GLUCOSINOLATES - *INFORMATION SHEET*

THE COMPOUNDS

Glucosinolates are a family of about 120 plant compounds. They are modified amino acids, carrying an S-glucose functional group and a variety of different side chains. The parent compounds can be broken down by a plant enzyme, myrosinase, which is liberated for reaction through processing of the plant tissue (e.g. cutting, cooking or freezing). Breakdown of glucosinolates produces a variety of bioactive products, including isothiocyanates, oxazolidine-2-thiones, nitriles, thiocyanate ion, and indole products (Cartea and Velasco, 2008; Fahey *et al.*, 2001).

SOURCES

The species in which glucosinolates occur that are important for animal or human feeding belong to the family Brassicaceae. These include: rapeseed, cabbage, cauliflower, Brussels sprouts, swede/turnip, calabrese/broccoli and Chinese cabbage, radishes, mustard seed and horse radish. Consistently high levels (>100 mg/100 g fresh weight) have been reported in Brussels sprouts, cress and mustard greens (McNaughton and Marks, 2003).

POTENTIAL HEALTH EFFECTS

The main health concern due to the presence of these compounds in foods is the potential for increase in goitre (enlargement of the thyroid gland) (Heaney and Fenwick, 1995; Tripathi and Mishra, 2007). Goitre is mainly due to a dietary deficiency of iodine but the differing prevalence of goitre in areas with low levels of iodine indicates that other factors are involved, one of which is the presence of goitrogens in the diet. Isothiocyanates and oxazolidine-2-thiones have been shown to have effects on the thyroid gland in animals and these effects occur even if iodine is sufficient. Isothiocyanates, nitriles and indole products can all be further degraded to produce the thiocyanate ion. This compound is able to cause goitre in humans when dietary iodine is insufficient. However, despite several decades of conjecture, there is no evidence to support a causative role for dietary glucosinolates in human goitre (Cartea and Velasco, 2008; Heaney and Fenwick, 1995; McMillan *et al.*, 1986; Shapiro *et al.*, 2006).

Glucosinolates may also have beneficial effects. They have been shown to induce the activity of the phase II detoxification enzymes and inhibit phase I (activating) enzymes, exerting an anticancer effect (Cartea and Velasco, 2008; Jeffery and Araya, 2009; Johnson, 2002). They have also been shown to provide protection from oxidative stress through elimination of reactive oxygen species (Jeffery and Araya, 2009; Traka and Mithen, 2009). It is thought that this activity may be behind the observed link between vegetable (Brassica) intake and cancer protective effects (London *et al.*, 2000; Verhoeven *et al.*, 1996).

On balance, there is greater evidence for a positive impact of glucosinolates on human health than a negative impact. However, strong evidence for a causal relationship with human positive or negative health effects is currently lacking.

ESTIMATES OF DIETARY EXPOSURE

Calculations of dietary exposure are limited by the shortage of reliable data on levels in plants and also the variation in natural levels between seasons and different strains. Exposure to total glucosinolates by adult New Zealanders was estimated to be 17 mg/day, with approximately one-third of glucosinolate exposure due to consumption of cabbage (Cressey and Thomson, 2007).

UK estimates based on daily intakes of cabbage, cauliflower, Brussels sprouts and swede/turnip were 46 mg/day if all vegetables are considered to have been consumed fresh/raw and 29 mg/day if all vegetables are considered to be consumed cooked (Sones *et al.*, 1984). A German study gave similar estimates of 46 and 36 mg/day for winter and summer respectively (Holst and Williamson, 2004).

FACTORS INFLUENCING RISK

The effects of normal dietary levels of intake of glucosinolates are not known and there is little reliable evidence of harmful dose levels in human beings. Cooking by boiling will reduce exposure through leaching of glucosinolates into water (Fahey *et al.*, 2001; McNaughton and Marks, 2003; Song and Thornalley, 2007) and inactivation of myrosinase (McMillan *et al.*, 1986). However, steaming, microwaving or stir-frying results in no significant decrease in glucosinolate concentrations (Song and Thornalley, 2007).

SAFETY ASSESSMENTS

The safety of glucosinolates in the human diet has not been assessed by any national or international organisation.

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 glucosinolates		
Regulatory Limits		
No regulatory limits have been set for glucosinolates in New Zealand or Australia		

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