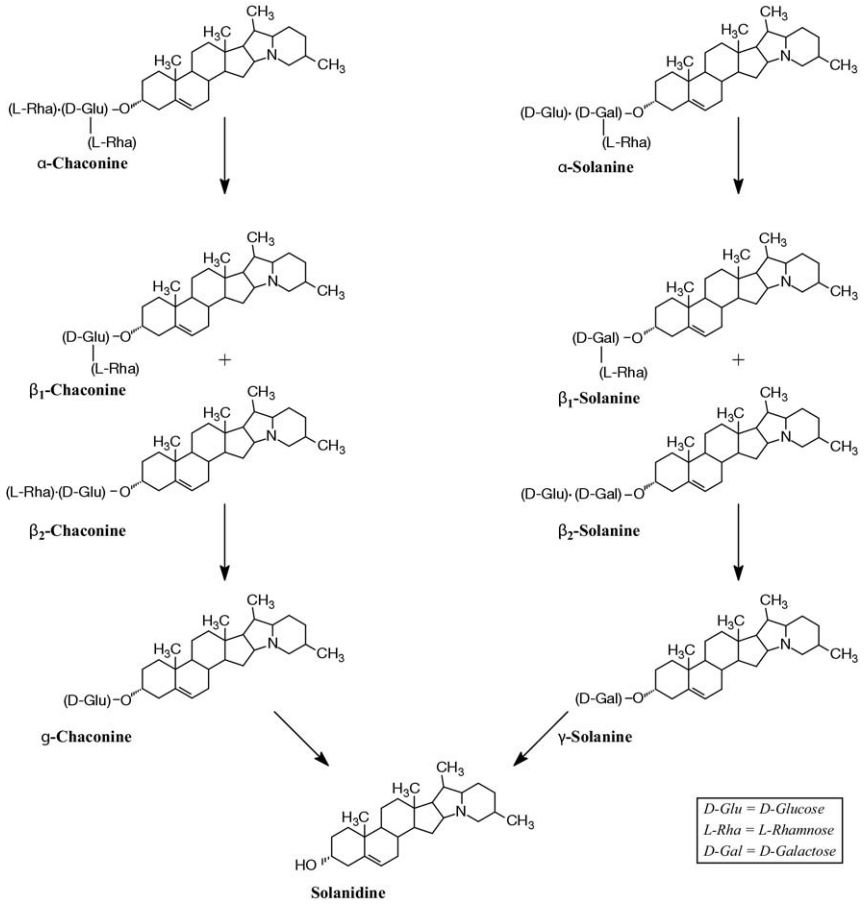

FIG 1. Hydrolysis of the trisaccharide side chains of potato glycoalkaloids α-chaconine and α-solanine to the aglycone solanidine. Adapted from Friedman M, et al. Postharvest changes in glycoalkaloid content of potatoes. Adv Exp Med Biol 1999;459:124.

abdominal pain, vomiting, thirst, restlessness, and apathy preceded death, but no convulsions or fever were reported. There are no conclusive data that constituents of potatoes or tomatoes are teratogenic to humans.[31] In studies of hamsters, acute maternal toxicity limited the administration of dosages high enough to induce statistically significant levels of terata in litters receiving α-chaconine and α-solanine.[32] However, the administration of toxic doses of the aglycone solanidine and the derivative solanidine *N*-oxide produced a statistically significant ($P < 0.005$) increase in the incidence of malformations. Although teas brewed from the tomato plant have been

associated with solanine-like poisoning in some reference books,[33] there are few data that indicate tomatoes cause toxicity.

Diagnostic Testing

Methods for the detection and quantitation of glycoalkaloids include gas chromatography,[34] high performance liquid chromatography (HPLC),[35,36] HPLC with electrospray ionization and tandem mass spectrometry,[37] and gas chromatography mass spectrometry.[38] Enzyme-linked immunosorbent assay (ELISA) methods allow the rapid determination of glycoalkaloid content in plant parts from the potato including the tuber.[39] HPLC is commonly used for the determination of glycoalkaloid content of fresh and processed potatoes, including the concentration of individual glycoalkaloids in various plant parts. The limits of detection for individual glycoalkaloids in serum are about 0.3 ng/mL as determined by HPLC.[27] The AOAC International (Association of Analytical Chemists International, Gaithersburg, MD; http://www.eoma.aoac.org/) official method of analysis for glycoalkaloids in potato tubers involves HPLC with ultraviolet detection (HPLC/UV).[40] The analytical range for this method is 10-200 mg α-solanine/kg and 20-250 mg α-chaconine/kg. Following the ingestion of approximately 0.41 mg α-solanine/kg body weight and 0.59 mg α-chaconine/kg body weight, the mean peak concentration of these glycoalkaloids were about 8 ng/mL and 14 ng/mL, respectively.[27] In general, glycoalkaloid content of potatoes should not exceed 200 mg/kg fresh potatoes.

Treatment

The treatment of solanine poisoning is entirely supportive. Most patients with solanine poisoning develop vomiting and diarrhea; therefore, decontamination measures are not usually necessary. There are no clinical or experimental data to indicate that decontamination measures improve outcome during solanine poisoning. Dehydration and electrolyte imbalance are the most common serious complications of solanine poisoning, and patients with substantial vomiting and diarrhea should be evaluated for the presence of these complications. Treatment involves standard measures to correct fluid and electrolyte imbalance. For those seriously ill patients who do not respond to fluid replacement, cardiac monitoring and vasopressors may be required. Those patients with neurologic abnormalities or serious electrolyte imbalance should be observed until these complications resolve.

REFERENCES

1. Lee MR. The Solanaceae: foods and poisons. J R Coll Physicians Edinb 2006; 36:162-9.
2. Edwards RO Jr. Poisoning from plant ingestions. J Fla Med Assoc 1965;52:875-81.
3. van Eck J, Snyder A. Eggplant (*Solanum melongena* L.). Meth Mol Biol 2006;343:439-47.
4. Jadhav SJ, Sharma RP, Salunkhe DK. Naturally occurring toxic alkaloids in foods. CRC Crit Rev Toxicol 1981;9:21-104.
5. Friedman M. Potato glycoalkaloids and metabolites: roles in the plant and in the diet. J Agric Food Chem 2006;54:8655-81.
6. Friedman M, Roitman JN, Kozukue N. Glycoalkaloid and calystegine contents of eight potato cultivars. J Agric Food Chem 2003;51:2964-73.
7. Friedman M, McDonald GM. Postharvest changes in glycoalkaloid content of potatoes. Adv Exp Med Biol 1999;459:121-43.
8. Whitaker BD, Stommel JR. Distribution of hydroxycinnamic acid conjugates in fruit of commercial eggplant (*Solanum melongena* L.) cultivars. J Agric Food Chem 2003;51:3448-54.
9. Coxon DJ, Curtis RF, Howard B. Ipomearone, a toxic furanoterpenoid in sweet potatoes (*Ipomea batatas*) in the United Kingdom. Food Cosmet Toxicol 1975;13:87-90.
10. Slanina P. Solanine (glycoalkaloids) in potatoes: toxicological evaluation. Food Chem Toxicol 1990;28:759-61.
11. Dimenstein L, Lisker N, Kedar N, et al. Changes in the content of steroidal glycoalkaloids in potato tubers grown in the field and in the greenhouse under different conditions of light, temperature and daylength. Physiol Mol Plant Pathol 1997;50:391-402.
12. Sotelo A, Serrano B. High-performance liquid chromatographic determination of the glycoalkaloids α-solanine and α-chaconine in 12 commercial varieties of Mexican potato. J Agric Food Chem 2000;48:2472-5.
13. Phillips BJ, Hughes JA, Phillips JC, et al. A study of the toxic hazard that might be associated with the consumption of green potato tops. Food Chem Toxicol 1996;34:439-48.
14. Takagi K, Toyoda M, Fuiyama Y, et al. Effect of cooking on the contents of α-chaconine and α-solanine of potatoes. J Food Hyg Soc Japan 1990;31:67-73.
15. Bushway RJ, Ponnampalam R. α-Chaconine and α-solanine content of potato products and their stability during several modes of cooking. J Agric Food Chem 1981;29:814-7.
16. Gonmori K, Shindo S. Effect of cooking on the concentration of solanine in potato. Res Pract Forensic Med 1985;28:91-3.
17. Friedman M, Levin CE. α-Tomatine content in tomatoes and tomato products determined by HPLC with pulsed amperometric detection. J Agric Food Chem 1995;43:1507-11.
18. Kozukue N, Han JS, Lee KR, et al. Dehydrotomatine and alpha-tomatine content in tomato fruits and vegetative plant tissues. J Agric Food Chem 2004;52:2079-83.
19. Nigg HN, Ramos LE, Graham EM, et al. Inhibition of human plasma and serum butyrylcholinesterase (EC3.1.1.8) by α-chaconine and α-solanine. Fund Appl Toxicol 1996;33:272-81.

20. Patil BC, Sharma RP, Salunkhe DK, et al. Evaluation of solanine toxicity. Food Cosmet Toxicol 1972;10:395-8.
21. Roddick JG, Rijnenberg AL, Osman SF. Synergistic interaction between potato glycoalkaloids α-solanine and α-chaconine in relation to destabilization of cell membranes. J Chem Ecol 1988;14:889-902.
22. Dalvi RR, Bowie WC. Toxicology of solanine: an overview. Vet Hum Toxicol 1983;25:13-5.
23. Johns T, Keen SL. Taste evaluation of potato glycoalkaloids by the Ayamara: a case study in human chemical ecology. Hum Ecol 1986;14;437-52.
24. Zitnak A, Filadelfi MA. Estimation of taste thresholds of three potato glycoalkaloids. J Can Inst Food Sci Technol 1985;18:337-9.
25. Sinden SL, Deahl KL, Aulenbach BB. Effect of glycoalkaloids and phenolics on potato flavor. J Food Sci 1976;41:520-3.
26. Nishie K, Gumbmann MR, Keyl AC. Pharmacology of solanine. Toxicol Appl Pharmacol 1971;19:81-92.
27. Hellenas K-E, Nyman A, Slanina P, et al. Determination of potato glycoalkaloids and their aglycone in blood serum by high-performance liquid chromatography. J Chromatogr Biomed Appl 1992;573:69-78.
28. Norred WP, Nishie K, Osman SF. Excretion, distribution and metabolic fate of 3H-alpha-chaconine. Res Commun Chem Pathol Pharmacol 1976;13: 161-71.
29. McMillan M, Thompson JC. An outbreak of suspected solanine poisoning in school boys: an examination of criteria of solanine poisoning. Q J Med 1979;48:227-43.
30. Hansen AA. Two fatal cases of potato poisoning. Science 1925;61:348-9.
31. Chaube S, Swinyard CA. Teratological and toxicological studies of alkaloidal and phenolic compounds from *Solanum tuberosum* L. Toxicol Appl Pharmacol 1976;36:227-37.
32. Gaffield W, Keeler RF. Induction of terata in hamsters by solanidane alkaloids derived from *Solanum tuberosum*. Chem Res Toxicol 1996;9:426-33.
33. Hardin JW, Arena JM. Human poisoning from native and cultivated plants, 2nd ed. Durham, NC: Duke University Press, 1974. p. 140.
34. Bushway RJ, McGann DF, Bushway A. Gas chromatographic method for the determination of solanidine and tis application to a study of fed-milk transfer in the cow. J Agric Food Chem 1984;32:548-51.
35. Sotelo A, Serrano B. High-performance liquid chromatographic determination of the glycoalkaloids alpha-solanine and alpha-chaconine in 12 commercial varieties of Mexican potato. J Agric Food Chem 2000;48:2472-5.
36. Kodamatani H, Saito K, Niina N, et al. Simple and sensitive method for determination of glycoalkaloids in potato tubers by high-performance liquid chromatography with chemiluminescence detection. J Chromatogr A 2005;1100: 26-31.
37. Cataldi TR, Lelario F, Bufo SA. Analysis of tomato glycoalkaloids by liquid chromatography coupled with electro-spray ionization tandem mass spectrometry. Rapid Commun Mass Spectrometry 2005;19:3103-10.
38. van Gelder WM, Tuinstra LG, Van Der Greef J, et al. Characterization of novel steroidal alkaloids from tubers of *Solanum* species by combined gas chromato-

graphy-mass spectrometry. Implications for potato breeding. J Chromatogr 1989;482:13-22.
39. Friedman M, Bautista F, Stanker LH, et al. Analysis of potato glycoalkaloids by a new ELISA kit. J Agric Food Chem 1998;46:5097-102.
40. Horwitz W, Official methods of analysis of AOAC International, 17th ed. Gaithersburg, MD: AOAC International, 2000, Official method 997.13.