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OPINION

of the *Agence française de sécurité sanitaire des aliments*
(French Food Safety Agency)

Opinion relative to the request for the formulation of recommendations concerning the diet of individuals with Glucose-6-Phosphate dehydrogenase (G-6-PD) deficiency

In a letter received on 27 May 2006 the Directotae General for Health (DGS) consulted Afssa (French Food Safety Agency) for its opinion relative to the formulation of recommendations concerning the diet of individuals with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency. In parallel with this request for Afssa's opinion, the DGS also consulted the *Agence française de sécurité sanitaire des produits de santé* (Afssaps – French Agency for the safety of healthcare products) for its opinion relative to the formulation of recommendations for the use of medicines in individuals with G-6-PD deficiency.

Expert report methodology

The joint expert report was produced on the basis of a draft opinion formulated from the studies of experts appointed by Afssa's "Human Nutrition" specialist expert committee. This draft opinion was then discussed at the "Human Nutrition" specialist expert committee's meetings on 20 April and 18 May, before being validated on 7 July 2006.

The "Human Nutrition" specialist expert committee issues the following opinion:

With respect to glucose-6-phosphate dehydrogenase, the pentose phosphate pathway and glutathione

In the various cell types on the body, several antioxidant systems use nicotinamide adenine dinucleotide phosphate (NADPH) for its reducing capacity. The pentose phosphate pathway is the main pathway for $NADPH^+$ reduction. It is a cytoplasmic pathway that starts from glucose-6-phosphate and enables the synthesis of 2 NADPH due to the activity of glucose-6-phosphate dehydrogenase and 6-P gluconate dehydrogenase. NADP is involved in numerous biosynthesis pathways (amino acids, deoxyribonucleotides, steroids, lipids). NADPH is one of the cofactors of nitrogen monoxide synthases. It is also involved in various antioxidant systems (involving thioredoxin reductase, haeme oxygenase, coenzyme Q reductase and glutathione reductase). In the presence of NADPG, glutathione reductase reduces oxidised glutathione into reduced glutathione (GSH).

With respect to conditions related to G-6-PD deficiency

G-6-PD deficiency is a genetic condition linked to a deficiency in an enzyme, glucose-6-phosphate dehydrogenase. This is an X-linked genetic defect; the deficiency is therefore passed on by females but primarily affects males; cases can occur in females, however, although they are less common *(Jolly D, 2000).* This disease mainly affects male populations of African, Indian, Mediterranean basin and South-East Asian descent. The clinical expression of G-6-PD deficiency can take several forms:

- either the mutation linked to the genetic defect simply causes impaired function of the enzyme; in this case, the reduction in enzymatic activity is not marked enough to be symptomatic. This is the case for the majority of deficiencies;
- or the G-6-PD enzyme is almost totally absent and, in this case, serious clinical conditions can occur:
    - severe neonatal jaundice in newborns,
    - potentially fatal haemolytic anaemia at any age.

There are two different types of G-6-PD deficiency, some of which do not cause haemolytic reactions (types A+ and B+). For other types of deficiency (A- and B-), chronic haemolysis, and sometimes acute haemolysis, are sometimes reported. There are numerous G-6-PD variants, since a hundred and fifty mutations have been described by Arese *(Arese P, 1982)*. The clinical expression varies depending on the type of variant; the most common ones are the Mediterranean variant (severe form), the African A- variant (moderate form) and the Canton (severe form) and Mahidol (moderate form) variants in South-East Asia *(Ducrocq R, 2004).*

Acute haemolysis is usually triggered:

- by oxidising medicines: certain antibiotics (especially quinolones and sulphonamides), antimalarial drugs containing quinine, some analgesics, etc. This aspect of the risk assessment is handled by Afssaps;
- by the consumption of broad or fava beans (hence the term “favism”) or certain substances (quinine, vitamin C) that can be found in foods. This aspect of the risk assessment falls within Afssa’s scope.

Due to the polymorphism of the deficiency, beans and the medicines and substances cited above do not pose a risk to all deficient individuals. It is nonetheless prudent to avoid them systematically since the tolerance threshold is specific to each individual. Furthermore, there is no known link between a particular mutation and the tolerance of the carrier to beans and the medicines and substances indicated above.

With respect to estimation of the frequency of the deficiency in France and available indicators

There is no data available concerning the prevalence and/or incidence of this deficiency within the general population in France since no epidemiological studies have been conducted to assess the number of individuals with a G-6-PD deficiency. The "Vigifavisme" association for patients and their families estimates that "there may be between 250,000 and 450,000 people in France" living with this deficiency.

WHO (the World Health Organisation) reports an estimated prevalence in France of 0.39%, i.e. 3.9 per 1000 male individuals *(Report of a WHO working group on G-6-PD deficiency, 1985).* Relative to a population of 62 million inhabitants (with a 50% distribution of births between boys and girls), the estimated figure is close to 120,000 people with this deficiency in France. However, the WHO estimate does not take into account the prevalence of the disease in French overseas regions and territories, which could be high (12% of males in Guadeloupe and between 4 and 14% in French Guyana according to the "Vigifavisme" association).

As far as severe forms of the condition are concerned, more recent information can be obtained from PMSI (*Programme de médicalisation des systèmes d'information* – information system medicalisation programme) data and be used as indicators. The PMSI provides quantified and standardised information for measuring the activity of public and private hospitals participating in the public sector service, i.e. 613 hospitals in France. In the PMSI, only item (D55.0) entitled "anaemia due to G-6-PD deficiency" is available.

According to data obtained from the *Agence Technique d'Information Hospitalière* (Technical Agency for Hospital Information) for the year 2004 (the last year for which PMSI data are available), a principal diagnosis of "anaemia due to G-6P-D deficiency" (D55.0) was made for 55 patients and a diagnosis linked to this principal diagnosis was made for 15 patients. It can therefore be considered that at least 70 patients were hospitalised one or more times during the course of 2004 linked to this diagnosis, which represents the severe form of the disease. PMSI data also reveal that in the same year (2004), 317 patients presenting this genetic deficiency were hospitalised for treatment of a condition other than haemolytic anaemia due to G-6-PD deficiency (PMSI associated diagnostic item). On a regional scale, the main two regions of mainland France having recorded this diagnosis of haemolytic anaemia are the Ile de France region (27/70) and the PACA region (12/70) *(ATIH, ref. No. DL-CL-92, January 2006)*.

A diagnosis of anaemia due to a G-6-PD deficiency warranting hospitalisation is therefore rarely made. However, this in no way reduces the seriousness of the prognosis but partially explains the poor level of knowledge about this disease amongst non-specialised medical personnel.

With respect to the foods involved in haemolytic reactions due to G-6-PD deficiency

1 - Leguminous species

Due to the existence of variations in the intensity of the deficiency between individuals, all subjects suffering from favism by definition present a G-6-PD deficiency but not all subjects with the deficiency are sensitive to beans. The mechanism of these reactions linked to eating beans is not fully known.
The different species of leguminous plants with seeds that have been reported in the literature belong to the Vicia genus.

- *The different species of the Vicia genus concerned*

The *Vicia faba* L. species is a leguminous plant belonging to the *fabaceae* family of the *Vicia* species. The ancestral form of *Vicia faba* is not known. The species is believed to have originated in the "Fertile Crescent". It is then thought to have migrated around the Mediterranean Basin, leading to the growing of "small-seeded field bean" crops in Northern Europe and "large-seeded broad bean" crops around the Mediterranean. At the same time, bean types are believed to have migrated towards Asia. It was not until the 16th century that the bean was introduced into Central and South America.

Cultivated since neolithic times, the species includes three botanical varieties (sub-species):

- ***V. faba var. minor*** **and** ***V. faba var. equina***. These botanical varieties have seeds that are respectively small (approximately 400-600 g per 1000 seeds) and medium (600-800 g per 1000 seeds) in size. They are mainly grown as animal feed, especially in Western and Northern Europe. The dried seeds are primarily used as protein supplements in animal feed. They are also used in human food, marketed in the form or dry seeds or flour. These field beans can also be used as fodder (consumed green, in silage or dried) or as a green manure.
- ***V. faba var. major***. The main feature of this botanic variety is the large size of its seeds (often more than 1000 g per 1000 seeds). They are intended for human consumption and are called "broad beans" or "fava beans". In these edible beans, the seed can be eaten young (raw or cooked) or dried (dry legume). The zone in which it is grown includes the Mediterranean basin, South America and China.

There is a big market in France for export to Egypt of dried seeds, especially field beans, for human consumption.

Numerous varieties of *Vicia faba* are marketed in Europe. In the French variety catalogue (*Comité Technique Permanent de la Sélection* – Permanent Technical Selection Committee), there is a list of edible broad bean varieties and a list of high-protein field bean varieties.

Other species of the *Vicia* genus are used in agriculture and are mainly used for animal feed. However, the existence of a few cases of direct consumption by humans cannot be excluded, whether these be marginal or accidental. An example is ***Vicia sativa L.*** (vetch), which is mainly grown as a fodder crop and a green manure; it has a very high chromosomal variability (there are 6 main sub-species and hybrids). There are also other types of vetch, which are less commonly grown: *Vicia villosa* Roth. (with 5 sub-species), *V. articulata* Hornem, *V. Ervilia* (L.) Willd., *V. pannonica* Crantz (2 sub-species), *V. narbonensis* L. (or Narbonne vetch, with two varieties: *serratifolia* and *heterophylla*).

- *The substances contained in beans posing a risk to individuals with G-6-PD deficiency*

On the basis of the current state of knowledge (*Franck, 2005; Arese, 1982; Arese et al., 1981)* only *two pyrimidinic substances* have been identified as substances posing a risk to individuals with G-6-PD deficiency:
a) **Divicine** (or 2,6-diamino-5-hydroxy-4(1H)-pyrimidonone). Divicine is the aglycone of a glycosylated form made by certain plants: **vicine** or 5-O-D-glucopyranoside, sometimes called vicioside, isolated from *Vicia faba, Vicia sativa* and *Vicia narbonensis* L.

b) **Isouramil** (or 6-amino-5-hydroxy-2,4(1H,3H)-pyrimidinedione, 6-amino-2,4,5-pyrimidinetriol or 6-aminobarbituric acid).
A single glycosylated derivative has been described: **convicine** (5-O-D-glucopyranoside), isolated from the seeds of *Vicia faba, Vicia sativa* and *Vicia narbonensis*.

The biogenesis of these structures from orotic acid is reminiscent of the very similar biogenesis of nucleotides from pyrimidine. These pyrimidic compounds have been identified as the substances responsible for favism disorders and have been well described since Ancient times and linked to bean poisoning.

The real active substances are aglycones, which are more lipophilic than glycosylated forms and which, after having being absorbed (and probably due to an availability favourable to their penetration along with a sufficient lifetime in red blood cells), are liable to produce oxidised derivatives at position 5 since a hydroxyl function has been released after hydrolysis. These aglycones (reduced) and their oxidised counterparts are part of a cascade of essential substances that also oscillate between two forms (oxidised and reduced) and are involved in several redox reactions. Divicine added *in vitro* to erythrocytes taken from individuals with G-6P-D deficiency induces a decrease in reduced glutathione concentrations *(Baker et al., 1984)*. Oxidant stress in the erythrocytes is believed to lead to oxidation of haemoglobin with release of reactive iron, liable, via Fenton's reaction, to modify the composition of the cell membranes *(Bracci et al., 2002).* Studies in animals have led to a better understanding of the mechanisms underlying the effects of divicine and isouramil on erythrocytes: fall in reduced glutathione content with haemolysis and lipid peroxidation *(D'Aquino et al., 1983)*; additional oxidant stress attributed to haemoglobin and/or to products derived from haemoglobin *(Chan et al., 1999; Chiu and Lubin, 1989; Dhaliwal et al., 2004)* and release of iron that can be chelated by desferioxamine *(Ferrali et al., 1992 and 1997).*

radical species
Fenton's reaction

Isouramil: R=OH
Divicine: R=NH_2

oxidised forms
(pyrimidine diones)

GSH-reductase

G-6-PD

Pyrimidines are secondary metabolites which, on the basis of the current state of knowledge, have only been found in a small number of seeds: in *Vicia faba* L, *Vicia sativa* L. and *Vicia narbonensis* species *(Pitz et al., 1980).*

In sum, on the basis of the current state of knowledge, *Vicia faba* L., a seed mainly used as a human food, has been reported as being dangerous in the event of G-6-PD deficiency in humans.

However, the various forms of consumption do not all pose the same degree of risk:

- *Fresh or frozen seeds:*

Fresh bean seeds are the edible parts of the plant with the highest vicine and convicine contents *(Ramsay and Griffiths, 1996).* These parts are reputed to be the key components triggering a haemolytic crisis (the vicine + convicine content is often more than 2% of the dry matter in fresh seeds). The fresh seeds also contain beta-glucosidases and ascorbate *(Arigoni et al, 1992; Sisini et al, 1981)* which play a role in the intensity of the crisis. Marked variations in the contents of these various ingredients have been reported depending on the variety of bean, the growing conditions and the stage at which the seed was harvested.

On the basis of the current state of knowledge, the consumption of raw fresh seeds by individuals with G-6-PD deficiency, and particularly by children with this deficiency, represents the situation posing the maximum risk.

In *Vicia faba* natural genetic resource collections, a gene has been discovered (the vc- gene) that reduces the vicine and convicine contents of fresh or dried seeds by a factor of between 10 and 20 *(Duc et al., 1989)*. This gene has not yet been introduced into edible bean varieties intended for human consumption.

- *Use of dried beans or bean flours as food ingredients:*

In France, beans and bean flours are ingredients used mainly in bakery products. The INCA (*Enquête individuelle de la consummation alimentaire* – Individual diet survey) nomenclature recipe data base set up by Afssa to act as a support for exposure studies contains around 10,500 recipes created using information indicated on the packaging of food products. From 887 foods recorded in this recipe data base, it appears that only *"pain de mie"* (white batch bread) includes the ingredient "bean" in its composition. Beans are also used to make a number of complex products, such as powdered vegetable soups or purees for example.

The vicine and convicine contents of dry-harvested seeds (or dry seeds harvested at the mature stage) are approximately 50% lower than those of fresh seeds (harvested at the immature stage). Conventional varieties have a vicine + convicine content that ranges from 0.5% to 1.5% of the dry matter of the mature seed. Registered varieties with the vc- gene have contents ranging from 0.01% to 0.1%. The dry seeds also have a lower ascorbate and beta-glucosidase content than fresh seeds.

On the basis of the current state of knowledge, the seeds of dry beans and dry bean flours, used especially as food ingredients, pose a lower risk than fresh bean seeds.

Directive 2003/89/EC, applicable since 25 November 2005, requires the food industry to label ingredients deliberately added to finished products.
The presence of beans in vegetable soups or purees must therefore now be indicated in the list of ingredients.
However, for bakery and pastry products, which include unpackaged products not subject to the labelling obligation, it is common for the presence of beans not to be indicated.
Bean flour is one of the ingredients authorised by order 26/04/1945 as an additive that may be incorporated into bread flour (in the same way as gluten, wheat malt, soy flour). The maximum incorporation rate for bean flour in bread flour is set at 2% (Decree of 23 October 1954). Bean flour is therefore considered to be an "improving ingredient", in particular improving bleaching of white batch bread. Bean flour can also be used in the production of *"pain de tradition française" (*traditional French bread), also called *"pain traditionnel français"* or *"pain traditionnel de France"* (Decree No. 93-1074 of 13/09/1993). The dough for this bread may contain up to 2% bean flour (Article 2 of decree No. 93-1074) *(JO No. 213 of 14 September 1993, Syndicat national des fabricants de produits intermédiaires pour boulangerie, pâtisserie et biscuiterie, Confédération nationale de la Boulangerie-Pâtisserie française).*

- *Effects of bean storage and preparation conditions:*

Apart from the observations outlined above concerning dry beans or bean flours, there are few data available enabling assessment of the effects of temperature on beans, nor the consequences of long-term storage prior to consumption. The beta-glucosidase activity and ascorbate are reputed to be sensitive to cooking whereas vicine and convicine are often considered to be relatively resistant to cooking and also to germination treatments *(Hussein et al., 1986).*

- *Effects of bean pollens:*

"Bean pollens" have been considered to have been responsible for haemolytic reactions. Based on the current state of knowledge, no scientific evidence supports this hypothesis. In fact, the quantities that can be absorbed by inhalation appear to be low, given the stickiness and poor diffusion in the air of the pollen.

2 - Other substances

According to the bibliographic analysis, beans are the only foods that have been shown to be responsible for triggering haemolytic reactions.
Two substances are also reported as being a cause of haemolytic reactions in subjects with G-6-PD deficiency:

- vitamin C (ascorbic acid): erythrocytes are known to have a high capacity to regenerate vitamin C from dehydroascorbic acid by glutathione-dependent reduction *(May, 1998).* In the absence of glucose, reduction of dehydroascorbate depletes human enterocytes of reduced glutathione *(May et al., 1996).* Reactions have been described following the oral intake of vitamin C in individuals with G-6-PD deficiency *(Mehta et al., 1990).* In addition, Afssa reiterates that the vitamin C safety limit for RDIs (Recommended Daily Intakes) is 1 g *(Martin et al., 2001; CSHPF opinion, 1996).*

  At this stage, it does not appear to be possible to set a threshold dose for vitamin C that could pose a risk to consumers with G-6-PD deficiency based on solid scientific evidence. Afssaps and Afssa are currently in the process of carrying out an in-depth examination of this specific point and will issue an additional recommendation as soon as possible.

- quinine: medicines containing quinine can potentially cause acute haemolysis in individuals with G-6-PD deficiency *(Bennett et al., 1967; Rey et al., 1971).* Since Afssaps has classed this active substance as belonging to type II ("Substances requiring precautions for use in individuals with G-6-PD deficiency"), it is recommended that patients with this deficiency be offered an alternative treatment to quinine and its derivatives.
  A certain number of beverages aimed at consumers contain quinine derivatives. The maximum authorised quinine concentration in these beverages is 70 mg/L. Theoretically, the occurrence of reactions after drinking these beverages cannot therefore be excluded.

Furthermore, when administered experimentally in large quantities (10 to 160 mg/kg/d) by gavage in rats for 13 weeks, certain aniline derivatives can induce a reduction in red cell count and a reduction in the haemoglobin content of red blood cells *(Hejtmancik et al., 2002).* Also in rats, doses of aniline (in hydrochloride form) of between 10 and 100 mg/kg bodyweight for 1 to 4 weeks produce haemoglobin adducts *(Zwirner-Baier et al., 2003).* Since aniline is a synthesis impurity of certain food colourings (existence of a regulatory maximum level of 10 mg/kg in E105 fast yellow), these data suggest that aniline may therefore have a negative impact on erythrocyte metabolism at high doses.

In conclusion, the "Human Nutrition" specialist expert committee considers that:

Concerning the recommendations relative to the diet of individuals with G-6-PD deficiency:

- Based on the current state of knowledge, it appears that disorders linked to G-6-PD deficiency can only occur with a very small number of foods of plant origin commonly eaten in France. Only the consumption of varieties of *Vicia faba* (broad beans or field beans), the most frequently consumed of the *Vicia* species by humans, and, to a lesser extent, *Viscia sativa* (vetch), eaten rarely or not at all by humans, is contraindicated in individuals with G-6-PD deficiency, irrespective of the preparation or storage conditions for these foods. To date, no other foods have been reported to have vicine or convicine contents that are high enough to be the direct cause of a haemolytic reaction in individuals with G-6-PD deficiency.
  Food supplements[1] containing beans or bean extracts should also be contraindicated in individuals with G-6-PD deficiency.
  Afssa recommends that beans not be consumed in the event of G-6-PD deficiency, irrespective of their preparation and consumption conditions.

- Apart from beans, only two substances that can be found in foods are reported as being the cause of haemolytic reactions linked to G-6-PD deficiency. These are quinine and vitamin C.

With respect to quinine:

- Considering that the quinine present in certain beverages has been reported as being the cause of haemolytic reactions in individuals with G-6-PD deficiency;
- Considering that Afssaps recommends an alternative treatment to the prescription of quinine in the event of G-6-PD deficiency;

[1] food supplements are "foodstuffs the purpose of which is to supplement the normal diet and which are concentrated sources of nutrients or other substances with a nutritional or physiological effect, alone or in combination, marketed in dose form, namely forms such as capsules, pastilles, tablets, pills and other similar forms, sachets of powder, ampoules of liquids, drop dispensing bottles, and other similar forms of liquids and powders designed to be taken in measured small unit quantities". Journal Officiel de la République Française, Decree No. 2006-352 of 20 March 2006, JO No. 72 of 25 March 2006 page 4543.

Considering that there are nutritional alternatives to the consumption of beverages containing quinine;

Afssa recommends that quinine-containing beverages not be consumed in the event of G-6-PD deficiency.

With respect to vitamin C:

- Considering that the vitamin C present in certain foods can cause haemolytic reactions in individuals with G-6-PD deficiency;
- Considering that reactions have in fact been reported following the oral intake of vitamin C by individuals with G-6-PD deficiency;

Afssa recommends caution in individuals with G-6-PD deficiency in the event of the consumption of large quantities of products naturally rich in vitamin C (see list appended according to *S.W Souci et al., La composition des aliments; tableaux des valeurs nutritives; Medpharm Scientific Publishers CRC Press; Stuttgart)* and also in the event of consumption of foodstuffs enriched with vitamin C (in particular, certain fruit juices).
More specific recommendations will be proposed once joint examination of the issue with Afssaps has been completed.
It is also recommended that food supplements[1] containing vitamin C not be consumed in the event of G-6-PD deficiency.

- With respect to aniline, which can be found as a synthesis impurity in certain food colourings,
  - due to the reproduction of an experimentally-induced haemolytic phenomenon in rats, under specific exposure conditions (at high doses);
  - due to the absence of data in the scientific literature reporting the existence of reactions occurring in humans:

  no specific recommendations appear to be warranted at this stage in terms of prevention.

Concerning the recommendations in terms of labelling:

- If broad bean or field bean flour is used as an ingredient by the food industry, this must be indicated on the labelling of the finished product in accordance with Directive 2003/89/EC, applicable since 25 November 2005 (Decree of 2 August 2005).

- However, this labelling is subject to numerous exemptions in the bakery-pastry product sector. Although no data concerning the precise bean flour content of bakery products are available, the use of this flour as an improving agent and not as an ingredient for nutritional or organoleptic purposes suggests that the level of 2% is generally complied with. Furthermore, the scientific literature does not report any haemolytic reactions occurring in patients with G-6-PD deficiency following the consumption of foodstuffs from the bakery-pastry sector. A risk of reactions could emerge if certain bakers were to express the desire to create a product with a particularly high bean content. Accordingly, it would be useful to warn the profession against the excessive use of beans in recipes

and to recommend that the level of 2% required by the Order of 23 October 1954 not be exceeded.

- More generally, the food and catering industries' awareness of favism could be raised in the form of an information guide describing the disease and its potential consequences. This guide would be aimed at catering professionals, shopkeepers (particularly in the bakery-pastry products sector), as well as the managers and personnel of company and school canteens.

- Favism is also a condition that is not well known by health professionals, apart from certain specialists. In order to be able to provide clear information in terms of prevention to patients informed about their G-6-PD deficiency, it is important to make this condition known to future doctors (university training) and to practising doctors (professional training).

Other recommendations:

- Considering the existence of a gene (vc-) in broad beans and field beans that could reduce the vicine and convicine contents by a factor of between 10 and 20, bean plant-breeders should be encouraged to incorporate this gene in their selection work, particularly in *Vicia faba* varieties intended for human consumption.

- The fundamental studies that exist on favism and G-6-PD deficiency are fragmented. Research aimed at gaining a better understanding of the risk factors for the occurrence of a haemolytic reaction (human mutations, quantities of foods ingested, in particular) in individuals with this deficiency should be encouraged. In terms of diagnostic data, reference analytical methods to measure vicine and convicine contents in foods should also be defined.

References

Pascale BRIAND

ANNEX

Source: *La composition des aliments; tableaux des valeurs nutritives*
Souci SW, Fachmann W, Kraut H. (2000) 6th ed. Medpharm Scientific Publishers CRC Press; Stuttgart, 1182 pages.

Vitamin C content of:
mean values (extreme values found)

Fruits:

Pip fruits:

Apple: 12 mg/100 g (3-25) of edible matter
Dried apple: 12 mg/100 g
Apple puree: 2 mg/100 g (1-2.9)
Pear: 4.6 mg/100 g (2-9.9)
Tinned pears: 2 mg/100 g (1-4)
Quince: 13 mg/100 g

Core fruits:

Apricot: 9.4 mg/100 g (5-15)
Dried apricot: 11 mg/100 g (3-17)
Tinned apricots: 4 mg/100 g
Morello cherry: 12 mg/100 g
Cherry: 15 mg/100 g (8-37)
Tinned cherries: 5 mg/100 g (3-6)
Mirabelle: 7.2 mg/100 g (3-14)
Peach: 9.5 mg/100 g (5-29)
Dried peach: 17 mg/100 g (12-19)
Tinned peaches: 4 mg/100 g (3-6.1)
Plum: 5.4 mg/100 g (2.4-14)
Prune: 4 mg/100 g (2-5)
Tinned plums: 1.5 mg/100 g (1-2)
Greengage: 5.8 mg/100 g (5-8)

Berries:

Boysenberries: 13 mg/100 g
Blackberries: 17 mg/100 g (12-21)
Strawberries: 63 mg/100 g (45-94)
Tinned strawberries: 30 mg/100 g (15-55)
Bilberries: 22 mg/100 g (10-44)
Tinned bilberries without added sugar (whortleberries): 12 mg/100 g (9-14)
Raspberries: 25 mg/100 g (16-30)
Tinned raspberries: 5 mg/100 g (2-9)
Redcurrants: 36 mg/100 g (26-47)
Blackcurrants: 177 mg/100 g (30-40)
Whitecurrants: 35 mg/100 g (30-40)
Cranberries: 11 mg/100 g (10-12)
Lingonberries: 12 mg/100 g (11-20)

Gooseberries: 35 mg/100 g (30-48)
Grapes: 4.2 mg/100 g (2-7.4)
Raisins: 1.00 mg/100 g (1-2)

Wild fruits:

Sorbs: 98 mg/100 g (78-117)
Rosehips: 1250 mg/100 g (250-2900)
Elderberries: 18 mg/100 g (10-29)
Dogwood: 78 mg/100 g
Sea buckthorn berries: 450 mg/100 g (100-1200)

Exotic fruits:

Acerola: 1700 mg/100 g (1000 to 2000)
African ackee: 26 mg/100 g
Pineapple: 19 mg/100 g (10-25)
Tinned pineapple: 7 mg/100 g (5-9)
Orange: 49 mg/100 g (39-65)
Avocado: 13 mg/100 g (9-16)
Banana: 12 mg/100 g (7-21)
Cyphomandra (tree tomato): 24 mg/100 g (17-30)
Breadfruit: 21 mg/100 g (12-29)
Carissa: 47 mg/100 g (38-56)
Cashew apple: 252 mg/100 g (150-400)
Chayote: 17 mg/100 g (14-20)
Cherimoya: 15 mg/100 g (9-24)
Date (dried): 3 mg/100 g
Durian: 42 mg/100 g (32-58)
Fig: 2.7 mg/100 g (0.7-3.3)
Dried fig: 2.5 mg/100 g (0-5)
Pomegranate: 7 mg/100 g (5-20)
Grapefruit: 44 mg/100 g (38-55)
Guava: 273 mg/100 g (132-450)
Jaboticaba: 17 mg/100 g
Jackfruit: 9 mg/100 g
Japanese medlar: 4 mg/100 g
Jujube: 58 mg/100 g (46-70)
Persimmon: 16 mg/100 g (6-50)
Cape gooseberry: 28 mg/100 g (10-40)
Carambola: 34 mg/100 g (29-38)
Kiwi: 46 mg/100 g (17-295)
Kumquat: 38 mg/100 g (36-40)
Lime: 44 mg/100 g (38-46)
Lychee: 39 mg/100 g (25-50)
Longan: 56 mg/100 g
Mammey apple: 14 mg/100 g (4-22)
Mandarin: 30 mg/100 g (29-31)
Mango: 37 mg/100 g (28-55)
Mangosteen: 2.70 (2-4)
Naranrilla: 67 mg/100 g (31-78)
Okra: 36 mg/100 g (25-47)
Prickly pear: 23.00 mg/100 g (17-42)
Papaya: 80 mg/100 g (50-130)

Granadilla: 24 mg/100 g (17-40)
Rambutan: 53 mg/100 g
Rose apple: 22 mg/100 g
Sapotier: 12 mg/100 g
Sapote: 23 mg/100 g
Tamarind: 3 mg/100 g
Water chestnut: 5 mg/100 g (4-6)
Watermelon: 6 mg/100 g
Lemon: 51 mg/100 g (35-62)
Cantaloupe melon: 32 mg/100 g

Nuts:

Sweet chestnuts: 27 mg/100 g (0-60)
(Peanuts: not assayed)
Hazelnuts: 3 mg/100 g
Coconut: 2 mg/100 g
Coconut milk: 2 mg/100 g
Almond: from 0.8 to 6.5 mg/100 g
Brazil nuts: 700 mg/100 g
Pecan nuts: 2 mg/100 g
Pistachio nuts: 7 mg/100 g (0-14)
Walnuts: 2.6 mg/100 g

Fruit juices:

Pineapple juice: 11 mg/100 g (8-12)
Apple juice: 1.4 mg/100 g (0.7-2)
Fresh orange juice: 49 mg/100 g (23-69)
Packaged orange juice: 43 mg/100 g (32-53)
Concentrated orange juice: 225 mg/100 g (112-364)
Fresh grapefruit juice: 43 mg/100 g (35-54)
Packaged grapefruit juice: 36 mg/100 g (31-43)
Fresh raspberry juice: 25 mg/100 g (12-44)
Raspberry syrup: 16 mg/100 g (12-21)
Elderberry juice: 26 mg/100 g
Redcurrant nectar: 6 mg/100 g (3-10)
Blackcurrant nectar: 30 mg/100 g (20-48)
Mandarin juice: 32 mg/100 g (31-32)
Granadilla juice: 30 mg/100 g (20-40)
Sea buckthorn berry juice: 266 mg/100 g (111-664)
Grape juice: 1.70 mg/100 g (0.8-2.9)
Lemon juice: 53 mg/100 g (46-62)

Fruit and berry preserves:

Orange preserves: 4 mg/100 g (1.7-7)
Apricot preserves: 1.1 mg/100 g
Blackberry preserves: 0.4 mg/100 g
Strawberry preserves: 5.8 mg/100 g (4.6-12.3)

Rosehip preserves: 51 mg/100 g (10-140)
Raspberry preserves: 2.7 mg/100 g (0.3-11)
Redcurrant preserves: 16 mg/100 g (12-24)
Cherry preserves: 1.2 mg/100 g (0-2.9)

Miscellaneous

Roots and tubers:

Horseradish: 114 mg/100 g (90-260)

Stems/flowers/leaves:

Cauliflower: 67 mg/100 g (57-124)
Green cabbage: 105 mg/100 g (60-392)
Brussels sprouts: 112 mg/100 g (73-152)
Broccoli: 100 mg/100 g (88-118); boiled and drained: 90 mg/100 g
Fennel: 93 mg/100 g (60-120)
Parsley: 161 mg/100 g (150-182)
Asparagus: 20 mg/100 g (5-33)
Spinach: 51 mg/100 g (15-120); boiled and drained: 29 mg/100 g
Tinned spinach: 14 mg/100 g (13-14)

Mushrooms:

oyster mushrooms: 600 micrograms per 100 g; ceps: 7 mg/100 g, cultivated mushrooms: 3 to 9 mg/100 g; tinned: 1 to 2 mg/100 g