Potato glycoalkaloids: Some unanswered questions

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Despite its status as a food of global importance, the potato tuber contains toxic glycoalkaloids (GA) that cause sporadic outbreaks of poisoning in humans. Probably because GA are natural compounds present in a staple food that has been used for millenia, their effects on humans have not been investigated as thoroughly as they would have been had they been synthetic additives – the use of which is stringently regulated. As a result, a number of lingering uncertainties remain concerning both the presence of GA in potato tubers and potato products and their effects on humans.

As one of the world's major agricultural crops, the cultivated potato (*Solanum tuberosum* L.) is consumed daily by millions of people from diverse cultural backgrounds. Potatoes are grown in ~80% of all countries, and worldwide production stands in excess of 300 million tonnes per annum, a figure exceeded only by wheat, maize and rice[1]. Although the success of the potato as a starchy staple has been enhanced by breeding for resistance to diseases and pests, early domestication was probably more concerned with reducing or eliminating the levels of the natural bitter-tasting steroidal toxicants in the tubers, known as glycoalkaloids (GA). Despite the fact that levels of GA are much lower in modern cultivars than in wild progenitors, if the potato were to be introduced today as a novel food, it is quite possible that its use would not be approved because of the presence of these potentially toxic compounds.

GA were first identified in potatoes by Baup[2] early in the 19th century, since when a vast body of information on their chemistry, biochemistry, distribution, physiology and toxicology has accrued, and been reviewed[3–5]. The two related compounds α-solanine and α-chaconine account for ~95% of the GA present in *S. tuberosum* (Fig. 1). These consist of a nonpolar lipophilic steroid nucleus that is extended by two fused nitrogen-containing heterocyclic rings at one end and bound to a polar water-soluble trisaccharide at the other. Solanine and chaconine share the same aglycone, namely solanidine, but differ in their carbohydrate component. In addition, small amounts of the aglycone (i.e. solanidine) and partial glycosides (i.e. solanidine coupled to the mono- or disaccharides) are normally present, probably as intermediates of biosynthesis and/or degradation.

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Are GA present in potatoes and potato products?

GA are found throughout the potato plant with levels varying considerably among different organs (Table 1) and among the same organs in different plants and varieties. In the tuber, GA are concentrated mainly in the outer 1.5-mm layer (Table 1). Levels of GA in commercial tubers are normally less than the widely accepted 'safety limit' of 200 mg/kg fresh weight (see below), and although surveys show values in excess of this limit in 2–9% of samples[6,12], peeling usually removes most of the GA in the tuber. However, jacket potatoes and, more recently, potato skin preparations have a relatively high content of GA; levels in excess of the 200 mg/kg limit (and up to eightfold higher) have been reported for potato skin preparations and potato crisps made from unpeeled potatoes[13,14] (Table 2). Moreover, heat processing does not inactivate potato GA[5].

By and large, the levels of GA in commercially available, quality potato tubers are not thought to represent a health hazard to humans. However, many companies routinely test potatoes or potato-based ingredients for GA, as genetic and pre- and postharvest factors can increase levels in the tuber. For example, a cool and wet growing season is thought to have been partly responsible for the withdrawal of the 1986 crop of the variety Magnum Bonum in Sweden, which had unacceptably high levels of GA[21]. Small potatoes, which have a higher surface area to volume ratio, tend to have higher levels of GA, on a weight for weight basis, than do larger potatoes[22]. Synthesis of GA can also be markedly stimulated in tubers subjected to physiological stresses such as physical damage (e.g. cutting and bruising, which can arise during harvest or transit), exposure to light (which, independently, can stimulate chlorophyll synthesis leading to 'greening' of tubers) or microbial and/or herbivore attack[3]. Such effects on tubers that are subsequently used for skin-on or peel-based products, which have higher base levels of GA, could therefore pose potential health risks.

Levels of GA in tubers and foliage are positively correlated with each other, and it has not yet proved possible to reduce the levels in tubers (to enhance edibility and safety) while maintaining or increasing the levels in leaves (which contribute to resistance to disease and predation) using conventional breeding practices. Consequently, genetic engineering approaches are now being considered, but realization of the experimental or commercial

PII: S0924-2244(96)10013-3

potential of such approaches still appears to be some way off.

How toxic are potato GA to humans?

Although the claim that GA from blighted potatoes (i.e. potatoes infected with late blight – *Phytophthora infestans*) might be responsible for anencephaly and spina bifida[23] has not been upheld, at least 12 separate cases of human poisoning from potato consumption, involving nearly 2000 people and 30 fatalities, have been recorded[24]. Available information suggests that the susceptibility of humans to glycoalkaloid poisoning is both high and very variable: oral doses in the range 1–5 mg/kg body weight are marginally to severely toxic to humans whereas 3–6 mg/kg body weight can be lethal[24,25]. The narrow margin between toxicity and lethality is obviously of concern. Although serious glycoalkaloid poisoning of humans is rare, there is a widely held suspicion that mild poisoning is more prevalent than supposed; however, because the symptoms (e.g. abdominal pain, vomiting, diarrhoea) are similar to those of other common gastrointestinal ailments, it is rarely diagnosed or treated. The widely accepted safety limit for the levels of GA in tubers remains at 200 mg/kg fresh weight – a level that was proposed more than 70 years ago[26] when little information was available concerning subacute and chronic glycoalkaloid toxicity. However, owing to the large and often unpredictable variations in levels of GA, which can arise from differences in variety, locality, season, cultural practices and stress factors, and the fact that so many aspects of the biochemistry and toxicity of these compounds remain poorly understood, it has been suggested that the limit should be reduced to 60–70 mg/kg (Ref. 27).

What is the mode of toxicity of potato GA in humans?

Glycoalkaloid poisoning elicits a wide variety of symptoms – ranging from gastrointestinal disorders, through confusion, hallucination and partial paralysis to convulsions, coma and death – but is thought to stem from one or both of two quite distinct modes of action. The first is inhibition of the enzyme acetylcholinesterase, which is responsible for hydrolysing the neurotransmitter acetylcholine, a key process in nerve impulse conduction across cholinergic synapses[28]. Neurological symptoms such as weakness, confusion and depression, which have been noted in patients suffering from glycoalkaloid poisoning, are likely manifestations of this antiacetylcholinesterase activity[29]. Chaconine and solanine are equally potent inhibitors of acetylcholinesterase[28].

The other major biological action of GA is their ability to disrupt sterol-containing membranes[30]. This action is thought to be responsible for damaging cells in the gastrointestinal tract and also in other tissues or organs into which GA are transported (e.g. blood, liver) following absorption. With regard to this effect, chaconine is the more active of the two GA in many organisms and membrane systems. Because of the different potencies

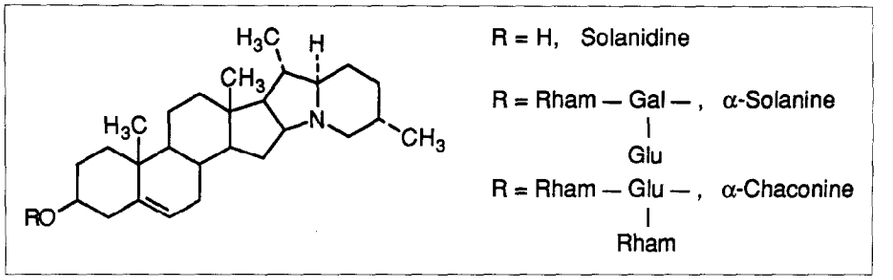

Fig. 1
Structures of the principal potato glycoalkaloids. Glu, Gal and Rham represent the sugar residues glucose, galactose and rhamnose, respectively.

Table 1. Levels of glycoalkaloids (GA) in various parts of the potato plant

Plant part	GA concentration (mg/kg fresh weight)	Refs
Flowers	2150–5000	6, 7
Leaves	230–1000	8, 9
Stems	23–33	7
Roots	180–400	7
Bitter-tasting tuber	250–800	10
Whole tuber	10–150	6
Skin (2–3% of tuber)	300–640	7
Peel (10–12% of tuber)	150–1068	6, 7
Flesh	12–100	7
Cortex	125	11
Pith	Not detectable	11
Sprouts	2000–7300	6, 7

Table 2. Levels of glycoalkaloids (GA) in various commercial potato products and preparations

Product or preparation	GA concentration (mg/kg product)	Refs
Boiled peeled potato[a]	27–42	15
Baked jacket potato[a]	99–113	16
Chips (US French fries)	0.4–8	13
Fried skins	567–1450	16
Frozen mashed potato	2–5	16
Frozen baked potato	80–123	16
Frozen chips	2–29	16
Frozen skins	65–121	17
Frozen fried potato	4–31	16, 18
Canned peeled potato	1–2	16
Canned whole new potato	24–34	19
Crisps (US potato chips)	23–180	13, 16, 20
Crisps (with skin)	95–720	14
Dehydrated potato flour	65–75	16
Dehydrated potato flakes	15–23	16

[a] Non-commercial preparation

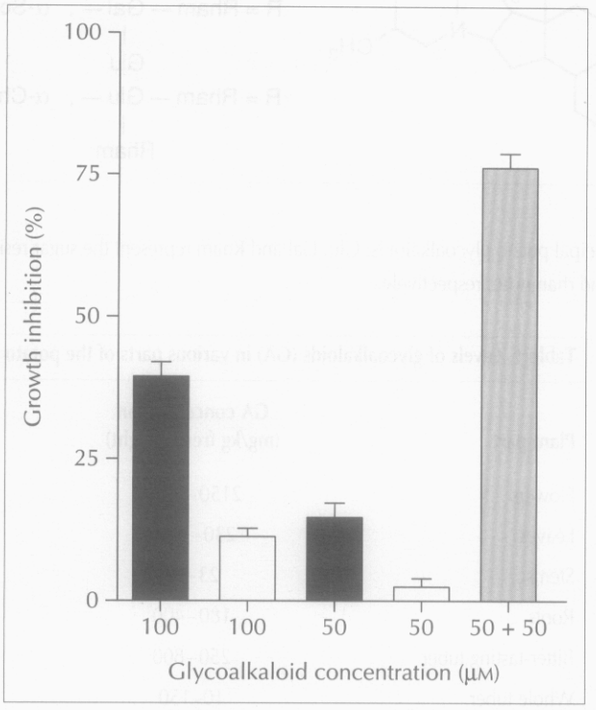

Fig. 2

Effects of the glycoalkaloids chaconine (black bars) and solanine (white bars), individually and in a 1 : 1 combination (grey bar), on the growth of the fungus *Alternaria brassicicola*. Data are means of four replicates. The thin vertical bars represent the standard error for each mean. Redrawn from Fewell and Roddick[33], with permission.

of solanine and chaconine in these different systems, the overall effect of ingested GA should therefore be dependent on which system they primarily act on in the body. As yet, this is far from clear because of the difficulties of conducting toxicological studies with humans. The relative rapidity of symptoms of glycoalkaloid toxicity (0.5–12 hours) suggests that their primary toxic effect may be due to gastrointestinal damage[25] with the secondary occurrence of neurological disorders, which is in keeping with studies with laboratory mammals[31]. Uncertainty concerning the effects of ingested GA is compounded by the fact that, in combination, solanine and chaconine can interact synergistically, which results in a marked intensification of the overall activity.

What is the nature of the synergism between solanine and chaconine?

The synergistic action (i.e. an interaction between two or more factors whereby the combined activity of all the factors is greater than the sum of their individual activities) of solanine and chaconine in disrupting membranes was first demonstrated using liposomes (synthetic membrane vesicles)[32], and has since been demonstrated with plant and animal cells as well as fungi[33] (Fig. 2). The synergistic activity relates to the membrane-disrupting properties of the two potato GA but apparently not to their inhibition of acetylcholinesterase[34]. Interestingly, its magnitude varies with the solanine : chaconine ratio (Fig. 3)[33,35]. Whether the synergism between these GA is of relevance to toxicity in animals, including humans, is not yet known. However, it is highly likely, given that synergism has been observed in the case of glycoalkaloid-mediated damage of blood cells[35]. Because of this, published toxicity data (e.g. LD_{50} – the dose that is lethal to 50% of the test organisms) for individual GA in laboratory mammals may give a misleading impression of the toxicity of glycoalkaloid mixtures in potato, and therefore require re-evaluation. Clearly, this synergistic interaction needs to be borne in mind when considering the toxicity of GA to humans and when analysing potato products for the presence of GA.

Synergistic interactions are not restricted to solanine and chaconine but also occur between other GA such as the two solasodine-based GA solasonine and solamargine[36] (Fig. 4). These GA are not present in tubers of commercial potato varieties but have been found in tubers of some wild *Solanum* spp. and hybrids used in potato breeding[6]. The fact that 'alien' GA can be introduced into potatoes through breeding[6] stresses the need for knowledge of how the GA in *S. tuberosum* interact with other GA, and also for closer consideration of the exact content of GA in potato tubers. Such an approach would differ from the usual approach of assessing total levels of GA, regardless of profiles, in potatoes and potato products.

How significant is the profile of GA in the potato?

Because the overall effects of solanine and chaconine mixtures depend on the ratio of the GA, values for acceptable total levels of GA in tubers might need to vary according to the particular ratio of GA present, which itself can vary in different potato tissues. Many studies on potato GA have not addressed the question of the profile of GA directly, providing a figure only for total levels. In some cases this is because of the assay method employed. Immunoassays have been developed to provide highly practicable and sensitive means of screening levels of GA in potatoes and potato products[37]. However, recently developed immunoassays based on monoclonal antibodies are not able to discriminate between solanine and chaconine (or other GA), and cannot therefore provide information on glycoalkaloid profiles[38]. High-performance liquid chromatography, on the other hand, offers both qualitative and quantitative information on GA present (for example, see Ref. 39), giving a more complete profile, although it is not as convenient as immunoassays for screening large numbers of samples.

As indicated above, production of GA is very much influenced by environmental factors. The effects of the environment on the accumulation of potato GA have been extensively investigated, but very few of the studies have considered how the ratio of the two major potato GA (or the glycoalkaloid profile) is affected by environmental factors. In view of the synergistic interactions mentioned above, such information could prove valuable in assessing the health risks posed by potato GA.

In addition, the effects of the use of wild germplasm in breeding programmes should perhaps be more closely

monitored with respect to how it might cause changes in the qualitative and/or quantitative make-up of GA in potatoes; new varieties might possess novel combinations of GA, the interactions of which might result in unpredictable degrees of toxicity. For example, solasodine-based GA have been found in hybrid tubers of *S. tuberosum* and the wild potato *Solanum vernei*[6]. Although the actual levels were low, concern arises because these compounds can interact synergistically with solanidine-based GA[36], and are also considered to have teratogenic potential[40]. Wild germplasm might, in addition, alter glycoalkaloid profiles by bringing about changes in the way the synthesis of GA responds to environmental factors. In the variety Kennebec, 'ageing' of tuber slices results in the synthesis of the tomatidenol-based GA α- and β-solamarine (Fig. 4), apparently as a result of germplasm inherited from the wild species *Solanum demissum*[41]. Knowledge of the toxicological/pharmacological properties of potato GA, other than solanine and chaconine, found both in tubers and in breeding stock is virtually non-existent, and again further investigation is warranted. Speculation that potato GA may interact with other natural compounds present in potatoes, such as saponins or phenolics, arises from various reports that extracts of *Solanum* spp. are more toxic than the pure GA. In a dietary context, the effects of additives and/or condiments such as vinegar (the acidity of which could influence the solubility of GA, which are bases), cheese (which contains cholesterol, which is known to complex with GA) and salt (which is generally physiologically active) provide further unknowns.

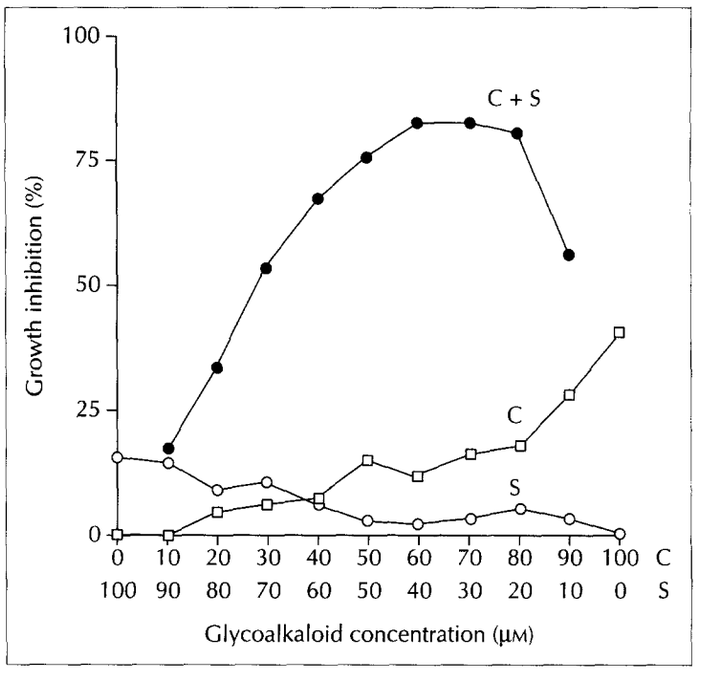

Fig. 3
Effects of the glycoalkaloids (GA) solanine (S) and chaconine (C), individually and in different combinations, on the growth of the fungus *Alternaria brassicicola*. In combination treatments, the total GA concentration was 100 μM. Each point is based on four replicate determinations. Redrawn from Fewell and Roddick[33], with permission.

How are potato GA metabolized?

One aspect of the biochemistry of potato GA that has received relatively little attention is the fate of the GA once consumed. The GA certainly appear to be much more toxic than their aglycones; for example, hamsters force-fed with various GA showed severe intestinal epithelial necrosis, whereas no such lesions arose in aglycone-treated animals[31]. Incidentally, the aglycones also show minor to negligible inhibition of acetylcholinesterase. Hence, the toxicity of potato GA is likely to be influenced by the detoxification of GA as a result of hydrolysis in the gut environment.

The extent of metabolism of GA in the human gut is, however, far from clear. Apparently, enzymic hydrolysis does not occur in the upper digestive tract of humans or other monogastric animals[42]; moreover, *in vitro*, acid-catalysed hydrolysis by human gastric juice is limited and variable[43]. Reports that the aglycone solanidine can be detected in the blood[25] following oral consumption of a potato meal containing intact GA suggest that hydrolysis occurs at some stage, although intact GA are the main form found in the plasma. Also of interest is the fact that chaconine and solanine appear to be metabolized differentially in human blood[25], resulting in changing solanine : chaconine ratios, which could influence any synergistic interaction between the two GA and hence

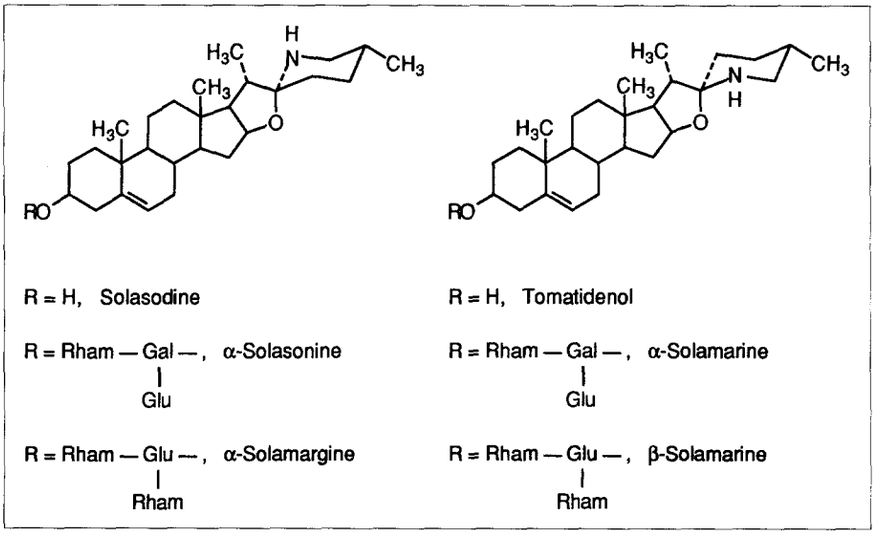

Fig. 4
Structures of solasodine-based and tomatidenol-based glycoalkaloids. Glu, Gal and Rham represent the sugar residues glucose, galactose and rhamnose, respectively.

overall toxicity. Thus, it appears that GA consumed as constituents of potato flesh can pass through the gut without being metabolized and enter the bloodstream.

Can potato GA elicit chronic toxicity?

Most of the recorded instances of toxicity from potato GA are of acute toxicity, although greater awareness of the potential threat from GA, coupled with breeding for lower levels of tuber GA and more stringent quality control of foods, have largely eliminated such occurrences. However, the fact that potatoes (and hence GA) are consumed on a daily or fairly frequent basis in many countries raises questions about the possible chronic toxic effects from prolonged ingestion of GA. A value of 14 mg for mean daily intake has been estimated for the UK[44], based on an average daily consumption of 140 g potatoes and a whole-tuber content of 100 mg/kg. Because tubers are generally peeled before being eaten, the value is likely to be less than 14 mg, although regular consumption of skin-on preparations or products could significantly elevate intake. Hopkins[44] stresses how an equivalent intake of potentially toxic synthetic additives would not be permitted without extensive toxicity testing beforehand (e.g. genotoxicity, carcinogenicity). Although the potato GA have been Ames-tested for mutagenicity and found to be negative[45], reservations have been expressed about the comprehensiveness of the tests[44].

Of particular importance is the extent of accumulation of GA from the diet as influenced by metabolism in the body. Absorption of potato GA in humans is apparently proportional to the amount ingested[46], but once in the bloodstream, excretion rates appear to be low, implying that the compounds might accumulate in various organs of the body, including the liver[47]. The question has also been raised as to whether accumulated GA might be mobilized during periods of stress such as pregnancy, illness or starvation[47]. Long-term studies of the relationship between dietary levels of GA and levels of GA in the blood, urine and faeces of humans are needed to elucidate the body burden of these compounds in normal individuals and also whether humans can develop tolerance to GA following continuous exposure.

The question of chronic glycoalkaloid toxicity is, therefore, still wide open. Amazingly, there appear to have been no studies with experimental animals involving prolonged exposure to repeated subacute doses of potato GA, although there is clearly a need for such data, especially in relation to reproduction[44]. Species differences in sensitivity and response to GA also need to be considered. Indications, to date, suggest that the hamster may be a suitable test animal because it displays a greater acute sensitivity to ingested GA than either rats or mice, and it also shows low excretion rates and significant accumulation of GA in various organs[48].

Conclusions

Throughout their long history as a human food source, potatoes have in general proved to be safe. Nevertheless, potatoes and potato products do contain GA that are known toxins. Even though levels of GA have been substantially reduced by breeding, such that outbreaks of acute poisoning are very rare, the possibility of chronic toxicity gives rise to some fundamental uncertainties over the toxic effects of GA in humans. Further work is required to define more clearly:

- the extent and importance of toxicity-enhancing synergistic interactions among GA (and possibly other food components);
- how breeding for new cultivars, as well as physiological stresses, might alter glycoalkaloid profiles and subsequent overall toxicity;
- the metabolism of GA and the kinetics of their long-term accumulation and excretion in humans and/or other appropriate species.

Answers to these questions are essential if earlier calls for a reduction in the acceptable upper limit for GA in potato tubers are to be evaluated or implemented on an informed scientific basis.

Acknowledgement

David Smith gratefully acknowledges receipt of a BBSRC/CCFRA CASE Studentship.

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Bovine spongiform encephalopathy (BSE)

As we go to press, clinical evidence has just been released that may indicate the emergence of cases of a new form of the human neurodegenerative disease CJD. In the absence of any other explanation for the cases, it is speculated that they may be linked to prior consumption of products derived from BSE-infected cattle. The neurodegenerative disease BSE of dairy and beef cattle appears to have developed as a result of feeding ruminant-derived protein to other ruminant farm animals. Readers may be interested in the following Internet sites providing regularly updated information on the topic:

http://dairy.umd.edu/varner/bse.html
(site maintained by Mark Varner, Extension Dairy Scientist at the University of Maryland, USA)

http://inet.uni-c.dk/~iaotb/bse.htm
(site maintained by Torsten Brinch, an 'independent observer' in Lunderskov, Denmark)

http://www.bright.net/~fwo/BSE/bse.html
(site maintained by The Owenlea Farm, Homerville, OH, USA)

http://www.bmj.com//bmj/bse.htm
(site maintained by the *British Medical Journal*)