chapter three

Antinutritives

M.M.T. Janssen

3.1 Introduction

It is a well-known fact that food components can also cause toxic effects without being the active agents themselves. The substances in question are known as antinutritives. They induce their toxic effects indirectly, by causing nutritional deficiencies or by interference with the functioning and utilization of nutrients.

Antinutritives can interfere with food components before intake, during digestion in the gastrointestinal tract, and after absorption in the body. The adverse effects of antinutritives usually do not manifest themselves as readily as those of the directly acting toxic food components. The conditions under which antinutritives may have important implications are malnutrition or a marginal nutritional state.

Antinutritives can be of natural as well as synthetic origin. They can be classified as follows:

- type A: substances primarily interfering with the digestion of proteins or the absorption and utilization of amino acids; these are also called *antiproteins;*
- type B: substances interfering with the absorption or metabolic utilization of minerals *(antiminerals);*
- type C: substances that inactivate or destroy vitamins or otherwise increase the need for vitamins *(antivitamins).*

Antinutritives are mainly found in plant material. In a number of cases, drugs, antibiotics, and pesticides have been reported to be antinutritive. This chapter deals with food components of natural origin only.

3.2 Type A antinutritives (antiproteins)

Especially people depending on vegetables for their protein supply are in danger of impairment by antiproteins. This is often the case in less developed countries.

3.2.1 Protease inhibitors

Protease inhibitors are proteins which inhibit proteolytic enzymes by binding to the active sites of the enzymes. Their specificities for the different proteases are broadly overlapping. This category of antinutritives occurs in many plants, and in a few animal tissues.

Proteolytic enzyme inhibitors were first found in eggs around the turn of the century. They were later identified as ovomucoid and ovoinhibitor, both of which inactivate trypsin. Also, chymotrypsin inhibitors are found in eggs, especially in the egg white. Other foods in which trypsin and/or chymotrypsin inhibitors are found are legumes (e.g., soybeans), vegetables (e.g., alfalfa), milk, wheat and potato.

The protease inhibitors in soybeans, kidney beans and potatoes also inhibit elastase, a pancreatic enzyme acting on elastin, an insoluble protein in meat. Since protease inhibitors are proteins, they can be expected to be heat labile. Protease inhibitors that are indeed heat labile are particularly sensitive to moist heat, whereas dry heat is less effective. Autoclaving soybeans for 20 min at 115°C or 40 min at 107 to 108°C is necessary for maximum destruction of its inhibitors. Prior soaking in water for 12 to 24 hr makes the heat treatment more effective. Boiling at 100°C for 15 to 30 min is sufficient to improve the nutritional value of soaked soybeans.

However, several protease inhibitors are relatively heat resistant. An example is the trypsin inhibitor in milk. In raw milk, the activity of trypsin can be reduced by 75 to 99%. The inhibitor is unaffected by temperatures up to 70°C. Pasteurization for 40 sec at 72°C destroys only 3 to 4%, heating at 85°C for 3 sec, 44 to 55%, and heating at 95°C for 1 hr 73% of the inhibitor. Other relatively heat-resistant protease inhibitors are the trypsin inhibitor in alfalfa, the chymotrypsin inhibitor in potatoes, and the trypsin inhibitor in lima beans.

3.2.2 Lectins

Lectins is the general term for plant proteins that have highly specific binding sites for carbohydrates. The majority of the lectins are glycoproteins. A carbohydrate-free lectin occurs in jack beans (concanavalin A). The lectin in kidney beans is probably a lipoprotein. The mode of action of lectins may be related to their ability to bind to specific cell receptors in a way comparable to that of antibodies. They can agglutinate red blood cells. Therefore, they are also called hemagglutinins.

This section discusses the origin of lectins in relation to their interference with the absorption of amino acids, fats, vitamins, and thyroxine. It will become clear that the lectins in legumes not only belong to type A antinutritives but also to types B and C.

Lectins occur in plants, especially legumes such as peanut, soybean, lima, kidney, mung, jack, hyacinth, castor and fava bean, and lentil and pea. They are also found in potato, banana, mango, and wheat germ.

Lectins can contribute to a large extent to the protein content of plants. Bean lectins can disturb the absorption of nutrients and other essential substances from the intestines. *In vitro* studies have shown that bean lectins bind to the rat intestinal mucosal cells. In experimental animals fed on a raw soybean diet, the absorption of amino acids, thyroxine, and fats decreased whereas the requirement for the lipophilic vitamins A and D increased significantly. Interference with the absorption of thyroxine could explain the goitrogenic effect of soybeans. However, this effect may also be caused by interference with the absorption of iodine, as iodine supplementation to the diet has a positive effect on soybean goiter.

Among the edible pulses the kidney bean and the hyacinth bean are highly antinutritive. However, one of the most toxic plants is the castor bean. The toxin is ricin which causes intestinal cell necrosis.

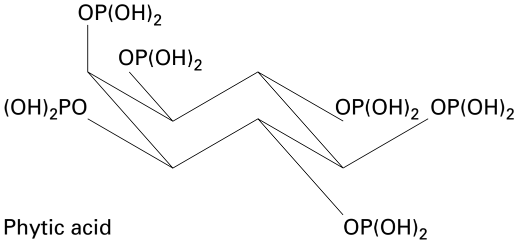
The lectins, being proteins, can easily be inactivated by moist heat. Dry heat is ineffective. The hemagglutinin activity of several pea varieties and bean species decreases on germination. Soybeans can lose as much as 92% of their lectin activity during the first day of germination.

3.3 *Type B antinutritives (antiminerals)*

Substances interfering with the utilization of essential minerals are widely distributed among vegetables, fruits, and cereal grains. The levels of antiminerals in foods seldom cause acute effects if the diet is well-balanced.

3.3.1 *Phytic acid*

Phytic acid, the hexaphosphoric ester of myo-inositol, is a strong acid. It forms insoluble salts with many types of bivalent and tervalent heavy metal ions. In that way, phytic acid reduces the availability of many minerals and essential trace elements.



Phytic acid

Phytic acid has been shown to have a negative effect on iron absorption in humans. The absorption of iron depends mainly on the levels in the iron pools, the amount and the chemical form ingested, and the presence of ascorbic acid.

Ferric phytate is least soluble in diluted acid, i.e., it is insoluble in the stomach. At the pH in the duodenum, ferric phytate dissolves in the form of ferric hydroxide.

Phytic acid prevents the complexation between iron and gastroferrium, an iron-binding protein secreted in the stomach. Results from animal experiments and human studies indicate interference of phytic acid with the absorption of magnesium, zinc, copper, and manganese.

An important factor in the precipitation of phytates is the synergistic effect of two or more different cations, which can act together to increase the quantity of phytate that precipitates. For instance, zinc-calcium phytate precipitates maximally at pH 6, which is also the pH of the duodenum, where mainly calcium and trace metals are absorbed. An element makes its deficiency felt as soon as it becomes limiting due to binding to phytic acid.

The results of chemical analysis of the mineral and trace element contents of diets may give a false impression because of the interaction of phytic acid with the elements concerned at the levels of exposure or absorption. The highest levels of phytate occur in grains and some legumes. Generally, grains make up the bulk of the diet especially in developing countries where the diet is often deficient. Therefore, the presence of phytates in grains is a cause for concern. The phytate contents of several foods are listed in Table 3.1.

The phytate of plant seeds is located primarily in the bran and germ. Therefore, brown and wholemeal bread contain more phytate than white bread. In human studies, a diet of

Table 3.1 Phytate contents of selected foods

Food	mg%
Grains	
Wheat	170–280
Rye	247
Maize	146–353
Rice	157–240
Barley	70–300
Oats	208–355
Sorghum	206–280
Buckwheat	322
Millet	83
Wheat bran	1170–1439
Legumes and vegetables	
Green bean (*Phaseolus vulgaris*)	52
Bean (*Phaseolus vulgaris*)	269
Bean (*Phaseolus lunatus*)	152
Soybean	402
Lentil	295
Green pea (*Pisum sativum*)	12
Pea (*Pisum sativum*)	117
Pea (*Lathyrus sativum*)	82
Chick pea	140–354
Vetch	500
Potato	14
Carrot	0–4
Nuts and seeds	
Walnut	120
Hazelnut	104
Almond	189
Peanut	205
Cocoa bean	169
Pistachio nut	176
Rapeseed	795
Cottonseed	368
Spices and flavoring agents	
Millet	83
Caraway	297
Coriander	320
Cumin	153
Mustard	392
Nutmeg	162
Black pepper	115
Pepper	56
Paprika	71

brown bread (containing 214 mg phytic acid per 100 g) resulted in a 33 to 62% decrease in calcium absorption after 3 to 4 weeks, compared to a white bread diet. Addition of bran to white flour gave similar results. Only if the calcium intake was increased to 1.0 to 1.4 g per day, the calcium absorption improved. Calcium absorption is influenced not only by dietary phytate but also by vitamin D and lipids. If vitamin D is limiting in the diet, calcium absorption will be less efficient and the phytate effect will become more pronounced.

In many foodstuffs phytase activity can reduce the phytic acid level. Phytase is an enzyme occurring in plants. It catalyzes the dephosphorylation of phytic acid. Soybeans show weak phytase activity. Rye contains the most active phytase of all cereal grains. The activity of phytase drastically reduces the phytate content of dough during bread-making. Dephosphorylation of phytic acid is facilitated by the increase in acidity of bread dough caused by the reactivity of the yeast. Phytase is added to animal feeds so that no extra phosphate needs to be added. Also, in this way the animals will excrete less phosphate, which may contribute to reduction of environmental pollution.

3.3.2 Oxalic acid

Oxalic acid (HOOC–COOH) can induce toxic as well as antinutritive effects. To humans, it can be acutely toxic. However, it would require massive doses of 4 to 5 g to induce any toxic effect. The oxalic acid levels usually found in food, however, are no cause for concern. This section, discusses the presence of oxalic acid in food in relation to its antinutritive effects. Like phytic acid, oxalic acid reduces the availability of essential bivalent cations. Oxalic acid is a strong acid and, with alkaline earth metal ions and other divalent metal ions, it forms salts that are hardly soluble in water. Calcium oxalate is insoluble in water at neutral or alkaline pH, and dissolves easily in an acid medium. In many animal experiments and human studies, negative effects of oxalate-rich foods have been found, especially on calcium absorption. Vegetable foods such as rhubarb, spinach, and celery, as well as cocoa have been shown to disturb the calcium balance in man.

Negative effects of oxalic acid on calcium absorption can be predicted from the oxalate/calcium ratio of foods. Foods with a ratio higher than 1 may decrease the calcium availability (see Table 3.2). Foods with a ratio of 1 or lower than 1 do not interfere with

Table 3.2 Foods with an oxalate/calcium ratio (meq/meq) >1

Food	Oxalate mg/100 g[a]	Oxalate/Ca. meq/meq
Rhubarb	805	8.5
Common sorrel	500	5.6
Garden sorrel	500	5.0
Spinach	970	4.3
Beet, leaves	610	2.5
Beet, roots	275	5.1
Purslane	1294	4.6
Cocoa	700	2.6
Coffee	100	3.9
Potato	80	1.6
Tea	1150	1.1
New Zeeland spinach	—[b]	3.9
Pig spinach	—	4.9
Orache	900	4.0
Amaranth	—	1.4

[a] Average.

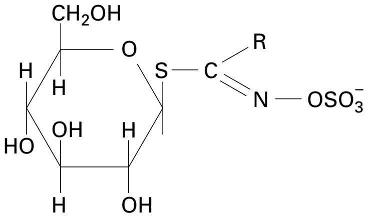
[b] No average available.

calcium absorption. Calcium is irreversibly bound to oxalic acid, so a food with an oxalate/Ca^{2+} ratio of 1 would not be a good calcium source, although it is rich in calcium.

The effects of oxalates may be influenced by the nutritional state of the subjects, the duration of the experiments, and the extent of calcium intake. For example, rats showed no serious effects after a diet containing 2.5% oxalate unless the diet was deficient in calcium, phosphorus, and especially vitamin D. Humans also show a remarkable ability to adapt to a drastic reduction in calcium intake. This can be attributed to the large calcium pool in the form of the skeletal system. Therefore, a decrease in calcium absorption, caused by oxalate, would not make much difference unless the calcium pool is (nearly) depleted. Consumption of foods rich in calcium, such as dairy products and seafood, as well as enhanced vitamin D intake are recommended only if large quantities of foods rich in oxalate are ingested. In this respect, the traditional way of preparing rhubarb is noteworthy. Before cooking the rhubarb , chalk is added to enhance its pallatability. The calcium, of course, is bound by the oxalic acid in the rhubarb, thereby inhibiting the chelation of calcium from other food sources. Today rhubarb varieties are available with lower oxalate levels.

3.3.3 Glucosinolates

A variety of plants contain a third group of antiminerals, the so-called glucosinolates, a class of thioglucosides, whose general structure is shown below.



Glucosinolate

Many glucosinolates are goitrogenic. Three types of goiter are distinguished: cabbage goiter (struma), brassica seed goiter, and legume goiter.

Cabbage goiter can be induced by the excessive consumption of cabbage. It seems that cabbage goitrogens inhibit iodine uptake by directly affecting the thyroid gland. Cabbage goiter can be treated by iodine supplementation.

Brassica seed goiter can result from the consumption of the seeds of Brassica plants, such as rutabaga (swede), turnip, cabbage, rape, and mustard, which contain substances that prevent thyroxine synthesis. This type of goiter can only be treated by administration of the thyroid hormone.

Legume goiter is induced by goitrogens in legumes such as soybeans and peanuts. It differs from cabbage goiter in that the thyroid gland is not involved directly. Inhibition of the intestinal absorption of iodine or the reabsorption of thyroxine has been shown in this case. Legume goiter can be treated by iodine therapy.

Fifty types of glucosinolates have been identified. Table 3.3 gives a list of foodstuffs which have been shown to induce goiter, at least in experimental animals.

Rutabaga, turnips, cabbage, peaches, strawberries, spinach, and carrots can cause a significant reduction in the iodine uptake by the human thyroid gland, with rutabaga being the most active.

Table 3.3 Goitrogenic foodstuffs

Food or feedstuff	Glucosinolate[a]	Specific chemical (R) Group
Broccoli (buds)	Glucobrassicin[b]	3-Indolylmethyl
	Gluconapin[c]	3-Butenyl
	Neoglucobrassicin[c]	3-(N-methoxyindolyl)methyl
	Progoitrin[c]	2-Hydroxy-3-butenyl
	Sinigrin[b]	Allyl
Brussel sprouts (head)	As in broccoli	
Cabbage (head)	As in broccoli	
Cauliflower (buds)	As in broccoli	
Charlock (seed)	Sinalbin[c]	*p*-Hydroxybenzyl
Crambe (seed)	Gluconapin[c]	(See broccoli above)
	Gluconasturtlin[c]	2-Phenylethyl
Garden cress (leaves)	Glucotropasolin[c]	Benzyl
Horseradish (roots)	Gluconasturtlin[c]	(See crambe above)
	Sinigrin[b]	
Kale (leaves)	As in broccoli	
Kohlrabi (head)	As in broccoli	
Mustard, black (seed)	Sinigrin[b]	(See broccoli above)
Mustard, white (seed)	Sinalbin[b]	(See charlock above)
Radish (root)		4-Methylthio-3-butenyl
	Glucobrassicin[c]	(See broccoli above)
Rutabaga (root)	Glucobrassicin[c]	(See broccoli above)
	Neoglucobrassicin[c]	(See broccoli above)
	Progoitrin[b]	(See broccoli above)
Turnips (root)	Gluconasturtlin[b]	(See crambe above)
	Progoitrin[c]	(See broccoli above)
		2-Hydroxy-4-pentenyl

[a] Trivial or common name of glucosinolate. The chemical name is formed by the designation of the R group prefixed to the term glucosinolate, e.g., glucobrassicin is 3-indolylmethyl-glucosinolate.

[b] Major glucosinolate.

[c] Minor glucosinolate component.

Hydrolysis, which takes place in damaged plant tissue before or after ingestion, yields the actual goitrogens: thiocyanates, isothiocyanates, cyclic sulfur substances, and nitriles. The thiocyanates and isothiocyanates formed from the glucosinolates are probably the substances responsible for cabbage goiter. As discussed in Chapter 2, goitrogens can also be formed from cyanogens, as their major biotransformation products are thiocyanates. An illustrative example of activation of a glucosinolate by hydrolysis is the potent antithyroid progoitrin occurring in the seeds of Brassica plants and the roots of rutabaga. This substance undergoes hydrolysis as shown in Figure 3.1.

The cyclization product of isothiocyanate III, goitrin (IV), is a powerful goitrogen. The R-enantiomer of goitrin found in crambe seeds is also a strong antithyroid agent. These substances interfere with the iodination of thyroxine precursors so that iodine therapy is not successful. The nitriles I and II obtained from progoitrin are highly toxic, but it is uncertain whether they are goitrogens. Mustard and rapeseed varieties have been bred with low thioglucoside concentration.

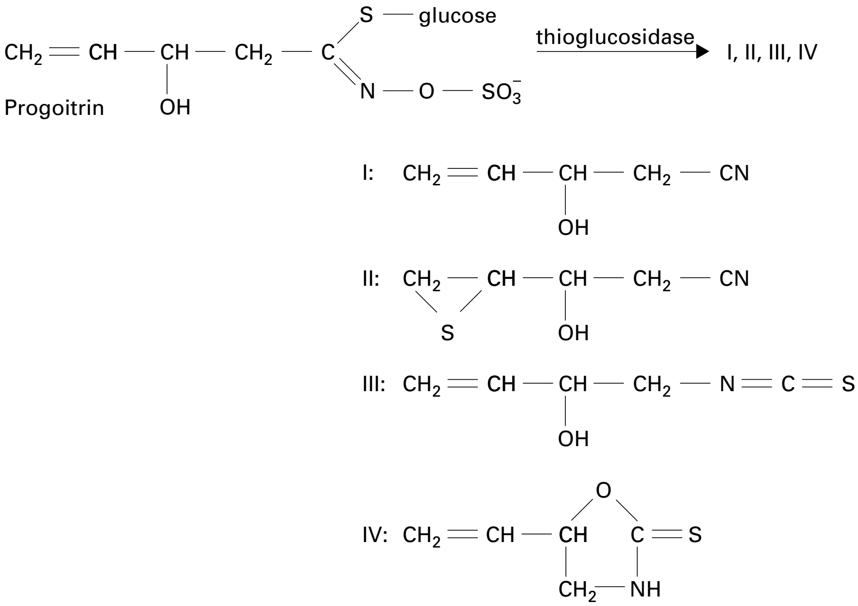


Figure 3.1 Hydrolysis of progoitrin.

3.3.4 *Dietary fiber*

Dietary fiber is a collective term for all food components derived from plant cell walls that are not digested by the endogenous secretions of the human digestive tract. It has no clearly defined composition. It may differ from foodstuff to foodstuff, and from diet to diet. Dietary fiber consists of pectic substances, hemicelluloses, plant gums and mucilages, algal polysaccharides, celluloses, and lignin. Further, tannins, indigestible proteins, plant pigments, waxes, siliceous materials, and phytic acid can be incorporated in the fiber matrix. These materials give bulk to the fecal matter, not only from their inherent mass, but also by their water-binding capacity. The amount of water bound can be four to six times the dry weight of the fiber.

Around 1970 it was suggested that dietary fiber is a protective factor against many diseases, prevalent in Western communities, e.g., colon cancer. This may be true, but harmful effects of overconsumption of fiber should not be overlooked. The various types of dietary fiber components have many reactive groups, including $-COOH$, $-HPO_3H$, $-OH$, $-SO_3H$ and $-NH_2$, to which metals, amino acids, proteins, and even sugars can be bound.

There are different ways of binding to dietary fiber. First, fiber components of many food products act like ion exchangers. Their binding capacity depends on pH and ionic composition of the bowel contents. Dietary fiber has the capacity to bind various metals, even if the phytic acid is removed. Disturbed Ca^{2+}, Mg^{2+}, Zn^{2+} and P balances have been observed in human subjects using diets rich in fiber in the form of whole wheat bread.

Secondly, amino acids and proteins are bound to dietary fiber. A diet containing 15% cellulose can cause a decrease in nitrogen absorption of as much as 8%. Carrageenans, which are highly indigestible, can cause a decrease in nitrogen absorption of about 16%. The interaction of dietary fiber with sugars does not result in a reduction of sugar absorption, but in a slow release of sugars into the bloodstream.

3.3.5 *Gossypol*

Gossypol is a plant antinutritive that would probably have remained unnoticed as a food hazard, if the seeds of the plant concerned had not gained importance as a dietary oil and protein source, especially in tropical and subtropical countries. This antinutritive is a yellow pigment present in all parts of the cottonplant. The highest levels are found in cottonseed.

Gossypol exists in three tautomeric forms (Figure 3.2): phenolic quinoid tautomer (I), aldehyde (II), and hemiacetal (III).

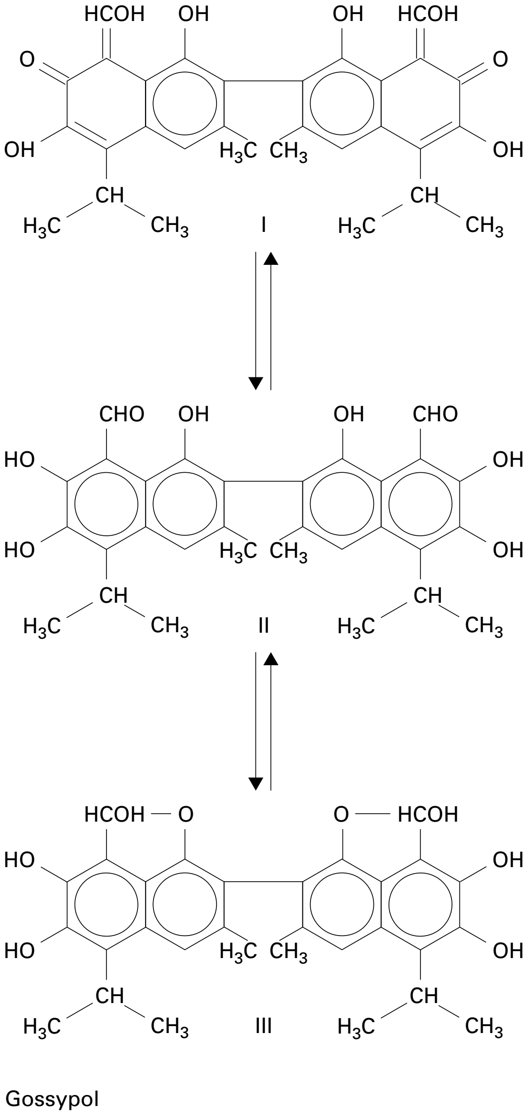


Figure 3.2 The three tautomeric forms of gossypol.

Gossypol is an antimineral as well as an antiprotein. It forms insoluble chelates with many essential metals, such as iron, and binds to amino acid moieties in proteins, especially to lysine. The protein binding suggests that gossypol can reduce the availability of food proteins and inactivate important enzymes.

Processing removes 80 to 99% of the gossypol. The pigment is extracted with the oil and subsequently removed by refining and bleaching. About 0.5 to 1.2% of the total gossypol generally remains in the processed meal. Less than 0.06% is free gossypol. The free gossypol concentration in cottonseed meal can also be lowered considerably by the formation of insoluble metal-gossypol complexes. The use of additives such as $FeSO_4$ and $Ca(OH)_2$ prevents the reaction of gossypol with lysine, even during heat treatment. Breeding of cotton with gossypol-free seeds has been succesful and this cotton variety is now planted commercially. In the US, the maximum allowable gossypol content of cottonseed products for use in human food has been set at 0.045%.

3.4 Type C antinutritives (antivitamins)

As defined in the introduction to this chapter, antivitamins are a group of naturally occurring substances which can decompose vitamins, form unabsorbable complexes with them or interfere with their digestive or metabolic utilization.

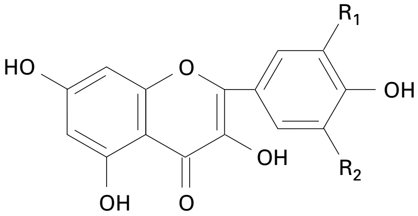
Only the more relevant examples of this type of antinutritives are discussed in this section, including ascorbic acid oxidase, antithiamine factors, and antipyridoxine factors.

3.4.1 Ascorbic acid oxidase

Ascorbic acid oxidase is a copper-containing enzyme that mediates the oxidation of free ascorbic acid first to dehydroascorbic acid and next to diketogulonic acid, oxalic acid, and other oxidation products (see Chapter 6, Section 6.6.2.1).

Ascorbic acid oxidase occurs in many fruits and vegetables such as cucumbers, pumpkins, lettuce, cress, peaches, bananas, tomatoes, potatoes, carrots, and green beans. Its activity varies with the type of fruit or vegetable. The enzyme is active between pH 4 and 7. Its optimum temperature is about 38°C. When plant cells are disrupted the compartmentalization of substrate and enzyme is removed. Therefore, if vegetables and fruits are cut, the vitamin C content decreases gradually. In fresh juices, 50% of the vitamin C content is lost in less then one hour. Being an enzyme, ascorbic acid oxidase can be inhibited effectively by blanching of fruits and vegetables.

Ascorbic acid can also be protected against ascorbic acid oxidase by substances of plant origin. Flavonoids, such as the flavonoles quercetin and kempferol, present in vegetables and fruits, strongly inhibit the enzyme. (As far as risk evaluation is concerned, it should be noted that quercetin has also been reported to induce adverse effects: see Part 1, Chapter 2, Section 2.2.1.1).



Kempferol ($R_1 = R_2 = H$)
Quercetin ($R_1 = OH; R_2 = H$)

3.4.2 *Antithiamine factors*

A second group of antivitamins is the antithiamine factors. They interact with vitamin B_1, also known as thiamine. Antithiamine factors can be distinguished as thiaminases, tannins, and catechols. The interaction with vitamin B_1 can lead to serious neurotoxic effects as a result of vitamin B_1 deficiency. Normally, antithiamine factors pose no appreciable risk to humans. They only cause thiamine deficiency in people whose diet is already low in thiamine.

Thiaminases are found in many fish species, freshwater as well as saltwater species, and in certain species of crab and clam. These antithiamine factors are enzymes that split thiamine at the methylene linkage (Figure 3.3).

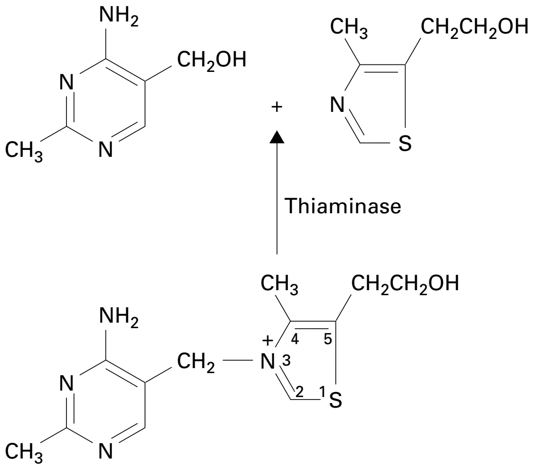
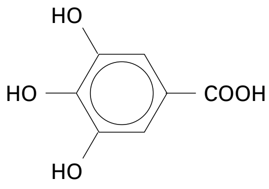


Figure 3.3 Degradation of thiamine by thiaminase.

Thiaminases contain a nonprotein coenzyme, structurally related to hemin, the red pigment component of hemoglobin. The coenzyme is the actual antithiamine factor. Cooking destroys thiaminases in fish and other sources.

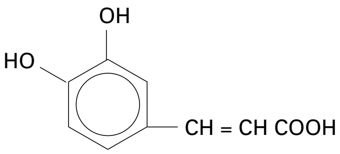
Antithiamine factors can also be of plant origin. *Tannins*, occurring in a variety of plants, including tea, are believed to be responsible for inhibition of growth in animals, and for inhibition of digestive enzymes. A study in volunteers on the effects of tannins in tea leaves, tea infusions and betel nuts on thiamine, has shown that the tannins were responsible for thiamine destruction. Tannins are a complex of esters and ethers of various carbohydrates. A component of tannins is gallic acid.



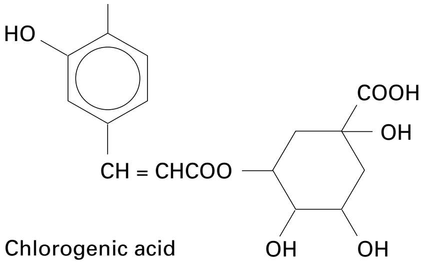
Gallic acid

Gallic acid is obtained by hydrolyzing tannins. The interaction of these substances with thiamine is oxygen-, temperature-, and pH-dependent. It appears to proceed in two phases: a rapid initial phase, which is reversible on addition of reducing agents, such as ascorbic acid, and a slower subsequent phase, which is irreversible.

A variety of antithiamine factors are the *ortho-catechol* derivatives. A well-known example is present in bracken. So-called fern-poisoning in cattle is attributed to this factor. Possibly, there are two types of heat-stable antithiamine factors in this fern, one of which has been identified as caffeic acid (3,4-dihydroxycinnamic acid).

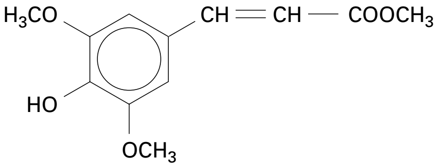


Caffeic acid



Chlorogenic acid

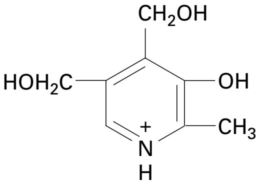
Caffeic acid can also be formed on hydrolysis of chlorogenic acid by intestinal bacteria. Chlorogenic acid is found in green coffee beans and green apples. Other *ortho-catechols*, such as methylsinapate occurring in mustard or rapeseed, also have antithiamine activity.



Methylsinapate

3.4.3 *Antipyridoxine factors*

A variety of plants and mushrooms contain pyridoxine (a form of vitamin B_6) antagonists. The antipyridoxine factors have been identified as hydrazine derivatives.



Pyridoxine
(a form of vitamin B_6)

Linseed contains the watersoluble and heat-labile antipyridoxine factor linatine. Linatine is γ-glutamyl-1-amino-D-proline. It readily undergoes hydrolysis to the hydrazine derivative, 1-aminoproline, the actual antipyridoxine factor (Figure 3.4).

Antipyridoxine factors have also been found in wild mushrooms, the common commercial edible mushroom, and the Japanese mushroom shiitake. Commercial and shiitake mushrooms contain agaritine. Agaritine is hydrolyzed in the mushroom by γ-glutamyltransferase to the active agent 4-hydroxymethylphenylhydrazine (Figure 3.5). The hydrolysis of agaritine is accelerated if the cells of the mushrooms are disrupted. Careful

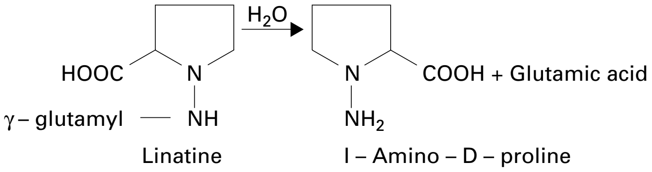


Figure 3.4 Hydrolysis of linatine.

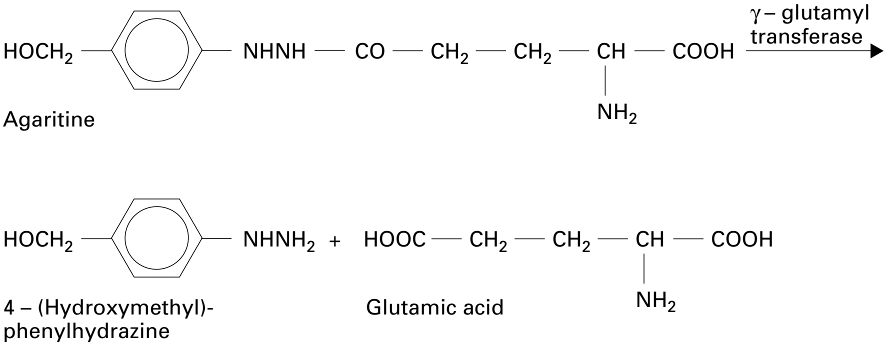


Figure 3.5 Hydrolysis of agaritine.

handling of the mushrooms and immediate blanching after cleaning and cutting can prevent hydrolysis.

The mechanism underlying the antipyridoxine activity is believed to be condensation of the hydrazines with the carbonyl compounds pyridoxal and pyridoxal phosphate — the active form of the vitamin — resulting in the formation of inactive hydrazones.

3.5 *Summary*

Antinutritives induce their toxic effects indirectly by causing nutritional deficiencies or by obstructing the utilization or functioning of nutrients, mainly proteins, minerals, and vitamins. Especially in the case of marginal nutritional status or malnutrition, the effects of antinutritives can become manifest.

The majority of antinutritives are of natural origin. They are distinguished as three types: antiproteins, antiminerals, and antivitamins. Antiproteins interfere with the digestion of proteins or the absorption and utilization of amino acids. Well-known examples are protease inhibitors and lectins. Both groups are proteins. Protease inhibitors inhibit proteolytic enzymes. Lectins belong not only to the antiproteins but also to the antiminerals and antivitamins. They interfere with the absorption of amino acids as well as that of iodine and vitamins. Antiminerals interfere with the absorption of minerals and essential trace elements, resulting in the reduction of their bioavailability. They include acids, such as phytic and oxalic acid, that form insoluble salts with bivalent and tervalent heavy metal ions. Another group of antiminerals, the glucosinolates, interfere with the absorption of iodine, thus causing goiter. Antivitamins interfere with vitamins in various ways. They can decompose them, form unabsorbable complexes, or disturb their physiological utilization. Relevant examples of this type of antinutritives are ascorbic acid oxidase, antithiamine, and antipyridoxine factors. Many antinutritives can be eliminated from food by various ways of food processing.

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