Review Article

Oxalate content of foods and its effect on humans

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Oxalic acid and its salts occur as end products of metabolism in a number of plant tissues. When these plants are eaten they may have an adverse effect because oxalates bind calcium and other minerals. While oxalic acid is a normal end product of mammalian metabolism, the consumption of additional oxalic acid may cause stone formation in the urinary tract when the acid is excreted in the urine. Soaking and cooking of foodstuffs high in oxalate will reduce the oxalate content by leaching. The mean daily intake of oxalate in English diets has been calculated to be 70–150 mg, with tea appearing to contribute the greatest proportion of oxalate in these diets; rhubarb, spinach and beet are other common high oxalate-content foods. Vegetarians who consume greater amounts of vegetables will have a higher intake of oxalates, which may reduce calcium availability. This may be an increased risk factor for women, who require greater amounts of calcium in the diet. In humans, diets low in calcium and high in oxalates are not recommended but the occasional consumption of high oxalate foods as part of a nuritious diet does not pose any particular problem.

Key words: oxalate content, oxalic acid, plants, synthesis, absorption, metabolism, toxic effects.

Introduction

Oxalic acid forms water-soluble salts with Na^+, K^+ and NH_4^+ ions; it also binds with Ca^{2+}, Fe^{2+} and Mg^{2+}, rendering these minerals unavailable to animals. However, Zn^{2+} appears to be relatively unaffected. In plants with a cell sap of approximately pH 2, such as some species of *Oxalis* and *Rumex*, oxalate exists as the acid oxalate ($HC_2O_4^-$), primarily as acid potassium oxalate. In plants with a cell sap of approximately pH 6, such as some plants of the goosefoot family, it exists as the oxalate ion ($C_2O_4^{2-}$), usually as soluble sodium oxalate and the insoluble calcium and magnesium oxalates.[1] Calcium oxalate ($Ca(COO)_2$) is insoluble at a neutral or alkaline pH, but freely dissolves in acid.

Occurrence in plants

Oxalate can be found in relatively small amounts in many plants. Oxalate-rich foods are usually minor components in human diets but are sometimes important in seasonal diets in certain areas of the world. Wood sorrel (*Oxalis cernua*) and halogeton (*Halogeton glomeratus*) are high in oxalates and are known to cause injury to grazing cattle and sheep.[2] The highest levels of oxalates are found in the following families:[2–4] amaranth family, for example *Amaranthus* (amaranth); aroid/arum family, for example *Colocasia* (taro) and *Xanthosoma* (caladium); goosefoot family, for example *Atriplex* (orach), *Beta* (beet, beetroot) and Spinacia (spinach); ice-plant family, for example *Tetragonia* (NZ spinach)*;* wood sorrel family, for example *Oxalis* (sorrel, yam); buckwheat family, for example *Rheum* (rhubarb) and *Rumex* (sorrel); and the purslane family, for example *Portulaca* (purslane).

The oxalic acid content is variable within some species; some cultivars of spinach (Universal, Winter Giant) contain 400 to 600 mg/100 g, while others range from 700 to 900 mg/100 g.[3] Oxalic acid accumulates in plants especially during dry conditions.[5] A study comparing two cultivars of spinach, Magic (summer) and Lead (autumn), revealed that the summer cultivar contained greater amounts of oxalate (740 mg/100 g fresh weight, FW) than the autumn cultivar (560 mg/100 g FW).[6] Reports of some tropical leafy vegetables revealed that dry vegetables had higher oxalate concentrations than did fresh vegetables.[7]

The distribution of oxalate within plants is also uneven. In general, oxalate content is highest in the leaves, followed by the seeds; it is lowest in the stems.[8] Reports show that the stems or stalks of plants, such as amaranth,[5] rhubarb, spinach and beet,[2,9] contain significantly lower levels of oxalates than do the leaves. In the buckwheat family, the content of oxalic acid in leaves is almost twice as great as the content in the stalk. However, in the goosefoot family, oxalic acid is more abundant in the stalk than in the petiole of the leaf. It must be noted that the leaves of rhubarb are rarely eaten and, therefore, the oxalate content of the leaf is of no concern in human nutrition.

Oxalic acid concentration tends to be higher in plants than in meats, which may be considered oxalate-free when planning low oxalate diets.[10] However, a species of snail (*Limicolaria aurora*), used as human food in Nigeria, has been reported to contain 381 mg total oxalate/100 g dry weight (DW).[11] The mollusc, dogwhelk (*Thais cattifera*), contains an even higher level of oxalate, 1686 mg/100 g DW.[12]

Fungi such as *Aspergillus niger*, *Penicillium*, *Mucor*, *Boletus sulphurens* and *Sclerotinia*, can synthesize oxalic acid at a rate of up to 4–5 g/100 g DW in isolated cultivation, in foodstuffs and on the surface of forages.[2,3] The giant mushroom (*Tricholoma giganteum*), a large, edible fungi, is reported to contain 89 mg/100 g DW oxalic acid,[13] but surprisingly, there appear to be no reports on oxalate content of

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commonly consumed mushrooms. Several common edible, tropical species of mushrooms, including termite and ear mushrooms, were reported to contain 80–220 mg oxalate/100 g DW.[14] These levels are relatively low compared with the levels found in spinach and rhubarb.

High oxalate levels in tropical plants are of some concern. Taro (*Colocasia esculenta*) and sweet potato (*Ipomoea batatas*) were reported to contain 278–574 mg/100 g FW[15] and 470 mg/100 g FW, respectively.[16] Total oxalate levels in yam (*Dioscorea alata*) tubers were reported to be in the range of 486–781 mg/100 g DW, although this may not be of nutritional concern given that 50–75% of the oxalates were present in the water-soluble form and therefore may have leached out during cooking.[17] Higher levels of oxalates are usually found in the leaves of these species. Peanut greens, commonly consumed in tropical climates, are reported to contain 407 mg/100 g.[16] The coriander leaf (*Coriandrum sativum*) contains 1268 mg/100 g,[18] while horsegram and santhi (*Boernavia diffusa*) contain 508 mg/100 g[19] and 3800 mg/100g,[20] respectively. Sesame seeds have been reported to contain relatively high quantities of oxalate, ranging from 350 to 1750 mg/100 g FW.[21,22]

Synthesis in plants

The biosynthesis of oxalates in plants has been extensively researched (Figure 1). Oxalate content has been reported to increase as a plant ages and becomes overripe.[23] The proportion of oxalic acid in the leaves of the goosefoot family can double during ripening[24] and occasionally accumulate to such a degree that it makes up more than 15% of the total DW,[25] which suggests that oxalates are an end product of metabolism and act as a 'dump' system.[2] However, it has been suggested that the accumulation of oxalate in plant tissues could be attributed to a shift in equilibrium towards biosynthesis rather than to degradation.[26] It has been reported that oxalate concentration in spinach leaves decreases more with time in cultivars with rapid growth rates than it does in those with slow growth rates, indicating that oxalate may be necessary and used in growth.[27]

Glyoxylate, glycolate and ascorbic acid are the precursors of oxalate in many plants.[25,28–31] Glyoxylate is synthesized from glycolate in the peroxisomes of green spinach leaves. Glycolate was shown to be more efficient as a precursor of oxalate in spinach leaves than was ascorbic acid.[32] Oxalate synthesis appears to be closely related to the glycolate cycle.[33] The presence of glycolic acid oxidase in spinach leaves has been reported. However, the accumulation of oxalate in roots and other non-green tissues which do not have this enzyme suggest that oxalate is transported to these tissues. The presence of glyoxylate oxidase, oxaloacetase and oxalate decarboxylase by which oxalate may be synthesized and degraded, has been confirmed in spinach leaves.[26] In green leaves, glycolate is thought to be derived from a C-5 unit during photosynthesis.[34] Oxalate concentration was reported to be higher in spinach plants exposed to light for 12 h than in plants kept in the dark,[35] thus indicating that a photosynthetic process is involved in oxalate formation. Experiments have indicated that photorespiration contributes to the biosynthesis of oxalate in spinach, and that CO_2 enrichment may lower the rate of oxalate biosynthesis and increase ascorbate content.[32,33]

Oxalates may act as a buffer system in plant tissues. The addition of calcium and nitrates to the growing medium of beet increases the amount of insoluble oxalate in the tissue. Accumulation of oxalate appears to be related to nitrate assimilation and cation–anion imbalance.[2] Another possible role of oxalate is in disease and pest resistance, similar to the function of protease inhibitors and tannins in legume seeds. Calcium oxalate, an important constituent in leaf and corm extracts of elephant foot yam (*Amorphophallus campanulatus*), has been reported to block growth and aflatoxin biosynthesis in *Aspergillus flavus*.[36] Oxalate oxidases, such as germin, are thought to degrade oxalate to produce the hydrogen peroxide required to fuel peroxidase reactions and to cross-link these polymers in the extracellular matrix of higher plants; the details of these reactions have not yet been established.[37]

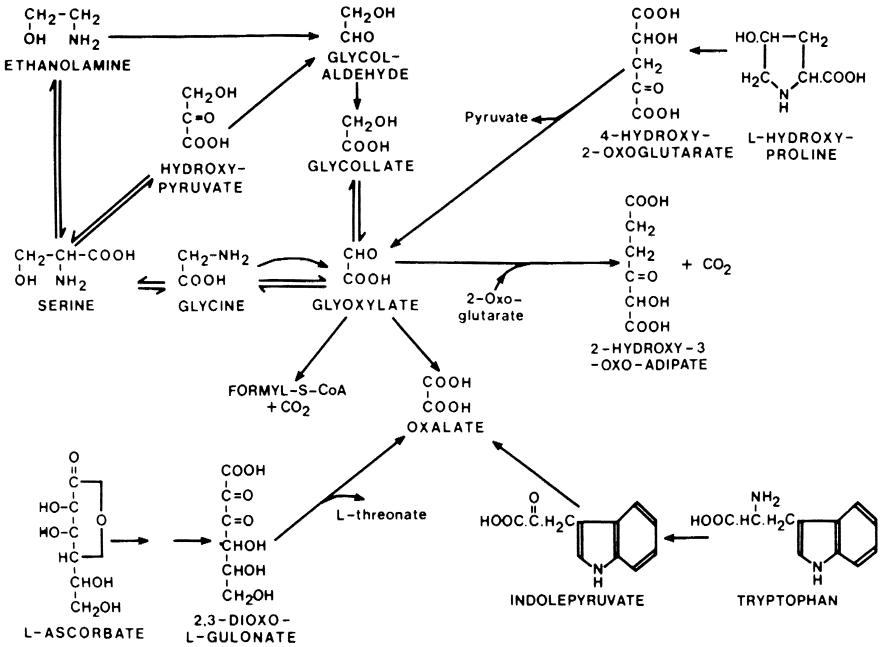

Figure 1. The biosynthesis of oxalic acid (Hodgkinson, 1977).[49] Reprinted from Hodgkinson, Oxalic Acid in Biology and Medicine, 1977, page 181, by permission of the publisher Academic Press Ltd, London.

Absorption and metabolism in mammals

Calcium combines with oxalate to form calcium oxalate in the intestinal lumen, making the calcium unavailable for absorption; calcium oxalate is excreted in the faeces. Free or soluble oxalate is absorbed by passive diffusion in the colon in humans; comparative studies between healthy individuals and those with ileostomies indicate that the colon is the principal site for oxalate absorption.[38–41] However, it has been suggested that the small intestine may be the major absorptive site rather than the colon.[42] The absorption of oxalates from individual foods varies depending on their dietary conditions and source; in general the absorption is relatively limited. It has been estimated that 2–5% of administered oxalate is absorbed in humans.[43,44] Using radiolabelled oxalate, 6.6% of the administered dose was absorbed when consumed with a normal diet, whereas 12% was absorbed when oxalate was consumed during fasting.[45] The percentage of oxalate absorption varied markedly, from 1% for rhubarb and spinach to 22% for tea, but generally absorption was higher at low doses.[46]

Oxalate is an end product of ascorbate, glyoxylate and glycine metabolism in mammals.[37,47,48] Glyoxylate, a key breakdown product of a number of amino acids, is oxidized to oxalate by three enzymes.[49] Urinary oxalate arises principally from endogenous sources with dietary oxalate accounting for only 10–15% of excreted oxalates.[38] Thirty-three to 50% of urinary oxalate is derived from ascorbate, 40% from glycine and 6–33% from minor metabolic pathways and dietary oxalate.[49,50]

Bacterial degradation in the rumen of cattle and sheep may facilitate the safe ingestion of quantities of oxalic acid that would be hazardous for other species. An anaerobic bacterium, *Oxalobacter formigenes* that can metabolize oxalic acid has been found in the rumen, and also in the hindgut of humans.[51] This bacteria degrades oxalate using a decarboxylase enzyme to produce formate, a less toxic acid, and CO_2.[37]

Toxic effects of oxalates

The ingestion of 4–5 g of oxalate is the minimum dose capable of causing death in an adult,[2,3,52] but reports have shown that 10–15 g is the usual amount required to cause fatalities.[3] Oxalic acid ingestion results in corrosion of the mouth and gastrointestinal tract, gastric haemorrhage, renal failure and haematuria.[9] Other associated problems include low plasma calcium, which may cause convulsions, and high plasma oxalates. Most fatalities from oxalate poisoning are apparently due to the removal of calcium ions from the serum by precipitation. High levels of oxalate may interfere with carbohydrate metabolism, particularly by succinic dehydrogenase inhibition;[53] this may be a significant factor in death from oxalate toxicity caused by animals grazing in pastures which contain high levels of *H. glomeratus*.[8]

Although sorrel is a herb and not normally consumed in high quantities, there has been one report of fatal oxalate poisoning after a man consumed an estimated 6–8 g of oxalate in vegetable soup containing 500 g of sorrel. Both fatal and non-fatal poisoning by rhubarb leaves is thought to be caused by toxic anthraquinone glycosides rather than by oxalates[2,54,55] as corrosive gastroenteritis was not observed.[56–58] These anthraquinone derivatives may also occur in the roots and stems of rhubarb or sorrel grass. Experiments involving the consumption by eight women of more than 30–35 g/day of cocoa, a high oxalate foodstuff, provoked symptoms of intoxication including loss of appetite, nausea and headaches. However, cocoa contains theobromine (1500–2500 mg/100 g) and tannic acid (4000–6000 mg/100 g), both of which are more toxic than the oxalic acid present (500–700 mg/100 g).[3] There appears to be a great deal of confusion as to what was responsible for these poisonings and it would be unwise to assume only one factor was the cause.

Effect on bioavailability of minerals

Only some of the minerals present in a food will be absorbed in the gastrointestinal tract due to the presence of dietary fibre, phytates and oxalates. High oxalate foods have been known to exert a negative effect on calcium and iron absorption. Calcium absorption from spinach, a high oxalate and high calcium food, was compared with calcium absorption from milk, a high calcium food, and showed that the calcium from spinach is not readily available, probably due to the high content of oxalates.[59] The adverse effect of oxalates is greater if the oxalate:calcium ratio exceeds 9:4. The adverse effects of oxalates must be considered in terms of the oxalate:calcium ratio in a food. This ratio varies widely and can be classified into three groups:[2,3] (i) plants with an oxalate to calcium ratio greater than two (e.g. spinach, rhubarb, beet leaves and roots, sorrel and cocoa); (ii) plants with a ratio of approximately one (e.g. potatoes, amaranth, gooseberries and currants); and (iii) plants with a ratio of less than one (e.g. lettuce, cabbage, cauliflower, green beans and peas).

Table 1 shows the oxalate and calcium content, as well as the ratio of these two items, in examples of foods that fall into the three main groups.

Foods that have a ratio greater than two and that contain no utilizable calcium have excess oxalate which can bind calcium in other foods eaten at the same time. Rhubarb, sorrel, beet and spinach are not good sources of calcium despite their apparently high levels. Calcium availability from spinach is low at 5.1%.[59] Foodstuffs having a ratio of approximately one do not encroach on the utilization of calcium provided by other products and, therefore, do not exert any demineralizing effects. However, these foods are not good sources of calcium. Foods with a ratio of one do not reduce the availability of calcium as far as other calcium sources are concerned.[9] Although parsley (*Petroselinum sativum*) contains average levels of oxalate (140–200 mg/100 g), its high calcium levels (180–290 mg/100 g) reduce the oxalate:calcium ratio to a low level.[3]

Oxalate appears only to interfere slightly with zinc absorption.[60] However, it was observed that zinc absorption was not affected by oxalic acid added to the diets of zinc-deficient rats.[61] A counteracting or protective mechanism may prevent the precipitation of zinc by oxalates. It has been reported that increasing the proportion of Mg^{2+} in solution inhibits the precipitation of calcium and zinc oxalates.[62] This observation explains the minor effect oxalates have on zinc absorption from some leafy vegetables, such as spinach, which has high levels of calcium and zinc, as well as relatively high levels of magnesium. Surprisingly, few studies on the effect of oxalates on calcium and zinc absorption when

Table 1. Oxalate (mg/100g, fresh weight (FW)), calcium (mg/100g FW) and oxalate/calcium (mEq) ratio of some common foods. These foods have been divided into three main groups on the basis of their oxalate/calcium ratio. Group 1, > 2.0; Group 2, 1.0–2.0; Group 3, < 1.0.[3,21,44]

Foodstuff	Oxalate (mg/100g FW)		Calcium (mg/100g FW)		Oxalate/Ca (mEq)
	Range	Mean	Range	Mean	
Group 1					
Rhubarb (*Rheum rhaponticum*)					
Victoria, forced, stewed	260		12·4		9·32
raw	275–1336	805	40–50	45	7·95
Common sorrel (*Rumex acetosa*)	270–730	500	35–45	40	5·56
Red beetroot (*Beta vulgaris*)	121–450	275	121–450	275	5·09
Garden sorrel (*Rumex patientia*)	300–700	500	40–50	45	4·94
Pig spinach (*Chenopodium* spp.)	1100		99		4·94
Purslane (*Portulaca oleracea*)	910–1679	1294	13–236	125	4·60
Spinach (*Spinacia oleracea*)	320–1260	970	80–122	101	4·27
Garden orach (*Atriplex hortensis*)	300–1500	900	100		4·00
NZ spinach (*Tetragonia expansa*)	890		100		3·96
Coffee (*Coffea arabica*)	50–150	100	10–15	12	3·70
Cashew (*Anacardium occidentale*)	231		41		2·50
Cocoa (*Theobroma cacao*)	500–900	700	100–150	125	2·49
Beet leaves (*Beta vulgaris* var. cicla)	300–920	610	100–120	110	2·46
Rhubarb (*Rheum rhaponticum*)					
Crimson, end of season, stewed	460		91·5		2·23
Group 2					
Potato (*Solanum tuberosom*)	20–141	80	10–34	22	1·62
Amaranth (*Amaranthus polygonoicles*)	1586		595		1·18
Tea (*Thea chinesis*)	300–2000	1150	400–500	450	1·14
Amaranth (*Amaranthus tricolor*)	1087		453		1·07
Rhubarb (*Rheum rhaponticum*)					
Victoria, end of season, stewed	620		266		1·04
Group 3					
Apple (*Malus* spp.)	0–30	15	5–15	10	0·67
Blackcurrant (*Ribes nigrum*)	2–90	50	19–50	35	0·63
Tomato (*Licopersicum esculentum*)	5–35	20	10–20	15	0·58
Parsley (*Petroselinum sativum*)	140–200	170	180–290	235	0·32
Cabbage (*Brassica oleracea*)	0–125	60	200–300	250	0·11
Lettuce (*Lactuca sativa*)	5–20	12	73–90	81	0·07

magnesium is deficient have been reported. Foods rich in oxalate, such as rhubarb, can protect teeth against acid erosion from cola beverages and lemon juice.[63] Therefore, it is necessary to properly investigate the reactions between minerals and oxalic acid.

Oxalic acid may cause greater decreases in mineral availability if consumed with a high fibre diet, although the decrease may only be temporary. Negative calcium, magnesium, zinc and copper balances were detected in males consuming a diet containing fibre and oxalates.[64,65] When spinach was replaced by cauliflower, a low oxalate vegetable, fibre had no effect on the minerals studied, indicating that the apparent negative balances obtained were due to the presence of oxalic acid. Decreased mineral balance may be due to the combined effects of a high fibre intake and oxalic acid, but it may be only a transient response.[66]

Adverse effects

A number of plants contain calcium oxalate crystals. These are not absorbed into the blood stream and remain largely undissolved within the digestive tract. Thus, they have no systemic toxicity but the sharp raphide crystals can penetrate the tissues of the mouth and tongue, causing considerable discomfort. Most of the plants that contain calcium oxalate crystals are members of the arum family.[8] It has been suggested that calcium oxalate crystals are responsible for the irritating sensation in kiwifruit (*Actinidia*).[67]

Oxalate is poorly absorbed under non-fasting conditions. It has been demonstrated that only 2–12% of oxalate is absorbed from foods but that once absorbed, free oxalate binds to calcium ions to form insoluble calcium oxalate. This may also result in a functional hypocalcaemia with tetany in acute cases.[38] Free oxalate and calcium can precipitate in the urine and may form kidney stones. Kidney stones are becoming more common in men between the ages of 30 and 50 years in industrialized countries.[38] These stones are comprised mainly of calcium oxalate (80%), which is relatively insoluble in urine, and calcium phosphate (5%). Oxalate crystallizes with calcium in the renal vasculature and infiltrates vessel walls causing renal tubular obstruction, vascular necrosis and haemorrhage, which leads to anuria, uraemia, electrolyte disturbances or even rupture. The risk factors involved in stone formation are a low volume of urine; an increased urinary excretion of oxalate; calcium or uric acid; a persistently low or high urinary pH; and a low concentration of urinary inhibitors, such as magnesium, citrate and high molecular weight polyanions.[68] Normal urine is usually supersaturated with calcium oxalate.[38] The normal urinary

excretion of oxalate is less than 40–50 mg/day with less than 10% coming from the diet.[38,47] Intakes of oxalate exceeding 180 mg/day lead to a marked increase in the amount excreted.[38] Small increases in oxalate excretion have pronounced effects on the production of calcium oxalate in the urine, implying that foods high in oxalate can promote hyperoxaluria (high oxalate excretion) and increase the risk of stone formation. As well as causing significant increases in urinary oxalate excretion in healthy individuals, rhubarb, spinach, beet, nuts, chocolate, tea, coffee, parsley, celery and wheat bran have been identified as the main dietary sources in the risk of kidney stone formation.[69,70] However, it has been reported that black tea increased oxalate excretion by only 7.9%, compared with increases of 300 and 400% for spinach and rhubarb, respectively.[71] Therefore 2–3 cups/day of black tea would not affect the risk of urinary stone formation. It appears that tea is a significant source of oxalate intake in English diets.[44]

The main reason for the strong relationship between the risk of calcium stones and urinary oxalate excretion appears to be the effect that the latter has on the supersaturation of urine with calcium oxalate.[72] The amount of oxalate excreted in the urine was higher in individuals with stones than in healthy individuals, suggesting that the former group absorbs more oxalate, consumes more oxalate-producing substances such as ascorbate, or metabolises more oxalate precursors.[38] Hyperoxaluria may also be caused by excessive absorption of oxalate from normal diets due to intestinal malfunction.[51] This is termed enteric hyperoxaluria and is the commonest cause of increased renal oxalate excretion. It has been indicated that people with abnormal gastrointestinal absorption might be at greater risk of hyperoxaluria than healthy individuals.[38] Within 2–4 h after ingestion of 600 g of both rhubarb and spinach by normal individuals, urinary oxalate was observed to exceed the normal concentration (0.3 mmol) and crystalluria occurred.[73] The rise in urinary oxalate excretion has been reported to disappear when a dietary oxalate restriction is imposed.[74] A diet low in oxalates has prevented stone formation in some cases involving gastrointestinal disorders associated with hyperoxaluria.[47] For those people with steatorrhoea, calcium binds to free fatty acids in the intestine to form soaps, thus allowing more free oxalate to be absorbed which results in higher urinary oxalate excretion.

A decrease in calcium intake should be accompanied by a lower oxalate consumption, because a low calcium and high oxalate diet enhances oxalate absorption and excretion, which carries an even greater risk of stone formation than high calcium excretion.[38] An increase in calcium intake may reduce urinary oxalate excretion by binding to more oxalate in the gut, thus reducing the risk of stone formation. Varying amounts of calcium did not result in significantly different levels of urinary calcium.[69] It has been suggested that if the population as a whole were to increase its urinary oxalate concentration to the upper limit of normal, most individuals would exceed the formation product of calcium oxalate and form crystals spontaneously in their urine.[72] The increase of urinary concentration of calcium alone, well into the hypercalciuric range, would only expose a small percentage of the population to the risk of crystalluria. From experimental work it was concluded that hypercalciuria plays, at most, a secondary role in the genesis of calcium stones compared with mild hyperoxaluria.[72]

As the majority of oxalate excreted in the urine is reported to be synthesized endogenously from ascorbate, glycolate, glyoxylate and glycine, excessive intake of these substances would not be advised. Excessive ascorbic acid intake may increase urinary levels of oxalate, making it a possible risk factor for kidney stone formation. Ascorbic acid doses greater than 500 mg/day were reported to induce a significant increase in urinary oxalate, and doses of 1000 mg/day would increase urinary oxalate excretion by 6–13 mg/day.[75] Although the recommended daily intake is 80 mg, some individuals have been reported to take at least 500 mg/day regularly. These doses will significantly increase the intrarenal urinary oxalate concentration and the risk of calcium oxalate stone formation.[75] The potential for the ingestion of large quantities of ascorbic acid as a cold preventative to produce undesirably large increases of urinary oxalate needs to be considered.

Effects of processing

Oxalates may be removed from food by leaching in water, although this is not the most effective method.[9] Although the amount of oxalate in raw soybean (*Glycine max*) is relatively low, soaking and germination of the seed reduced the oxalate concentration.[76] Cooking germinated soybeans reduced their oxalate concentration to below that of uncooked germinated soybeans. Soaking followed by cooking also proved to be effective, although not as effective as germination. Oxalate content in horsegram seeds (*Macrotyloma uniflorum*) decreased by 38% when dehulled (508 and 315 mg/100 g, for seed and dehulled seed, respectively).[19] Roasting was found to be the least effective method. The roasting of chicory roots was reported to increase the oxalate content.[77]

A 40–50% loss of total oxalates by leaching was reported when yam tubers (*Dioscorea alata* and *D. esculenta*) were boiled compared with being steamed (20–25%) and baked (12–15%).[78] Although cooking proved most effective in terms of the reduction of total oxalates, water-soluble minerals also leached out at the same time. Blanching has been reported to decrease the oxalic acid content in spinach.[79,80] However, blanching, by conventional and microwave methods, made only small reductions in the oxalic acid content of sweet potato, peanut and collard leaves, whereas other antinutritional factors such as tannic and phytic acid were reduced significantly.[16] A 72.4% decrease in total oxalate concentration was observed after cooking conophor seeds (*Tetracarpidium conophorum*), a popular Nigerian snack, which have a bitter taste when raw but are palatable when cooked.[81] Conophor seeds are rich in potassium and also contain significant amounts of phosphorus, calcium, magnesium, iron and zinc. Cooking caused a 33.3, 28.6 and 60.0% decrease in sodium, iron and copper, respectively, but other minerals were little affected. Mineral leaching appears to vary between plant species. Spinach, orach and silverbeet are generally eaten after being boiled. However, rhubarb, cocoa and common and garden sorrel may be consumed in the raw state and, therefore, should be eaten in smaller quantities.

Fermentation, frequently used in Asian countries, has been reported to decrease the oxalate content of foods. A marked decrease in oxalic acid content was reported in

Icacinia manni (a starch tuber) upon fermentation.[82] It was observed that oxalic acid decreased by 37% (86–54 mg/100 g FW) during souring of poi (a cooked taro paste) at 20°C.[83]

Conclusions

Foods high in oxalates should be consumed in moderation to ensure optimum intake of minerals from the diet. Although some foods are reported to be high in calcium and other essential minerals, the amount available may be limited due to the presence of oxalates. For instance, spinach is a high calcium food (93–111 mg/100 g FW), yet due to its high oxalate content (779 mg/100 g FW) the calcium availability is almost negligible.[49] The availability of magnesium, iron, sodium, potassium and phosphorus may also be restricted. It is recommended that high oxalate foods be accompanied by calcium-rich foods such as dairy products and shellfish.[54,55]

High oxalate foods should be cooked to reduce the oxalate content. Soaking raw foods will also reduce the oxalate content but other useful nutrients such as vitamin C may also be lost at the same time. Oxalates tend to occur in higher concentrations in the leafy parts of vegetables rather than in the roots or stalks.

Vegans, vegetarians, women or inhabitants of tropical countries should be aware that some foods contain high levels of oxalates. The diets of vegans and those with lactose intolerance may be low in calcium due to the exclusion of dairy products, unless supplemented by some other high calcium food products. If high oxalate foods were to be consumed in conjunction with a low calcium diet, then the consumer may be at risk of hyperoxaluria and stone formation. It appears that leafy tropical plants tend to contain higher levels of oxalates than plants from temperate climates. People living in these areas are at possible risk of stone formation due to hyperoxaluria, and mineral deficiencies if sufficient minerals are not consumed.

Women tend to be more susceptible than men to calcium and iron deficiencies, while osteoporosis is of concern to females, especially after menopause. Therefore, women should eat red meats, which are low in oxalate, rather than vegetables, which can be high in oxalates, to satisfy their iron intake. The risk of stone formation is three times greater in males and thus, they should avoid eating excess amounts of high oxalate foods. Sufferers of hyperoxaluria and kidney stones are also advised to restrict their diet to low-oxalate foods because although urinary oxalate arises predominantly from endogenous sources, it can be influenced by dietary intake. People suffering from fractures should also be aware of the potential effects of oxalates on mineral availability, given that high calcium is required for bone repair.

The occasional consumption of high oxalate foods as part of a mixed diet does not pose health problems. Problems are more likely to occur in people with diets of little variety.

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草酸及其盐类是一些植物的代谢最终产物。由於草酸盐可與鈣及其它矿物質結合，食入此類植物對動物体会造成不良影响。縱使草酸是哺乳動物体内一种正常的代謝物，攝取過多仍會造成輸尿管結石。含高草酸盐食品经过浸泡煮熟後，特由於淬取过沪作用減少其含量。在傳統英國風味食物中，平均一天草酸盐攝取約70至150毫克，其中菜葉佔最大比例，其次大黃，菠菜及甜菜也佔有相當比例。素食者由於攝取大量蔬菜，也因此食入大量草酸盐，最終造成体内鈣质減少。在婦女同胞，由於她们需大量鈣质，鈣质缺乏或減少对其是一种危机。雖然低鈣高草酸食品並不為人們所推薦，但偶爾攝取高草酸盐食物作為調節食品並不會造成問題。

Oxalate content of foods and its effect on humans

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食品中草酸鹽含量對人體新陳代謝的影響

摘要

草酸及其鹽類是一些植物的最終代謝產物。由于草酸鹽可與鈣及其它礦物質結合盡管草酸是哺乳動物體内的正常代謝物質，攝入此類植物過多會給動物帶來不良的影響如輸尿管結石。含高草酸鹽的食品經過浸泡煮熟后，通過萃取過濾減少其含量。在傳統英國風味食品中，每日平均草酸鹽的攝入量是70至150毫克。其中茶葉占的比例最大，其次爲大黄，菠菜和甜菜。素食者由于食入大量蔬菜。因此攝入大量的草酸鹽，最終導致體内鈣質減少。婦女對鈣質的需求量較大，體内鈣質的減少是婦女健康的一大危機。雖然低鈣高草酸食品并不爲人們所推薦，偶爾攝入高草酸鹽食物作爲調節食品不會出現不良的影響。

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ออกซาเลตในอาหารและผลกระทบของออกซาเลตต่อมนุษย์

กรดและเกลือของกรดออกซาลิก เป็นผลิตผลสุดท้ายที่เกิดขึ้นจากขบวนการเผาผลาญในเนื้อเยื่อของพืชหลายชนิด การบริโภคพืชเหล่านี้อาจจะให้ผลเสียกับร่างกาย เพราะเกลือออกซาเลตสามารถรวมตัวกันแคลเซียมและแร่ธาตุอื่นๆได้ กรดออกซาลิกเป็นผลิตผลสุดท้ายของขบวนการเผาผลาญซึ่งเกิดขึ้นโดยปกติในสัตว์เลี้ยงลูกด้วยนมทั่วไป การบริโภคกรดออกซาลิกเพิ่มขึ้นอาจเป็นสาเหตุทางก่อนำในทางเดินปัสสาวะ เมื่อกรดออกซาลิกถูกขับออกมาทางปัสสาวะ การแช่และการปรุงอาหารจะลดปริมาณออกซาเลตโดยการถูกชะล้างไปกับน้ำ จากการศึกษาอาหารที่ชาวอังกฤษบริโภคต่อวันโดยเฉลี่ยมีออกซาเลต 70-150 มก. โดยอาหารมีปริมาณสัดส่วนสารนี้สูงสุดในตาน เหล่านั้น รูบาร์บ (Rhubarb), ผักขม (Spinach) และหัวบีท (Beet) เป็นพืชที่พบโดยทั่วไปที่มีปริมาณออกซาเลตสูง ผู้บริโภคอาหารมังสวิรัติซึ่งบริโภคผักมากจะได้รับออกซาเลตในปริมาณสูงกว่าคนโดยทั่วไป ซึ่งอาจจะลดความเป็นประโยชน์ของแคลเซียม สิ่งนี้ก่อให้เกิดความเสี่ยงสำหรับผู้หญิงซึ่งมีความต้องการแคลเซียมในปริมาณสูง ไม่แนะนำให้บริโภคอาหารที่มีแคลเซียมต่ำและมีออกซาเลตสูง แต่การบริโภคอาหารที่มีออกซาเลตสูงเป็นส่วนประกอบหนึ่งของอาหารสมดุลย์เป็นครั้งคราว ไม่ก่อให้เกิดปัญหาใดๆ

References

1. James LF. Oxalate poisoning in livestock. In: Keeler RF, van Kampen, James LF, eds. Effects of poisonous plants on livestock. New York: Academic Press, 1978: 139–145.
2. Fassett DW. Oxalates. In: Toxicants occurring naturally in foods, 2nd edn. Washington: National Academy of Sciences, 1973: 346–362.
3. Gontzea I, Sutzescu P. Natural antinutritive substances in foodstuffs and forages. Basel: S Karger, 1968.
4. Linder MC. Nutritional biochemistry and metabolism with clinical applications, 2nd edn. New York: Elsevier, 1991.
5. Bressani R. Amaranth. In: Macrae R, Robinson RK, Sadler MJ, eds. Encyclopaedia of food science, food technology and nutrition. London: Academic Press, 1993: 135–140.
6. Watanabe Y, Uchiyama F, Yoshida K. Compositional changes in spinach (*Spinacia oleracea* L.) grown in the summer and the fall. J Jap Soc Hort Sci 1994; 62: 889–895.
7. Aletor VA, Adeogun OA. Nutrient and anti-nutrient components of some tropical leafy vegetables. Food Chemistry 1995; 53: 375–379.
8. Osweiler GD, Carson TL, Buck WB, Gelder GA. Clinical and diagnostic veterinary toxicology, 3rd edn. Iowa: Kendall/Hunt Dubuque, 1985.
9. Concon JM. Food toxicology – principles and concepts. New York: Marcel Dekker, 1988.
10. Massey LK, Roman-Smith H, Sutton RAL. Effect of dietary oxalate and calcium on urinary oxalate and risk of formation of calcium oxalate kidney stones. J Am Diet Assoc 1993; 93: 901–906.
11. Udoh AP, Akpanyung EO, Igiran IE. Nutrients and anti-nutrients in small snails (*Limicolaria aurora*). Food Chemistry 1995; 53: 239–241.
12. Udoh AP, Effiong RI, Edem DO. Nutrient composition of dogwhelk (*Thais cattifera*), a protein source for humans. Trop Sci 1995; 35: 64–67.
13. Fujita T, Komemushi S, Yamagata K. Contents of amino acids, organic acids and 5′-nucleotides in *Tricholoma giganteum*. J Sci Food Agric 1991; 55: 159–162.
14. Aletor VA. Compositional studies on edible tropical species of mushrooms. Food Chemistry 1995; 54: 265–268.
15. Holloway WD, Argall ME, Jealous WT, Lee JA, Bradbury JH. Organic acids and calcium oxalate in tropical root crops. J Agric Food Chem 1989; 37: 337–341.
16. Mosha TC, Gaga HE, Pace RD, Laswai HS, Mtebe K. Effect of blanching on the content of antinutritional factors in selected vegetables. Plant Foods Hum Nutr 1995; 47: 361–367.
17. Wanasundera JPD, Ravindran G. Nutritional assessment of yam (*Dioscorea alata*) tubers. Plant Foods Hum Nutr 1994; 46: 33–39.
18. Anantha Samy TS, Kamat VN, Pandya HG. Oxalate content of leafy vegetables. Current Sci (Bangalore) 1960; 29: 133.
19. Sudha N, Mushtari Begum J, Shambulingappa KG, Babu CK. Nutrients and some anti-nutrients in horsegram (*Macrotyloma uniflorum* (Lam.) Verdc.). Food Nutrition Bull 1995; 16: 81–83.
20. Duhan A, Chauhan BM, Punia D. Nutritional value of some nonconventional plant foods of India. Plant Foods Hum Nutr 1992; 42: 193–200.
21. Meena BA, Umapathy KP, Pankaja N, Prakash J. Soluble and insoluble oxalates in selected foods. J Food Sci Technol 1987; 24: 43–44.
22. Ishi Y, Takiyama K. Extraction of calcium, oxalate and calcium oxalate crystals from sesame seeds (in Japanese with English abstract). Bunseki Kagaku 1994; 43: 151–155.
23. Yoshikawa T, Nakagawa K, Kobayashi T, Tokieda S, Nagai S. Studies on high quality production and shipment of spinach I: Effects of varieties and growth stage on oxalic acid content (in Japanese with English abstract). Kinki Chogoku Agric Res 1988; 75: 71–76.
24. Grütz W. Beziehungen zwischen Nährstoffversorgung und Oxalsäurebildung in der Pflanze. Landwirtschafthche Forsch 1956; 8: 121–135.
25. Chang C, Beevers H. Biogenesis of oxalate in plant tissues. Plant Physiol 1968; 43: 1821–1828.
26. Hitomi E, Tamaki Y, Tomoyeda M. Biogenesis and degradation of oxalate in spinach. J Jap Soc Hort Sci 1992; 61: 431–435.
27. Hirooka M, Sugiyama N. Effect of growth rates on oxalate concentrations in spinach leaves. J Jap Soc Hort Sci 1992; 61: 575–579.
28. Seal SN, Sen SP. The photosynthetic production of oxalic acid in *Oxalis corniculata*. Plant Cell Physiol 1970; 11: 119–128.
29. Yang JC, Loewus FA. Metabolic conversion of L-ascorbic acid to oxalic acid in oxalate-accumulating plants. Plant Physiol 1975; 56: 283–285.
30. Williams M, Saito K, Loewus FA. Ascorbic acid metabolism in geranium and grape. Phytochemistry 1979; 18: 953–956.
31. Chang C, Huang AHC. Metabolism of glycolate in isolated spinach leaf peroxisomes: kinetics of glyoxylate, oxalate, carbon dioxide, and glycine formation. Plant Physiol 1981; 67: 1003–1006.
32. Fujii N, Watanabe M, Watanabe Y, Shimada N. Rate of oxalate biosynthesis from glycolate and ascorbic acid in spinach leaves. Soil Sci Plant Nutrition 1993; 39: 627–634.
33. Fujii N, Watanabe M, Watanabe Y, Shimada N. Relationship between oxalate synthesis and glycolate cycle in spinach. J Jap Soc Hort Sci 1994; 62: 789–794.
34. Beevers H, Stiller ML, Butt VS. Metabolism of organic acids. In: Steward FC, ed. Plant physiology. New York: Academic Press, 1966, Vol. IVB: 119–262.
35. Kitchen JW, Burns EE, Langston R. The effects of light, temperature and ionic balance on oxalate formation in spinach. Proc Am Soc Hort Sci 1964; 85: 465–470.
36. Prasad G, Sahay SS, Masood A. Inhibition in aflatoxin biosynthesis by the extract of *Amorphophallus campanulatus* (OL) and calcium oxalate. Lett Appl Microbiol 1994; 18: 203–205.
37. Lane BG. Oxalate, germin, and the extracellular matrix of higher plants. FASEB J 1994; 8: 294–301.
38. Hughes J, Norman RW. Diet and calcium stones. Can Med Assoc J 1992; 146: 137–143.
39. Dobbins JW, Binder HJ. Importance of the colon in enteric hyperoxaluria. N Engl J Med 1977; 296: 298–301.
40. Modigliani R, Labayle D, Aymes C, Denvil R. Evidence for excessive absorption of oxalate by the colon in enteric hyperoxaluria. Scand J Gastroenterol 1978; 13: 187–192.
41. Hofmann AF, Laker MF, Dharmsathaphorn K, Sherr HP, Lorenzo D. Complex pathogenesis of hyperoxaluria after jejunoileal bypass surgery (oxalogenic substances in diet contribute to urinary oxalate). Gastroenterology 1983; 84: 293–300.
42. Prenen JAC, Boer P, Mees MJD. Absorption kinetics of oxalate from oxalate-rich food in man. Am J Clin Nutr 1984; 40: 1007–1010.
43. Archer HE, Dormer AE, Scowen EF, Watts RWE. Studies on the urinary excretion of oxalate by normal subjects. Clin Sci 1957; 16: 405–411.
44. Zarembski PM, Hodgkinson A. The oxalic acid content of English diets. Br J Nutrition 1962; 16: 627–634.
45. Earnest DL, Williams HE, Admirand WH. A physico-chemical basis for treatment of enteric hyperoxaluria. Trans Assoc Am Physicians 1975; 88: 224–234.
46. Finch AM, Kasidas GP, Rose GA. Urine composition in normal subjects after oral ingestion of oxalate-rich foods. Clin Sci 1981; 60: 411–418.
47. Smith LH, Van den Berg CJ, Wilson DM. Current concepts in nutrition – nutrition and urolithiasis. N Eng J Med 1978; 298: 87–89.
48. Hanson CF, Frankos VH, Thompson WO. Bioavailability of oxalic acid from spinach, sugar beet fibre and a solution of sodium oxalate consumed by female volunteers. Food Chem Toxicol 1989; 27: 181–184.
49. Hodgkinson A. Oxalic acid in biology and medicine. New York: Academic Press, 1977.
50. Hagler L, Herman RH. Oxalate metabolism I. Am J Clin Nutr 1973; 26: 758–765.
51. Allison MJ, Cook HM, Milne DB, Gallagher S, Clayman RV. Oxalate degradation by gastrointestinal bacteria from humans. J Nutr 1986; 116: 455–460.
52. Figdor PP. Uremia as a symptom of oxalic acid poisoning (in German with English abstract). Wien Med Wochenschr 1961; 111: 111–114.
53. James LF. Serum electrolyte, acid-base balance, and enzyme changes in acute Halogeton glomeratus poisoning. Can J Comp Med 1968; 32: 539–543.

54. Schmid W. Assay of anthraglycoside drugs. Use of the leaves of medicinal and edible rhubarb (in German with English abstract). Deutsche Apotheker Zeitung (Stuttgart) 1951; 91: 452–454.
55. von Streicher E. Acute kidney failure and ecterus following poisoning with rhubarb leaves (in German with English abstract). Dtsch Med Wochenschr 1964; 89: 2379.
56. Robb EF. Death from rhubarb leaves due to oxalic acid poisoning. JAMA 1919; 73: 627–628.
57. Tallqvist HV, Väänänen I. Death of a child from oxalic acid poisoning due to eating rhubarb leaves. Annales Paediatriae Fenniae 1960; 6: 144–147.
58. Kalliala H, Kauste O. Ingestion of rhubarb leaves as cause of oxalic acid poisoning. Annales Paediatriae Fenniae 1964; 10: 228–231.
59. Heaney RP, Weaver CM, Recker RR. Calcium absorbability from spinach. Am J Clin Nutr 1988; 47: 707–709.
60. Sollmann TH. A manual of pharmacology and its applications to therapeutics and toxicology, 8th edn. Philadelphia: Saunders 1957.
61. Welch RM, House WA, Van Campen D. Effects of oxalic acid on availability of zinc from spinach leaves and zinc sulfate to rats. J Nutr 1977; 107: 929–933.
62. Faboya OO. The interaction between oxalic acid and divalent ions — Mg^{2+}, Zn^{2+} and Ca^{2+} — in aqueous medium. Food Chemistry 1990; 38: 179–187.
63. Pierce EC, Appleman CO. Role of ether soluble organic acids in the cation-anion balance of plants. Plant Physiol 1943; 18: 224–238.
64. Kelsay JL, Behall KM, Prather ES. Effect of fiber from fruits and vegetables on metabolic responses of human subjects II. Calcium, magnesium, iron and silicon balances. Am J Clin Nutr 1979; 32: 1876–1880.
65. Kelsay JL, Jacob RA, Prather ES. Effect of fiber from fruits and vegetables on metabolic responses of human subjects III. Zinc, copper and phosphorus balances. Am J Clin Nutr 1979; 32: 2307–2311.
66. Kelsay JL. Effects of fiber, phytic acid, and oxalic acid in the diet on mineral availability. Am J Gastroenterol 1987; 82: 983–986.
67. Perera CO, Hallett IC, Nguyen TT, Charles JC. Calcium oxalate crystals: the irritant factor in kiwifruit. Food Science 1990; 55: 1066–1069.
68. Goldwasser B, Wienerth JL, Carson CC. Calcium stone disease: an overview. J Urol 1986; 135: 1–7.
69. Massey LK, Sutton RAL. Modification of dietary oxalate and calcium reduces urinary oxalate in hyperoxaluric patients with kidney stones. J Am Diet Assoc 1993; 93: 1305–1307.
70. Parivar F, Low RK, Stroller ML. The influence of diet on urinary stone disease. J Urol 1996; 155: 432–440.
71. Hesse A, Siener R, Heynck H, Jahnen A. The influence of dietary factors on the risk of urinary stone formation. Scanning Microsc 1993; 7: 1119–1128.
72. Robertson WG, Peacock M. The cause of idiopathic calcium stone disease: Hypercalciuria or hyperoxaluria? Nephron 1980; 26: 105–110.
73. Strenge A, Hesse A, Bach D, Vahlensieck W. Excretion of oxalic acid following the ingestion of various amounts of oxalic acid-rich foods. In: Urolithiasis. New York: Plenum Press, 1981: 789–794.
74. Marshall RW, Cochran M, Hodgkinson A. Relationships between calcium and oxalic acid intake in the diet and their excretion in the urine of normal and renal stone-forming subjects. Clinical Science 1972; 43: 91–99.
75. Urivetzky M, Kessaris D, Smith AD. Ascorbic acid overdosing: A risk factor for calcium oxalate nephrolithiasis. J Urol 1991; 147: 1215–1218.
76. Ologhobo AD. Improving the nutritive value of soyabean (*Glycine max* (L.) Merr.) through processing: Biochemical and protein quality studies. Tropical Agriculture (Trinidad) 1989; 66: 290–296.
77. Barlianto H, Maier HG. Acids in chicory roots and malt. Z Lebensm Unters Forsch 1995; 200: 273–277.
78. Wanasundera JPD, Ravindran G. Effects of cooking on the nutrient and antinutrient contents of yam tubers (*Dioscorea alata* and *Dioscorea esculenta*). Food Chemistry 1992; 45: 247–250.
79. Na Young Kim, Shuk Ja Joon, Myung Sook Jang. Effect of blanching on the chemical properties of different kinds of spinach. J Korean Soc Food Sci 1993; 9: 204–209.
80. Sam Soo Park, Myung Sook Jang, Kyu Han Lee. Effect of blanching condition on the chemical composition of the spinach grown in winter greenhouse. J Korean Soc Food Nutr 1994; 23: 62–67.
81. Edem DO, Ekwere ES, Eka OU. Chemical evaluation of the effects of cooking on the nutritive value of conophor seed (*Tetracarpidium conophorum*). Tropical Science 1994; 34: 377–380.
82. Antai SP, Obong US. The effect of fermentation on the nutrient status and on some toxic components of *Icacinia manni*. Plant Foods Hum Nutr 1992; 42: 219–224.
83. Huang AS, Lam SY, Nakayama TM, Hui Lin. Microbiological chemical changes in poi stored at 20°C. J Agric Food Chem 1994; 42: 45–48.