CYANOGENIC GLYCOSIDES - *INFORMATION SHEET*

THE COMPOUNDS

Cyanogenic glycosides or cyanoglycosides account for approximately 90% of the wider group of plant toxins known as cyanogens. The key characteristic of these toxins is cyanogenesis, the formation of free hydrogen cyanide, and is associated with cyanohydrins that have been stabilised by glycosylation (attachment of sugars) to form the cyanogenic glycosides.

Examples of cyanogenic glycosides include linamarin from cassava and amygdalin from the seeds of stone fruit. The amount of cyanogenic glycosides in plants is usually reported as the level of releasable hydrogen cyanide.

SOURCES

The major edible plants in which cyanogenic glycosides occur are almonds, sorghum, cassava, lima beans, stone fruits and bamboo shoots.

A cyanogenic food of particular economic importance is cassava (*Manihot esculenta*), which is also known by the names manioc, yuca and tapioca. Cassava is by far the most important cyanogenic food crop for humans and is an important source of dietary energy in tropical regions. The predominant cyanoglycoside in cassava is linamarin. It is present in leaves and tubers, both of which are eaten. Linamarin is also present in beans of the lima or butter type.

Amygdalin is the cyanogenic glycoside responsible for the toxicity of the seeds of many species of Rosaceae, such as bitter almonds, peaches and apricots. Sweet almonds are low in amygdalin as a result of breeding processes. Their use in marzipan is common but the preparation procedure should eliminate most of the cyanide.

Cyanogen levels can vary widely with cultivar, climatic conditions, plant part and degree of processing. Typical levels for some plant materials consumed by humans are:

Food	Major cyanogenic glycoside present	Cyanogen content (mg HCN/kg)
Cassava (*Manihot esculenta*) - root	Linamarin	15-1000
Sorghum (*Sorghum vulgare*) – leaves	Dhurrin	750-790
Flax (*Linum usitatissimum*) – seed meal	Linamarin, linustatin, neolinustatin	360-390
Lima beans (*Phaseolus lunatus*)		2000-3000
Giant taro (*Alocasia macrorrhizos*) – leaves	Triglochinin	29-32
Bamboo (*Bambusa arundinacea*) – young shoots	Taxiphyllin	100-8000
Apple (*Malus* spp.) – Seed	Amygdalin	690-790
Peach (*Prunus persica*) – Kernel	Amygdalin	710-720
Apricot (*Prunus armeniace*) – Kernel	Amygdalin	785-813 89-2170 2.2 (juice)
Plum (*Prunus* spp.) – Kernel	Amygdalin	696-764
Nectarine (*Prunus persica var nucipersica*) – Kernel	Amygdalin	196-209
Cherry (*Prunus* spp.)	Amygdalin	4.6 (juice)
Bitter almond (*Prunus dulcis*)	Amygdalin	4700

(Haque and Bradbury, 2002)
(Simeonova and Fishbein, 2004)
(Shragg *et al*., 1982)

HUMAN HEALTH EFFECTS

Potential toxicity of cyanoglycosides arises from enzymatic degradation to produce hydrogen cyanide, resulting in acute cyanide poisoning. The enzyme responsible (β-glucosidase) may arise from the plant material or from gut microflora. Clinical symptoms of acute cyanide poisoning include rapid respiration, drop in blood pressure, rapid pulse, headache, dizziness, vomiting, diarrhoea, mental confusion, stupor, blue discolouration of the skin due to lack of oxygen (cyanosis), twitching and convulsions.

Cyanide can be lethal to humans and the acute dose is in the region of 1 mg/kg body weight. Cases of acute poisoning have been associated with misuse, particularly of preparations from apricot pits, bitter almonds and cyanide rich apple seeds.

Cyanide is detoxified in the body, by the enzyme rhodanese in the presence of sulphur-containing amino acids, to produce thiocyanate. Goitre and cretinism due to iodine deficiency can be exacerbated by chronic consumption of insufficiently processed cassava. The detoxification product of cyanide, thiocyanate, is a similar size to the iodine molecule and interferes with iodine uptake by the thyroid, effectively increasing the dietary requirement for iodine. The effect is only seen in iodine deficient population and can be reversed by iodine supplementation.

Neurological effects:

- Konzo or spastic paraparesis is a motor neuron disease characterised by irreversible weakness in the legs. In severe cases, patients are not able to walk, and speech and arms may be affected. Konzo particularly affects

children and women of childbearing age in East Africa in times of food shortage and is associated with a high and sustained intake of cassava (*Manihot esculenta*) in combination with a low intake of protein (Davis, 1991; FSANZ, 2004).

- Tropical ataxic neuropathy (TAN) describes several neurological symptoms effecting the mouth, eyesight, hearing or gait of mostly older males and females. TAN is attributed to cyanide exposure from the chronic consumption of foods derived from cassava (FSANZ, 2004).

Although strong associations have been observed between chronic cassava consumption and these diseases, the observations are confounded by diverse nutritional deficiencies and a causal relationship has not been conclusively established (Davis, 1991; FSANZ, 2004; Speijers, 1993).

There are two known reports of cyanide poisoning in New Zealand from the consumption of apricot kernels. In one case a woman was admitted to North Shore hospital after consuming 60 ground apricot kernels mixed with orange juice (Atkinson, 2006). In an earlier case, reported by Waikato hospital (Tebbutt, 2001), 30 apricot kernels containing 3 mg cyanide/g kernel caused a significant poisoning.

Effects arising from chronic consumption of cyanogenic foods are not likely to be an issue for the general population in New Zealand since food security and dietary intake of protein is adequate and neither cassava, nor other cyanogenic foods are staples of the general diet.

ESTIMATES OF DIETARY EXPOSURE

No formal estimates of dietary exposure to cyanogenic glycosides are available.

Cassava consumption in New Zealand is relatively low. Fresh imports are mainly from the Pacific Islands (Fiji, Tonga). The varieties of cassava grown in Pacific Island countries contain low levels of cyanogenic glycosides (FSANZ, 2004). There is little evidence for consumption of bamboo shoots in New Zealand. Consequently, dietary exposure to cyanogenic glycosides in New Zealand is likely to be low.

FACTORS INFLUENCING RISK

Food processing procedures such as soaking, fermentation or drying will reduce the levels of hydrogen cyanide before consumption through the action of plant enzymes and subsequent leaching. Processes such as grating, soaking, fermentation and storage will allow time for conversion of cyanogenic glycosides to cyanide, while exposure to air or water will allow the cyanide to dissipate out of the food matrix.

Soaking: Soaking of cassava root resulted in a decrease in total cyanogen content of 13-52% after 24 hours, 73-75% after 48 hours and 90% after 72 hours ((Agbor-Egbe and Lape Mbome, 2006; Kendirim *et al.*, 1995).

Fermentation: Fermentation of cassava pulp or dough for 4-5 days results in a decrease in total cyanogens of 52-63% (Kendirim *et al.*, 1995; Obilie *et al.*, 2004). Soaking and fermentation of bitter apricot seeds decreased cyanogen levels by about 70% (Tuncel *et al.*, 1990)

Storage: storage of gari, a traditional African cassava meal product, for four weeks resulted in a decrease in total cyanogen content of 50-64% (Onabolu *et al.*, 2002).

Cooking: Boiling of cassava leaves in water with added palm oil resulted in a decrease in cyanogen levels of 96->99% (Ngudi *et al.*, 2003). Cooking of various cassava products (baton de manioc, fufu) resulted in reduction of the total cyanogen content of 32-55% (Agbor-Egbe and Lape Mbome, 2006). Steaming of another cassava product (akyeke) resulted in a 74-80% reduction in total cyanogen levels (Obilie *et al.*, 2004). 'Garification', a process whereby fermented and dried cassava mash is simultaneously cooked and dried in a shallow wok, resulted in a 90-93% reduction in total cyanogen content (Agbor-Egbe and Lape Mbome, 2006). Optimal cooking conditions for reduction of cyanogen levels in bamboo shoots (98-102ºC for 148-180 minutes) resulted in a 97% reduction in cyanogens (Ferreira *et al.*, 1995).

Overall, traditional African processes typically decrease the cyanogen content of cassava by 97->99%.

SAFETY ASSESSMENTS

Cyanogenic glycosides were assessed by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) in 1993 (Speijers, 1993), by Food Standards Australia New Zealand (FSANZ) in 2004 (FSANZ, 2004) and by the International Programme on Chemical Safety (IPCS) in 2004 (Simeonova and Fishbein, 2004).

None of these assessments established a safe level of exposure to cyanogenic glycosides, mainly due a lack of quantitative toxicological and epidemiological information.

SAFETY AND REGULATORY LIMITS

Safety limits are levels of dietary exposure that are without appreciable risk for a lifetime of exposure. Regulatory limits define the maximum amount of a substance that is permitted in a particular food.

Source	**Limit Type**	**Limit**
Safety Limits		
No safety limits have been set for cyanogenic glycosides		
Regulatory Limits		
Australia New Zealand Food Standards Code	Maximum Level	Total hydrocyanic acid*#: Confectionery 25 mg/kg Stone fruit drinks 5 mg/kg Marzipan 50 mg/kg Alcoholic beverages 1 mg/kg per 1% alcohol content

*Total hydrocyanic acid refers to the total potential of the food to form hydrocyanic acid, the acid form of cyanide.
#A maximum level of total hydrocyanic acid in ready-to-eat cassava chips is currently under review by FSANZ (proposal P1002)

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