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Cyanogenetic Glycosides

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Abstract
Anti-nutritional factors present in plant feedstuffs are a major limiting factor for its usage as feedstuff in larger amount and also its intake by the animals. Among various anti nutritional factors cyanogenetic glycosides (CG) is one important factor whose presence in plant feedstuffs causes toxicity in animals. This article discusses in detail about the structure, biosynthesis, effect and toxicity of CG in animals and also the processing methods to reduce the amount of this anti nutritional factor in the plant feedstuff.

Keywords: Cyanogenetic Glycosides, Hydrogen Cyanide, Cassava

1. Introduction
CG are defined chemically as glycosides of alpha hydroxinitriles and belong to the secondary metabolites of plants (Vetter, 2000)[1]. They are actually amino acid derived plant constituents. Major amino acids include valine, leucine, isoleucine, phenyl alanine and tyrosine. CG's are present in plant families like Compositae, Linaceae, Papaveraceae, Rosaceae, Polypodiaceae, Fabaceae, Poaceae etc. CG's are present in more than 2500 plant species. Hydrolysis of CG results in the formation of hydrogen cyanide (HCN) and it was first isolated from plants in 1802 by Scrade. HCN formation occurs during the process of chewing or digestion of CG.

2. Structure
Structure of CG consists of two parts ie an aglycone part and a sugar moiety. Aglycone part (alpha hydroxinitriles) occurs as aliphatic, aromatic and free hydroxynitrile substituents. Sugar moiety includes D-glucose, gentibiose and primeverose. Details about the structure of some important CG's are given below.

Table 1: Chemical structure of cyanogenetic glycosides

	Substituents	Glycoside	Sugar	Occurrence
Aliphatic substituents	R=R'=CH3	Linamarin	D- glucose	*Linum spp. Trifolium spp.*
	R=CH3, R'=CH3CH2-	Lotaustralin	D-glucose	*Lotus spp. Manihott spp.*
Aromatic substituents	Phenyl	Prunasin	D-glucose	*Prunus spp.*
	Phenyl	Amygdalin	Gentiobiose	*Prunus spp.*
	p hydroxyphenyl	Dhurrin	D-glucose	*Sorghum spp.*
Glycosides with free alpha hydroxynitrile		P-glucosyloxymandalonitrile P-glucosyl oxymandalonitrile		*Nandina spp.*

3. Biosynthesis of CG's
The general pathway of biosynthesis of cyanogenic glycosides was given by Tapper and Reay (1973) [2]. Alpha aminoacids are hydroxylated to form N hydroxylaminoacid. It is then converted to an aldoxime. Aldoxime get converted to nitrile. These nitriles get hydroxylated to form alpha hydroxynitrile. Glucosylation of alpha hydroxynitrile results in the formation of CG.

4. Cyanogenesis

The ability of certain organisms, plants to produce hydrocyanic acid (HCN, Prussic acid) is called as cyanogenesis. Cyanogenesis is a defence mechanism by which the plant protects itself from the organism damaging or feeding plant tissues by the release of chemical compound HCN which is highly toxic. Two enzymes are important in the process of cyanogenesis and they are beta glucosidases and hydroxynitrile lyase. Beta glucosidases releases the sugar and hydroxynitrile lyase releases HCN from plants. Glycoside is present in the epidermis and enzymes are present in the mesophyll, so there won't be any release of HCN from the intact tissue. During the time of digestion or chewing these parts come together and they react to release the toxic HCN. In presence of enzyme beta glucosidase, these compounds lose their sugar part to form cyanohydrins. Cyanohydrin in presence of water undergoes hydrolysis under the influence of enzyme hydroxynitrile lyase to form benzaldehyde and the toxic HCN.

5. Effect of HCN

The normal metabolic pathway of HCN is that it will be eliminated through lungs. HCN exerts its toxic action through three ways. First one is through the formation of thiocyanate. Detoxification process of HCN occurs in the liver by conversion of HCN to thiocyanate, which is a goitrogen with the help of enzyme rhodanase. Major toxicity action of HCN is through its inhibitive role on electron transport system (ETS). HCN inhibits the enzyme cytochrome oxidase in the final step of ETS, which is the transfer of electron to oxygen. This leads to cell death and if the concentration is very high, it causes even the death of the organism. Third way is through the combination of HCN with haemoglobin (HB) to form cyanHb. So Hb losses its power to transport oxygen.

Table 2: HCN content in different plants

Plant	Part	Major CG present
Cassava	Root	Linamarin
Sorghum	Leaves	Dhurrin
Flax	Seed meal	Linamarin, Linustatin, Neolinustatin
Bamboo	Young shoots	Taxiphyllin
Apple	Seed	Amygdalin
Peach	Kernel	Amygdalin
Apricot	Kernel	Amygdalin
Plum	Kernel	Amygdalin
Cherry		Amygdalin
Bitter almond		Amygdalin

(Modified from Simeonova and Fishbean, 2004) [3]

6 Toxicity in animals

6.1 Cattle

Lethal dose of HCN for cattle and sheep is about 2-4 mg/kg bwt. Lethal dose of cyanogens is 20 to 30 times greater because HCN comprises 5 to 10% of their molecular weight (Kumar, 1992) [4]. Ruminants are more sensitive to HCN poisoning because enzymes for release of HCN were destroyed by gastric HCL in horses and pigs. General symptoms noticed in cattle include anorexia, weakness, depression, circling, excessive salivation, vomiting, respiratory distress, muscular tremor and tenesmus. Acute onset of poisoning causes ruminal stasis, bright red mucus membrane, scant tarry faeces and finally death (Sargison *et al*., 1996) [5]. Post mortem examination shows congestion and petechial haemorrhage of heart, liver, kidney, rumen and intestine. *Invitro* studies done by Tanyildizi and Bozkurt (2004) [6] found that incubation of semen samples with linamarin and amygdalin caused significant decreases in spermatozoa motility and hyaluronidase activity. So it shows the toxic effect of HCN on semen activity.

6.2 Sheep and Goat

Phalaris aquatia feeding of sheep results in a condition known as sudden death syndrome. Potentially toxic levels of HCN (20-36 mg/100g) are present in *Phalaris aquatia* pastures (Bourke and Carrigan, 1992) [7]. CG decrease selenium status and increases susceptibility to nutritional myopathy. Jayasekhara and Horadagoda (1992) [8] found that HCN increases the WBC count in goats. Presence of amygdalin in feed found to decrease the feed intake in sheep (Burritt and Provenza, 2000) [9].

6.3 Horse and Pig

Incordination, frequent urination, urinary incontinance and haematuria were noticed in horses. No effect on weight gain, feed intake, FCE and protein efficiency in pigs at 0, 239 and 419 mg/kg HCN through Cassava (Iyayi and Tewe, 2004) [10]. Fetal malformations noticed in wild blackcherry feeding in pigs.

6.4 Poultry

Level of cassava meal over 10-15% in the diet of chicks found to reduce the weight gain. Adult hens are more tolerant to HCN compared to younger ones (Vetter, 2000) [1].

6.5 Dogs

In dogs HCN causes increased plasma thiocyanate concentration, urinary protein excretion and lowered serum albumin. It is found to be diabetoginic.

7 Processing methods

Different types of processing methods are used to reduce the CG content in plants. Most important processing methods include peeling, soaking, boiling, fermentation, ensiling and drying.

Peeling of cassava reduces the cyanide content by 50% as peel contains 650 ppm and pulp contains 310 ppm cyanide. Soaking is a processing method which reduces free cyanide content by 20%. Variation of soaking technique exists which is known as retting. Sun drying after retting reduces cyanide content by 98.6% (Ayenor, 1995) [11]. Boiling/Cooking can reduce free cyanide content by 96% within 15 minutes. After heating for 25 minutes bound cyanide get reduced by 55%. Boiling can destroy linamarase enzyme at 72 °C, thereby prevent the formation of linamarin. Ensiling reduces cyanide content by 36% and also it results in disintegration of intact glucoside (Gomez and Valdivieso, 1988) [12]. Drying is of two types - sun and electric drying. Sun drying reduces HCN content by 86%.

8 Estimation assays

Estimation of CG can be done by different techniques like alkaline picrate paper colour reaction, linamarin in beans by FID gas chromatography**,** taxiphyllin content of bamboo by HPLC and air steam distillation alkaline titration**,** modified colourimetric method (Vetter, 2000) [1] etc.

9 Treatment for CG toxicity in animals

In cattle normally intra venous injection of sodium nitrite (10g/100 ml of distilled water or isotonic saline) at 20 mg/kg

b. wt can be given. Sodium thiosulphate (20%) I/V at 500 mg/kg B.WT also had to be given along with sodium nitrate for better effect. For sheep, 1g sodium nitrite and 2.5 g sodium thiosulphate in 50 ml water can be given intra venously. For dogs dimethyl amino pyridine (DMAP) can be given intra muscularly at 5 mg/kg B.WT. Hydroxy cobalamine and Dicobalt EDTA can also be provided.

10 References

1. Vetter J. Plant cyanogenetic glycosides Toxicon 2000; 38:11-36.
2. Tapper BA, Reay PF. Cyanogenic glycosides and glucosinolates Chemistry and Biochemistry of herbage, 1973.
3. Simeonova FP, Fishbein L. Hydrogen cyanide and cyanides: Human health aspects. Concise International Chemical Assessment Document 61 Geneva: World Health Organization, 2004.
4. Kumar R. Anti-nutritional factors, the potential risks of toxicity and methods to alleviate them. In Legume trees and other fodder tree as protein sources for livestock. A Speedy and P Pugliese (Editors) FAO, Animal Production and Health Paper 1992; 102:145-160.
5. Sargison ND, Williamson DS, Duncan JR, McCance RW. Prunuspadus (Bird cherry) poisoning in cattle Vet Record 1996; 138(8):188.
6. Tanyildizi S, Bozkurt T. In vitro effects of Linamarin, Amygdalin and Gossypol Acetic Acid on Hyaluronidase Activity, Sperm Motility and Morphological Abnormality in Bull Sperm, In Turkish Journal of Veterinary and Animal Sciences. 2004; 28:819-824.
7. Bourke CA, Carrigan MJ. Mechanism underlaying Phalaris aquatica `sudden death' syndrome in sheep. Australian Veterinary Journal. 1992; 69:165-167.
8. Jayasekara S, Horadagoda NU. Hydrocianic acid content in Manihot Glaziovii (Cearra Rubber) Leaves and Toxicologic Effects in Goats, Sri Lankan Journal of Agricultural Sciences. 1992; 29:9-15.
9. Burritt EA, Provenza FD. Role of toxins in intake of varied diets by sheep, J Chem. Ecol. 2000; 26:1991.
10. Iyayi EA, Tewe OO. Effect of protein deficiency on utilization cassava peels by growing pigs. Cassava as livestock feed in Africa- Proceedings of the IITA/ILCA/ University. 2004.
11. Ayenor GA. Effect of retting of cassava on product yield and cyanide detoxification, Journal of Food Technology. 1995; 20:89-96.
12. Gomez G, Valdivieso M. The effects of ensiling cassava whole-root chips on cyanide elimination Nutrition Reports International 1988; 37:1161-1166.