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## Overview

- All potatoes contain toxic compounds called glycoalkaloids, of which the most well-known is solanine.
- Potatoes accumulate glycoalkaloids primarily to deter pests.
- Glycoalkaloids are usually heavily concentrated in the foliage, but are also present in the tubers.
- A safety limit of 20 mg of total glycoalkaloids per 100 g of potato was established a century ago.
- Most domesticated potatoes contain less than 10 mg / 100 g of glycoalkaloids in tubers.
- Potatoes grown from true seed will have a range of glycoalkaloid concentrations and some may exceed the 20 mg limit, particularly if they include wild potato genetics.
- Most people begin to taste bitterness at concentrations of 11 to 14 mg / 100 g of glycoalkaloids in tubers, but some people are less sensitive to bitter flavors.
- Some heirloom varieties and most wild potatoes can have much higher levels than the safety limit.
- Exposure to sunlight increases glycoalkaloids in potato tubers and turns them green or makes the color darken in red or blue potatoes.
- The color change in light exposed potato tubers is due to increased chlorophyll content and is not directly related to increased glycoalkaloid content, but it is a warning sign that potatoes have been exposed to conditions that could result in increased glycoalkaloids.
- Glycoalkaloids have a bitter or sometimes sour flavor and this is the best indication that tubers are unsafe to eat.
- The most common symptoms of minor glycoalkaloid poisoning are diarrhea and nausea that resolve within a day.
- Very high doses of glycoalkaloids can cause death, but this is very rare.
- Adults are unlikely to ever experience serious potato poisoning, but care should be taken to keep small children from eating foliage, berries, or wild or experimental potato tubers.

***All things are poison, and nothing is without poison, the dosage alone makes it so a thing is not a poison. -Paracelsus***

## Introduction

Potatoes are poisonous. You probably knew that if you are reading this blog, but it would be understandable if you had missed this fact throughout the years that you have been shoveling French fries down your gullet. A quick review of the information that turns up in Google searches on potato toxicity reveals a lot of misinformation but, even moreso, attempts to simplify the subject into few unambiguous rules. If your involvement with potatoes extends beyond just eating them, a deeper understanding will be helpful. The goal of this post is to lay out the facts about potato glycoalkaloids as we know them in a way that is useful to the amateur potato breeder.



Greened potatoes

We actually know surprisingly little about potato glycoalkaloid toxicity. For better or worse, ethical constraints prevent establishing toxic doses and effects through human feeding studies. So, we do animal studies and work backwards from incidents where people have accidentally been poisoned to try to figure out what is safe. This involves a bit of guesswork and nobody can give you an exact measurement of the safe amount of glycoalkaloids that a human can consume. Actually, that isn't quite right. There is always a safe number: zero. But I'm not going to stop eating potatoes and you probably aren't either. In general, modern potatoes have very low levels of glycoalkaloids, Andean potatoes have a slightly wider range, and wild potatoes mostly have very high levels. Potatoes that you grow from seed are much less predictable, particularly if they have recent progenitors in more than one of these groups.

It would be reasonable to ask why we haven't simply tried to breed glycoalkaloids out of potatoes. To some degree, we have. Modern potatoes are lower in glycoalkaloids than some heirloom types. However, there are good features of glycoalkaloids. One of them is flavor. Too much is bitter, but a small amount adds complexity to potato flavor. Another benefit is resistance to certain pests and diseases. Take away all of the glycoalkaloids and we would probably end up spraying more poisons on the crop to compensate. So, we have good incentive to learn to live with the potato's naturally evolved defenses.

Most of the time, when we discuss potato glycoalkaloids, we are interested only in the total glycoalkaloids present in the tuber, not specific glycoalkaloids or levels in other parts of the plant. The term for this is either "total glycoalkaloids" or "tuber glycoalkaloids," both of which are conveniently abbreviated TGA. I'm going to use that acronym a lot from this point on, since typing "glycoalkaloids" gets tedious fast.

# History

Views on the toxicity of potatoes have changed over time. When the potato was initially introduced to Europe, it was not received enthusiastically, in part because of concerns about toxicity. There is no evidence that these concerns were based on experience, but probably stemmed from fear of the unknown, as is often the case with the introduction of new foods. Of course, it is possible that tubers carried to Europe from the New World were in poor condition by the time they arrived. If people had tasted these potatoes rather than using them to grow a fresh crop, they might well have been bitter and toxic. As the potato caught on and became a staple crop, it was consumed in very large amounts. Potato poisoning was an occasional problem where consumption was very high, but the mechanism wasn't understood. Because environmental factors play a role in TGA concentration, the cause was unclear.

In the Andes, where the potato was domesticated, people are more accustomed to dealing with toxic compounds in potatoes. There are several common ways of dealing with toxic potatoes, including geophagy, the practice of consuming potatoes with clay to moderate glycoalkaloid absorption; chuño, a method of processing by freeze drying; and moraya, which involves freezing followed by a long soak in running water and finally drying. These methods are used exclusively with wild and frost-resistant domesticated species such as *Solanum ajanhuiri*, *S. curtilobum*, and *S. juzepczukii*. Presumably, these practices go back to the beginning of the domestication of the potato, when high glycoalkaloid levels would have been ubiquitous.

Potato glycoalkaloids began to be identified in the 1850s, but there was little understanding of their toxicity until the 20th century. According to Sinden (1974), some potato poisoning events were investigated in Germany in the 1920s. At the time, people were consuming as much as 4.4 pounds (2 kg) of potatoes per day. After analyzing the potatoes implicated in these poisonings, it was determined that TGA levels of 26mg/100g of tuber were present. A person consuming 2 kg of such potatoes would accumulate 520 mg of TGA, an amount that we would today consider safe only for a person who weighs at least 573 pounds (260 kg)! This was not a particularly unusual level of consumption. For example, Irish peasants were reported to consume 10 pounds (4.5 kg) of potatoes or more per day prior to the famine. It took a while for understanding of glycoalkaloid toxicity to spread. The only mention in Salaman's 1949 *The History and Social Influence of the Potato* is that an increase in solanine makes potatoes bitter but not harmful.

Based on the findings in Germany, 20 mg TGA per 100g of tubers was suggested as an upper limit for safety. The limit is calculated for fresh weight, but it that converts to about 100mg per 100g of dry weight, given that potatoes have about 80% water content. We still use the same limit today and it is very conservative, given that most modern potato varieties contain no more than half the limit level of glycoalkaloids and people typically don't eat such large portions of potatoes these days. Most people can taste bitterness in potatoes at 14 mg / 100 g, so the majority of cultivars fall below that level under normal circumstances.

## Potato Glycoalkaloids

Potatoes contain a number of different forms of glycoalkaloids. Alkaloids are organic compounds, generally comprised of one or more carbon rings which include or are connected to a nitrogen containing group. Alkaloids are an extremely common sort of compound produced by plants and most of them are

toxic if you consume enough. On the other hand, many of them are very useful and even health enhancing if you don't take too much. For example, caffeine, codeine, and morphine are alkaloids. Glycoalkaloids are a category of alkaloids to which one or more sugars and related groups are attached. The prefix glyco- means sugar, so you can think of glycoalkaloids as sugar alkaloids.

The glycoalkaloids are found throughout the potato plant, but are found in the highest concentrations in sprouts, flowers, immature berries, and foliage. Glycoalkaloids are generally considered to have evolved as a pest defense, so it makes sense that they would be found in higher concentrations in the parts of the plant that are most vulnerable to damage. Many glycoalkaloids deter both vertebrate and invertebrate feeding and some may also act as anti-fungal compounds. Humans are generally much more susceptible to glycoalkaloid toxicity than other animals.

Domesticated potatoes contain two glycoalkaloids of primary interest and several others are present in small quantities or more abundantly in wild potatoes or modern potatoes that have been bred from wild potato species. Glycoalkaloids are derived from steroidal glycosides, so both classes of compound are found in potatoes, although the glycoalkaloids are generally found in larger amounts as the end product.

<b>Steroidal glycosides</b>	<b>Derived glycoalkaloids</b>	<b>Notes</b>
Aceytlleptinidine Leptinidine	Leptine I and II	Uncommon in potato, but leptines are found in some wild species such as <i>S. chacoense</i> . They have been found to deter feeding by the Colorado potato beetle and other insects.
Demissidine	<b>Demissine</b>	A fairly common glycoalkaloid in low amounts in wild potatoes, particularly <i>S. demissum</i> .
Diosgenin	<b>Commersonine</b>	A fairly common glycoalkaloid in low amounts in wild potatoes, particularly <i>S. chacoense</i> and <i>S. commersonii</i> .
Solanidine	<b>Solanine (<math>\alpha</math> and <math>\beta</math> forms)</b>	Primarily in wild potatoes.
	<b>Chaconine (<math>\alpha</math>, <math>\beta_1</math>, <math>\beta_2</math>, <math>\Delta</math>, <math>\gamma</math> forms)</b>	One of the two primary glycoalkaloids of domesticated potatoes and many wild species. Generally found in similar quantities to chaconine.
	Dehydrocommersonine	Primarily in wild potatoes.
Solasodine	Solasonine	Primarily in wild potatoes.
	Solamarine	Primarily in wild potatoes.
Tigogenin		Primarily in wild potatoes.
Tomatidenol	<b>Solamarine (<math>\alpha</math> and <math>\beta</math> forms)</b>	Primarily in wild potatoes.

Tomatidine	Tomatine (α form)	Rarely seen in domesticated potatoes, but is the primary glycoalkaloid in some North American potato species like <i>S. jamesii</i> . It appears that we can not as easily taste differences in tomatine concentration as we can solanine or chaconine (Johns 1986), which may make potatoes that are high in tomatine riskier to work with. Tomatine has been noted as a defense against Colorado potato beetle.
	Sisunine	Primarily in wild potatoes.

Glycoalkaloids that occur in domesticated potatoes are bolded.

Most of this post will focus on solanine and chaconine because the rest are minor players and we really don't know that much about them.

Most modern potato varieties contain less TGA than 10mg/100g, but there are some heirloom varieties that exceed the 20mg/100g safety limit. A study of Norwegian varieties found that some contained high TGA levels, reaching 34.5mg/100g in the variety Prestkvern (Berug 1962); a study of German varieties found a maximum level of 35mg/100g (Maga 2009); and a study of Swedish varieties found that about 9% of early crop varieties exceeded 20mg/kg. A survey of modern American varieties found levels ranging from 1.8 to 13mg/100g (Wolf 1946), but there are many heirloom varieties available in the United States that have never been tested.

While TGA levels are generally pretty low in domesticated potatoes, they can be extremely high in wild potato species. There is almost no risk of confusing domesticated and wild potatoes, since most people have no access to wild potatoes. One possible exception in the United States is the "Four Corners Potato," *Solanum jamesii*. I have read a number of articles about this potato recently that imply that it is edible or at least provide no warning about its glycoalkaloid content. Johns (1990) found TGA levels as high as 128mg/100g for this species (although most samples tested have had much lower amounts), a level more than six times the 20mg/100g safety limit. The primary glycoalkaloid is tomatine, which, as mentioned above, we cannot as easily detect by flavor. If you ate these potatoes despite the bitterness, it would only take about three ounces to put a 70kg person into the danger zone. The native Americans who consumed this potato did so by eating it with clay to adsorb the glycoalkaloids.

## Health Impacts

Potato glycoalkaloids are associated with various effects on the human body, ranging from minor digestive discomfort to respiratory failure. People have died from eating potatoes, although very few in living memory. In modern times, there is not even one death annually on average that is attributed to glycoalkaloid poisoning. In the past, desperation forced people to attempt to eat potatoes that we would never eat today and also in large quantities. During the Irish potato famine, people were forced to eat blighted potato tubers, a very dangerous practice due to the elevated TGA levels produced by blight. The kind of potato poisoning that people experience today is usually very mild and is most often caused by eating too much greened potato. With modern varieties, adults are not likely to suffer much beyond some digestive distress by eating potato tubers, even if they are severely greened. The greater risk occurs with young children eating parts of the potato plant other than the tubers. A young child eating potato sprouts or possibly foliage could quickly get into a serious situation.

As mentioned above, potato breeders generally consider 20mg of TGA per 100g of tuber to be a safe level. This level is considered to be safe based on the average body weight and amount of potatoes

consumed. It would certainly be possible for someone with a low body weight to consume a very large amount of a potato with 20mg/100g TGA and get sick; it just isn't very likely. The safety limit is fairly arbitrary. You are obviously not going to drop dead if you eat a normal serving of a potato with 21mg/100g or even 25 or 30. But, as the level increases, the spread between body mass and quantity consumed decreases, putting more people into the danger zone.

The toxic dose (the dose at which symptoms of poisoning become apparent) for potato glycoalkaloids has been reported to fall between 2mg TGA per kg of body weight to 5mg/kg (Slanina 1990). The safest approach is to take the lowest number, 2 mg/kg. This is an important number, because you can use it to calculate how much TGA you can safely consume (roughly, anyway). If you weigh 75 kg (165 lb), then you can tolerate  $(2 \text{ mg/kg}) * 75 \text{ kg} = 150 \text{ mg TGA}$ . If you are consuming a potato that has 10mg/100g TGA, then you can consume  $150 \text{ mg} * 100 \text{ g} / 10 \text{ mg} = 1500 \text{ g}$  or 1.5 kg. If you are consuming a potato with 30mg/100g, then  $150 \text{ mg} * 100 \text{ g} / 30 \text{ mg} = 500 \text{ grams}$ , still a bit more than a pound. If you are a small person, weighing 100 lb (45 kg), then you have a limit of about 90 mg TGA, so you would only be able to eat 300 grams of that potato, still 2/3 of a pound, which seems like a pretty big portion for a 100 lb person.

I've been focusing here on acute effects of potato toxicity. Many people believe that there may also be chronic effects from lower exposure, producing symptoms like leaky gut syndrome or other immune/allergic reactions. The evidence for this is poor, so I don't have much to say on the subject. It isn't clear, for example, whether conditions like leaky gut syndrome are real or not, much less whether potato glycoalkaloids are involved. Undoubtedly, we will know more in the future, but until there is a good amount of research pointing mostly in one direction, I'm not taking a position.

The following chart shows effects of potato glycoalkaloids descending from the minimum detectable dose to the lethal dose.

<b>Bitterness</b>	The first way that we experience glycoalkaloids is bitter flavor. Some people can detect bitterness when TGA levels reach 11mg/100g (Mondy 1971), whether the glycoalkaloid is solanine, chaconine, or both. The perception of bitterness is roughly proportional to the level of glycoalkaloids, although there is probably a point past which we can no longer tell any difference. Ability to perceive bitterness varies and people with an experienced palate may be able to detect differences more easily.
<b>Burning sensation</b>	As glycoalkaloids increase, the bitterness is accompanied by a sensation of burning or tingling in the mouth and throat. This is a clear warning sign that the variety is high in glycoalkaloids. This is a unmistakable sensation. I am not very sensitive to bitter flavors in potato, but the burning sensation is strong and lasts a long time.
<b>Gastrointestinal distress</b>	Once the amount of glycoalkaloids consumed reaches about 2mg/kg of body weight, gastrointestinal distress follows in about 2 to 20 hours in some people (Slanina 1990). Tolerance varies, so some people will not experience effects until greater amounts are consumed. Diarrhea, nausea, abdominal pain, and vomiting can all occur. This is the worst symptom that most people with glycoalkaloid poisoning experience. Usually it resolves itself within 24 hours, since most of the glycoalkaloids have cleared the body by then. Beyond this point, you'd best get to a hospital.

**Neurological symptoms**

Potato glycoalkaloids inhibit acetylcholinesterase (Mensinga 2005). As the dose continues past 2mg/kg, neurological symptoms including apathy, drowsiness, shaking, confusion, and visual disturbances begin to set in. These can culminate in unconsciousness.

**Respiratory distress**

Rapid, shallow respiration, leading potentially to total respiratory failure.

**Death**

The lethal dose of potato glycoalkaloids hasn't really been established. Reports include concentrations as low as 3mg/kg to 6mg/kg, a range that overlaps with the toxic dose range. There are reasons to think that other factors may have been involved in potato related deaths. The best advice that I can offer here is not to test the limits.

## Factors Influencing TGA Levels

Glycoalkaloid levels are both genetically and environmentally determined. A perfectly safe potato can develop unsafe levels of glycoalkaloids through exposure to light. In response to light, the tubers develop chlorophyll in the skin. Chlorophyll is green, so white and yellow potatoes turn green, while red and blue potatoes become darker in color. Wild potatoes and potatoes with some wild genetics may also undergo an intensification of blue pigments in the skin. This is why we don't eat green potatoes or red or blue potatoes that have darkened. The chlorophyll itself is not poisonous, but serves as an indication that the tubers may also have developed unsafe TGA levels. The amount of greening is not necessarily proportional to the level of TGA. Katahdin is an example of a variety that tends to resist greening even at light levels that increase TGA content. The heirloom variety Magnum Bonum also appears to accumulate high TGAs without greening. It was banned in Sweden after testing found that TGA levels ranged from 6.1 to 66.5 mg/100g with 8% of samples exceeding 40mg/100g (Hellenas 1995). Conversely, some varieties do not develop particularly high TGA levels under greening conditions. In fact, in many cases, green potatoes are perfectly safe to eat, but there is no easy way to determine this short of tasting them for bitterness. The kind of light exposure matters. One study found that fluorescent light was sufficient to cause greening, but not TGA intensification (Gull 1960) and light delivered through a blue, red, or green filter does not result in significant increases in TGA.

Post-harvest storage alone increases TGA levels, sometimes significantly (Berug 1962). Storage temperature factors in as well; Zitnak (1953) found that storage at 4-8 C was associated with higher TGA levels than storage at 12-15 C. Diseases also contribute to increased TGA levels. Late blight particularly is known for this, but any disease that degrades the quality of the tuber should be suspected of increasing TGA levels. Cronk (1974) reported on comparisons in TGA levels between freshly harvested tubers and those that had been stored for 45 days, finding generally about a one-third TGA increase in the stored varieties. It is good to keep this in mind when field-tasting tubers. They are likely to increase in TGA content later. Long day potatoes accumulate more solanine than short day potatoes (Wolf 1946). Presumably, this is mostly a function of the available sunlight during tuberization. Exposure of tubers to sunlight during harvest can be a major contributor to TGA content. Berug (1962) found that TGA content increased minimally with up to four hours of exposure, but then much more significantly after six hours. Don't leave potatoes lying out in the field any longer than you have to.

Glycoalkaloid levels can be reduced through processing and cooking. The majority of the TGA content forms just under the skin, so peeling potatoes can reduce the concentration. Reported levels of reduction range from 30 to 80% (Maga 2009). On the other hand, much of the nutritional value of the

potato is found in the same region, so peeling may not be the best choice as a preventative measure. Boiling peeled potatoes reduces TGA levels further, as does deep frying. Dry cooking methods (baking, roasting, etc.) and steaming typically do not affect TGA levels significantly.

## Testing Seed Grown Potatoes

People have a tendency to worry excessively about the possibility of high glycoalkaloids in seed grown potatoes. It is important to recognize that there is potential for TGA levels that exceed the safety limits, but the potatoes are not going to hold you down and force-feed you. Potatoes with high glycoalkaloids taste bitter. Unless you have one of the rare conditions where you don't register bitter flavors, which you would almost certainly be aware of, you will definitely notice when potatoes are bitter. If you start eating a potato and realize that it is bitter, just stop eating it. You don't need to do anything drastic like taking an emetic! The small amount of bitter potato that you have swallowed is certain to be much too little to cause harm. The progeny of domesticated potatoes are all likely to be below 40 mg / 100 g, which means that you could probably eat a normal portion and still not experience any symptoms of glycoalkaloid poisoning.

TGA content is controlled by multiple genes, so progeny tend to be similar to the parent varieties in this regard, primarily spanning the values between the two parents (Sanford 1972). You aren't likely to find progeny that have a dramatically higher glycoalkaloid content than the parent varieties. If you are making crosses with wild potatoes, then you will want to be a little more careful, but the sensation of bitterness increases with concentration, so you are definitely not going to accidentally eat a large portion of high glycoalkaloid potatoes. I have eaten about 50 species of wild potato tubers, some of which probably had glycoalkaloid concentrations in excess of 300 mg / 100 g. You will not only notice that potatoes with dangerously high concentrations are bitter, but it is an experience that you will not soon forget. I once ate a wild potato so bitter that the sensation persisted for hours. In my experience, high glycoalkaloids in seed grown domesticated potatoes are pretty rare, probably less than 1 in 300 plants, and even then, the bitterness is usually mild.

## The Bitter End

Potatoes are very safe, but if you are pushing the limits with unusual varieties, doing your own breeding, or working with wild potatoes, it will benefit you to understand the information above. If you stick to breeding with domesticated potatoes, varieties with high TGA levels will be rare and probably never too far beyond the safety limit. Your taste buds can tell you a lot about the safety of a variety, but maybe not enough to risk consuming an enormous amount of it the first time you try it or feeding it to young children. Unless you are eating like a 19th century Irish peasant, you are probably not consuming potato glycoalkaloids at levels anywhere near the danger zone.

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