Apricot

Species (Family)

Prunus armeniaca L. (Rosaceae)

Synonym(s)

None

Part(s) Used

Kernel (seed), expressed oil

Pharmacopoeial and Other Monographs

Martindale 32nd edition[G43]

Legal Category (Licensed Products)

Apricot is not included in the GSL. Amygdalin (a cyanogenetic glycoside) is classified as a POM.[1]

Constituents

Acids Phenolic. Various quinic acid esters of caffeic, *p*-coumaric and ferulic acids.[2] Neochlorogenic acid major in kernel, chlorogenic in fruit.[3]

Glycosides Cyanogenetic. Amygdalin (mandelonitrile-Cyanide content of kernel varies from 2 to 200 mg/100 g.[3]

Tannins Catechins, proanthocyanidins (condensed).[4]

Other constituents Cholesterol, an oestrogenic fraction (0.09%) containing oestrone (both free and conjugated) and α-oestradiol.[5]

Other plant parts

Leaves and fruit contain various flavonol (kaempferol, quercetin) glycosides including rutin (major).[5]

Food Use

Apricot fruit is commonly eaten. Apricot is listed by the Council of Europe as a natural source of food flavouring (category N1 and N2). These categories limit the total amount of hydrocyanic acid permitted in the final product to 1 mg/kg. Exceptions to this are 25 mg/kg for confectionery, 50 mg/kg for marzipan and 5 mg/kg for fruit juices.[G16] In the USA, apricot kernel extract is listed as GRAS (Generally Recognised As Safe).[G65]

Herbal Use

Traditionally, the oil has been incorporated into cosmetic and perfumery products such as soaps and creams.[G34]

Dosage

None documented. Traditionally, apricot kernels have not been utilised as a herbal remedy.

Pharmacological Actions

During the late 1970s and early 1980s considerable interest was generated in apricot from claims that laetrile (a semi-synthetic derivative of amygdalin) was an effective treatment for cancer. Two review papers[6,7] discuss these claims for laetrile together with its chemistry, metabolism and potential toxicity.

The claims for laetrile were based on three different theories. The first claimed that cancerous cells contained abundant quantities of β-glucosidases, enzymes which release hydrogen cyanide from the laetrile molecule as a result of hydrolysis. Normal cells were said to be protected because they contained low concentrations of β-glucosidases and high concentrations of rhodanese, an enzyme which converts cyanide to the less toxic thiocyanate. However, this theory was disproved when it was shown that both cancerous and normal cells contain only trace amounts of β-glucosidases, and similar amounts of rhodanese. In addition, it was thought that amygdalin was not absorbed intact from the gastrointestinal tract.[6,7]

The second theory proposed that following ingestion, amygdalin was hydrolysed to mandelonitrile, transported intact to the liver and converted to a β-glucuronide complex. This complex was then carried to the cancerous cells, hydrolysed by β-glucuronidases to release mandelonitrile and subsequently hydrogen cyanide. This theory was considered to be untenable.[7]

A third theory proposed that laetrile is vitamin B_{17}, that cancer is a result of a deficiency of this vitamin, and that chronic administration of laetrile

would prevent cancer. Again this was not substantiated by any scientific evidence.[7]

A retrospective analysis of the use of laetrile by cancer patients reported that it may have slight activity.[6,7] However, a subsequent clinical trial concluded that laetrile was ineffective in cancer treatment. Furthermore, it was claimed that patients taking laetrile reduced their life expectancy as a result of lack of proper medical care and chronic cyanide poisoning.[6,7]

In order to reduce potential risks to the general public, amygdalin was made a prescription-only medicine in 1984.[1]

Side-effects, Toxicity

Laetrile and apricot kernel ingestion are the most common sources of cyanide poisoning, with more than 20 deaths reported.[6,7] Apricot kernels are toxic because of their amygdalin content. Hydrolysis of the amygdalin molecule by β-glucosidases, heat, mineral acids or high doses of ascorbic acid (vitamin C) yields hydrogen cyanide (HCN), benzaldehyde, and glucose. β-Glucosidases are not generally abundant in the gastrointestinal tract, but they are present in the kernels themselves as well as certain foods including beansprouts, carrots, celery, green peppers, lettuce, mushrooms and sweet almonds. Hydrolysis of the amygdalin molecule is slow in an acid environment but much more rapid in an alkaline pH. There may therefore be a delay in the onset of symptoms of HCN poisoning as a result of the transit time from the acid pH of the stomach to the alkaline environment of the small intestine.

Acute poisoning

Cyanide is rapidly absorbed from the upper gastrointestinal tract, diffuses readily throughout the body and promptly causes respiratory failure if untreated. Symptoms of cyanide toxicity progress rapidly from dizziness, headache, nausea, vomiting and drowsiness to dyspnoea, palpitations, marked hypotension, convulsions, paralysis, coma and death, which may occur from 1 to 15 minutes after ingestion. Antidotes for cyanide poisoning include nitrite, thiosulfate, hydroxocobalamin, cobalt edetate and aminophenol.[6,7]

Chronic poisoning

Principal symptoms include increased blood thiocyanate, goitre, thyroid cancer, lesions of the optic nerve, blindness, ataxia, hypertonia, cretinism and mental retardation.[6] These symptoms may develop as a result of ingesting significant amounts of cyanide, cyanogenetic precursors in the diet, or cyanogenetic drugs such as laetrile. Demyelinating lesions and other neuromyopathies reportedly occur secondary to chronic cyanide exposure, including long-term therapy with laetrile. Agranulocytosis has also been attributed to long-term laetrile therapy.[6,7]

Individual reports of adverse reactions and cyanide poisoning in patients using laetrile have been documented.[G45]

Normally, low concentrations of ingested cyanide are controlled naturally by exhalation or by rapid conversion to the less toxic thiocyanate by the enzyme rhodanese. Oral doses of 50 mg of hydrogen cyanide (HCN) can be fatal. This is equivalent to approximately 30 g kernels which represents about 50–60 kernels, and approximately 2 mg HCN/g kernel. Apricot seed has also been reported to contain 2.92 mg HCN/g.[8] A 500-mg laetrile tablet was found to contain between 5 and 51 mg HCN/g.

There may be considerable variation in the number of kernels required to be toxic, depending on the concentration of amygdalin and β-glucosidases present in the kernels, the timespan of ingestion, the degree of maceration of the kernels, individual variation in hydrolysing, and detoxifying abilities.

Systemic concentrations of β-glucosidases are low and therefore toxicity following parenteral absorption of amygdalin is low. However, cyanide poisoning has been reported in rats following intraperitoneal administration of laetrile, suggesting another mechanism of hydrolysis had occurred.[6,7]

It is thought that cyanogenetic glycosides may possess carcinogenic properties. Mandelonitrile (amygdalin = mandelonitrile diglucoside) is mutagenic and stimulates guanylate cyclase.[6,7]

Contra-indications, Warnings

Apricot kernels are toxic due to their amygdalin content. Following ingestion hydrogen cyanide is released and may result in cyanide poisoning. Fatalities have been reported following the ingestion of apricot kernels. Contact dermatitis has been reported for apricot kernels.[9]

Pregnancy and lactation Apricot kernels are toxic and should not be ingested. The ingestion of cyanogenetic substances may result in teratogenic effects.[6] However, one case has been reported where no acute toxicity was noted in the infant when laetrile was used during the third term of pregnancy. It was unknown whether chronic effects would be manifested at a later date.[6] Breeding rats fed ground apricot kernels had pups with normal birth weights, but with lower survival rates and lower weaning weights.[3]

Pharmaceutical Comment

Interest in apricot kernels was generated as a result of claims in the late 1970s that laetrile, a semi-synthetic derivative of the naturally occurring constituent amygdalin, was a natural, non-toxic cure for cancer. Apricot kernels were seen as an alternative source for this miracle cure. These claims have since been disproved and it has been established that laetrile (amygdalin) is far from non-toxic, particularly if administered orally. Fatal cases of cyanide poisoning have been reported following the ingestion of apricot kernels.

References

See also General References G10, G16, G32, G34, G43 and G57.

1 The Medicines (Cyanogenetic Substances) Order, SI 1984 No. 187, London: HMSO, 1984.

2 Möller B, Herrmann K. Quinic acid esters of hydroxycinnamic acids in stone and pome fruit. *Phytochemistry* 1983; 22: 477–481.

3 Miller KW *et al.* Amygdalin metabolism and effect on reproduction of rats fed apricot kernels. *J Toxicol Environ Health* 1981; 7: 457–467.

4 Awad O. Steroidal estrogens of *Prunus armeniaca* seeds. *Phytochemistry* 1973; 13: 678–690.

5 Henning W, Herrmann K. Flavonol glycosides of apricots (*Prunus armeniaca* L.) and peaches (*Prunus persica* Batch). 13. Phenolics of fruits. *Z Lebensm Unters Forsch* 1980; 171: 183–188.

6 Chandler RF *et al.* Laetrile in perspective. *Can Pharm J* 1984; 117: 517–520.

7 Chandler RF *et al.* Controversial laetrile. *Pharm J* 1984; 232: 330–332.

8 Holzbecher MD *et al.* The cyanide content of laetrile preparations, apricot, peach and apple seeds. *Clin Toxicol* 1984; 22: 341–347.

9 Göransson K. Contact urticaria to apricot stone. *Contact Dermatitis* 1981; 7: 282.