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Glucosinolates

Syndrome names:

- Goitre including congenital goitre
- Atypical interstitial pneumonia
- Rumenitis
- Fatty haemorrhagic liver syndrome of poultry (suspected association with glucosinolates)
- Perosis (lateral slipping of tendon from hocks in poultry) (Cheeke 1998)

See sinapine under Amino Acids & Proteins for information on egg taint from rapeseed or canola meal.

Chemical structure:

Glucosinolates are glycosides of β -D-thioglucose (Cheeke 1998). Crushing (including mastication) of fresh glucosinolate-containing plant material releases the enzyme thioglucosidase (myrosinase) which hydrolyses glucosinolates to glucose, hydrosulphuric acid ion and a sulphur- and-nitrogen-containing aglycone (Seawright 1989). Further hydrolysis of the aglycone yields a thiocyanate, an isothiocyanate or an organic nitrile (Seawright 1989). Intestinal bacteria also contain thioglucosidase and may contribute to hydrolysis of glucosinolates (Seawright 1989). Each plant contains several different glucosinolates and thus may produce a variety of such products, the amount and type formed depend on the parts of the plants involved and how they are treated (Seawright 1989).

Sources:

Plant sources:

Family Brassicaceae (Cruciferae)

Brassica spp.

Brassica juncea (Indian mustard, leaf mustard) (Ev210, 891)

Brassica napobrassica (swede turnip, rutabaga) (Ev210, 891)

Brassica napus (rape, canola) (Ev210, 891)

Brassica oleracea var. *acephala* (common kale, chou moellier) (Ev212, 891)

Brassica oleracea var. *botrytis* (broccoli, cauliflower) (Ev212, 891)

Brassica oleracea var. *capitata* (cabbage) (Ev212, 891)

Brassica oleracea var. *gemmifera* (Brussels sprouts) (Ev212, 891)

Brassica pekinensis (Chinese cabbage) (Ev213, 891)

Brassica rapa (turnips) (Ev213, 891)

Brassica tournefortii (wild turnip) (Ev213, 891)

Crambe abyssinica L. (crambe, Abyssinian kale)

Sinapis spp. (mustards) (?)

Sinapis alba [= *Brassica hirta*] (white mustard) (?) (Ev215, 914)

Sinapis arvensis [= *Brassica sinapistrum*, *Brassica kaber*] (charlock) (?) (Ev215, 914)

Raphanus raphanistrum (wild radish)

Rapistrum rugosum (turnip weed) - contains cheirolin (Stocks *et al.* 1984)

Family Limnanthaceae

Limnanthes alba Hartweg ex Benth. (meadowfoam) - native to western North America

Meals produced as by-products of oil production from seeds ("oil seeds") of *Brassica napus* (rape, canola), *Sinapis* spp. (mustards) and *Crambe abyssinica* (crambe).



Brassica napus (canola) crop, Western Australia. [RAM Photo]



Brassica napus inflorescence (flowers, immature fruits) [RAM Photo]

Toxicity:

Goitre

Several derivatives of glucosinolates are **goitrogenic** (goitrin, cheirolin, thiocyanates, isothiocyanates). Pigs and poultry are susceptible to goitre from dietary glucosinolates. Sheep and goats are susceptible to congenital goitre.

Atypical interstitial pneumonia

Indole glucosinolates (glucobrassicin)

Cattle only are affected by atypical interstitial pneumonia.

Rumenitis

Organic isothiocyanates

Cattle

Fatty haemorrhagic liver syndrome of poultry

Organic nitriles are suspected to be involved

Laying hens

Other effects

Poultry may develop perosis (slipped hock tendons) and lowered egg production and pigs may develop enlarged livers when fed rapeseed meal (Cheeke 1998).

Glucosinolates and their derivatives will cross the placenta (causing congenital goitre) and be secreted in milk.

Tissue residues of glucosinolate derivatives have been reported in cattle fed crambe meal (Van Etten *et al.* 1977).

Cruciferous vegetables (cabbage, broccoli, cauliflower *etc.*) have demonstrated protective effects against carcinogenesis, particularly against colonic neoplasia, and cholesterol-lowering effects. These effects are believed to be mediated through metabolites of glucosinolates, namely indole-3-carbinol (anticancer and cholesterol-lowering) and sulforaphane (anticancer). (Waldron *et al.* 1993; Stoewsand 1995).

Glucosinolates and their derivatives in roots can function to suppress the growth of soil-borne fungal pathogens of plants and *Brassica* spp. have been suggested as intercrops between grain crops to provide an alternative to the use of chemicals such as methyl bromide as soil fumigants, a process termed biofumigation (Kirkegaard & Sarwar 1999).

Organ systems affected:

Thyroid glands

Lungs

Upper gastrointestinal tract

Liver

Reproductive

Locomotary

Mode of action:

Goitre

Goitrins (oxazolidine-2-thiones) inhibit incorporation of iodine into thyroxine precursors and interfere with thyroxine secretion, actions that are not reversed by increased dietary iodine (Cheeke 1998).

Thiocyanates and **isothiocyanates** inhibit iodine uptake by the thyroid glands, an effect most pronounced when the iodine content of the diet is low and which can be reversed by increased dietary iodine (Cheeke 1998).

Nitriles from rapeseed meal may be partly converted to thiocyanate (Paik *et al.* 1980).

Atypical interstitial pneumonia

Glucosinolates hydrolysed in the alimentary tract form 3-hydroxymethylindole which can produce 'atypical interstitial pneumonia' similar to the effect of 3-methylindole (*q.v.*) derived from tryptophan (Gonzales *et al.* 1986).

Rumenitis

Organic isothiocyanates are very irritant to tissues which they contact, injuring blood vessel integrity (Seawright 1989).

Fatty haemorrhagic liver syndrome of poultry

Undescribed

Perosis

Undescribed

Conditions of poisoning:

Goitre

Low dietary iodine predisposes to congenital goitre (Seawright 1989). Goitre has been reported in lambs of ewes fed turnips (*Brassica rapa*) (Hercus & Purves 1936) and lambs of ewes and kids of goats fed raw meadowfoam meal (*Limnanthes alba*) (Throckmorton *et al.* 1981, White & Cheeke 1983). Sheep and goats are sporadically affected by congenital goitre after grazing *Rapistrum rugosum* in southern Queensland. Experimental feeding of flowering *Rapistrum rugosum* plants to adult sheep caused reduced serum thyroxine concentrations (Stocks *et al.* 1984).

Atypical interstitial pneumonia

Cattle grazing lush glucosinolate-containing crops or weeds

Rumenitis

Cattle fed old, non-viable seed of chou moellier (*Brassica oleracea* var. *acephala*) with glucosinolate content of 9.8 mg/g (Mason & Lucas 1983).

Fatty haemorrhagic liver syndrome of poultry

The syndrome is reported in laying hens fed a diet containing rape seed (Yamashiro *et al.* 1975), but there is contrary evidence (Wight *et al.* 1987).

Clinical signs:

Goitre

Enlarged thyroid glands

Growth depression

Atypical interstitial pneumonia (Cote 1944)

Onset 7-10 days after first access

dyspnoea (head and neck extended, open mouth, frothing at the mouth, grunting)

± ruminal atony, constipation

± diarrhoea

± jaundice

± subcutaneous emphysema

Rumenitis

Signs of abdominal pain, collapse and rapid death after access to source

Fatty haemorrhagic liver syndrome of poultry

Sudden death of hens in full lay

Perosis

Lameness and recumbency

Pathology:

Goitre

Hypertrophy and hyperplasia of the thyroid glands

Atypical interstitial pneumonia

Pulmonary oedema and emphysema (interstitial)

± subcutaneous emphysema

± liver necrosis (presumably hypoxic in origin)

Rumenitis

Acute rumenitis with marked oedema of the wall attributed to damage to submucosal and intramuscular blood vessels (Mason & Lucas 1983).

Fatty haemorrhagic liver syndrome of poultry

Hens in lay in fat condition with swollen fatty livers

Pale carcass tissues

Haemorrhage from or within the liver ± blood in the body cavity

Perosis

Lateral/medial displacement of the tendon at the hocks with lateral displacement of the lower limb(s)

Diagnosis:

Goitre

Syndrome + pathology + access to sources either directly or through placenta/milk

Atypical interstitial pneumonia

Syndrome + access to lush source plants

Rumenitis

Differential diagnosis should include other forms of chemical rumenitis such as inorganic arsenic (*q.v.*), cucurbitacins (*q.v.*)

Fatty haemorrhagic liver syndrome of poultry

Syndrome + Necropsy findings

Therapy: Nil

Prevention & control:

Non-ruminants can eat 5-10% rapeseed meal in the diet without ill effect. Ruminants can tolerate at least 10% (Cheeke 1998). Canola [= **Canadian oil low acid**] was developed in Canada to have significantly reduced concentrations of deleterious compounds and is the registered name of the rapeseed containing less than 2% of total fatty acids in the oil as erucic acid and less than 30 μ moles alkenyl glucosinolates/g of oil-free dry matter (Cheeke 1998). Residual glucosinolates in canola are mostly of the indole type which are not goitrogenic, but do yield nitriles (Bell 1993). Very low glucosinolate (VLG) canola, virtually equivalent as a protein supplement to soyabean meal, has been developed (Bell 1993).

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Aliphatic nitro compounds (Nitrotoxins)

Syndrome names:

- cracker heels (USA) (chronic intoxication)
- [See also Birdsville horse disease]

Compounds: (Stermitz & Yost 1978, Majak & Pass 1989, Moller & Seigler 1999)

- primary nitrocompounds have the general formula RCH_2NO_2 , in which the nitro (NO_2) group is linked through nitrogen to the α -carbon [aliphatic compounds are carbon compounds with the carbon atoms in chains rather than rings]
- in higher plants, the primary nitro group occurs in 4 aliphatic systems, namely
 - 3-nitro-1-propionic acid (NPA)**
 - 3-nitro-1-propanol (NPOH)**
 - 1-phenyl-2-nitroethane
 - 1-(4'-hydroxyphenyl)-2-nitroethane

the most common glycoside of NPOH is **miserotoxin** (3-nitro-1-propyl- β -D-glucopyranoside)

Sources:

Flowering plants

Family Fabaceae (Leguminosae):

- ***Astragalus* spp.** (North & South America, Europe, Asia) – NPA & NPOH derivatives
 - nitro compounds detected in over 450 species and varieties of *Astragalus* from North & South America, Europe and Asia
 - 14-22% of *Astragalus* spp. in western USA contain nitrocompounds; nitro content can exceed 30 mg NO₂ / g plant dry matter
 - NPA and NPOH derivatives do not usually occur together in the one species; an exception is *Astragalus palenae* toxic to stock in Argentina
 - *Astragalus miser* varieties *oblingifolius*, *serotinus* and *hylophilus* (timber milk vetch, Columbia milkvetch, Wasatch milkvetch) were early-recognised sources; source of the trivial name, miserotoxin
- *Lotus* spp. (birdsfoot trefoils) – NPA derivatives in 17 species
 - *Lotus pedunculatus* (large birdsfoot trefoil); UK (Simpson *et al.* 1999); poisoning with nitro-compounds under natural conditions not reported
- *Coronillia varia* (crown vetch) – NPA derivatives; poisoning of ruminants with nitro-compounds under natural conditions not reported; monogastrics possibly poisoned
- *Indigofera* spp. (indigos) – NPA derivatives in 65 species; poisoning with nitro-compounds under natural conditions not reported [see notes on Birdsville horse disease]
- *Hippocrepis comosa* (horseshoe vetch); UK (Simpson *et al.* 1999) – NPA derivative

Family Annonaceae

- *Annona squamosa* (sweetsop, custard apple) – 1-(4-hydroxyphenyl)- β -D-primeveroside
- *Dennettia tripetala* – 1-phenyl-2-nitroethane; monotypic genus, Nigeria

Family Corynocarpaceae – NPA derivatives

- *Corynocarpus laevigatus* (karak tree); New Zealand
- *Corynocarpus similis*

Family Lauraceae

- *Aniba canelilla* – 1-phenyl-2-nitroethane; South America
- *Ocotea pretiosa* – 1-phenyl-2-nitroethane

Family Malpighiaceae – NPA derivatives

- *Hiptage benghalensis*
- *Hiptage mandablota*
- *Heteropteris* spp. – 13 species including *H. angustifolia*
- *Janusia* spp. – 2 species

Family Papaveraceae

- *Eschscholtzia californica* (Californian poppy) – glucoside of 1-(4-hydroxyphenyl)-2-nitroethane (HPNE)

Family Ranunculaceae

- *Thalictrum aquilegifolium* (a meadow rue) – glucoside of 1-(4-hydroxyphenyl)-2-nitroethane (HPNE)

Family Rutaceae

- *Citrus unshiu* – 1-phenyl-2-nitroethane

Family Tropaeolaceae

- *Tropaeolum majus* (nasturtium) – 1-phenyl-2-nitroethane

Family Violaceae – NPA derivatives

- *Viola odorata*

Fungi

- *Penicillium atrovirens* – synthesises NPA

- *Aspergillus oryzae* and *A. soyae* produce NPA in fermented foods

Toxicity (vertebrates):

- NPA or a metabolite of NPA is the primary toxic agent, not nitrite
- NPOH and miserotoxin not toxic themselves, requiring conversion to NPA
- toxic nitro-bearing *Astragalus* spp. typically contain up to 20 mg NO₂ / g plant; toxin concentrated in leaves, some species contain up to 80 mg NO₂ / g plant (Williams & James 1978)
- **cattle, sheep**, horses
- horses: syndrome tends to be more acute than in ruminants; suggested miserotoxin causes poisoning in horses by some other mechanism than conversion to NPA
- lactating ruminants more susceptible; reason unknown
- experimental poisoning possible in pigs, rodents, rabbits, chickens, pigeons
- rabbits dosed PO with extracts of *Astragalus miser* died of methaemoglobinaemia, suggesting that miserotoxin is rapidly metabolised to release nitrite without hydrolysis of the glycosidic bond to release NPOH or NPA (Majak & Pass 1989)
- cattle, sheep: oral toxic dose NPOH or NPA = 20-60 mg/kg
- cattle: IV toxic dose NPOH = 30-35 mg/kg
- rat: oral acute LD₅₀ NPOH = 77 mg/kg; IP acute LD₅₀ NPOH = 61 mg/kg, NPA = 67 mg/kg
- rat: chronic IP toxic dose NPOH or NPA = 25 mg/kg twice daily for several days
- mouse: IP acute LD₅₀ NPA = 140 mg/kg

Toxicity (insects): (see Majak & Pass 1989)

- **honey bees** (*Apis mellifera*) poisoned by foraging on *Astragalus miser*, the nectar of which contains miserotoxin; confirmed experimentally (Majak *et al.* 1980) and on *Corynocarpus laevigatus* nectar (Palmer-Jones 1968)
- cabbage looper (*Trichopulsia ni*) larvae poisoned by *Coronilla varia*; attributed to NPA and esters; may act as feeding deterrents
- seed-eating beetle *Callosobruchus maculatus* poisoned by NPA
- leaf beetle *Chrysomela tremulae* uses 2-[6'-(3''-nitropropanoyl)-β-D-glucopyranosyl]-3-isoxazolin-5-one derived from NPA as a major component of its defense secretions

Metabolism of compounds:

- conjugates of NPA and NPOH are rapidly hydrolysed by rumen microbes; esterase liberates NPA, β-glucosidase liberates NPOH
- the glycosides can be absorbed intact from monogastric alimentary tracts, however esters of NPA should be hydrolysed by mammalian esterases in secretions of small intestine; miserotoxin is *not* converted to NPA in monogastric digestive tracts (Majak & Pass 1989)
- NPA & NPOH readily absorbed from the reticulorumen, NPOH being more rapidly absorbed; the different rates of absorption could partly explain the lesser toxicity of NPA to ruminants (Majak & Pass 1989)
- after absorption, NPOH is rapidly oxidised to NPA in liver by hepatic alcohol dehydrogenase; suppression of this alcohol dehydrogenase catalysis by ethanol or 4-methylpyrazole prevents poisoning in rats
- nitrite is produced from both NPOH and NPA by liver enzyme systems
- degree of oxidation of haemoglobin to methaemoglobin by the generated nitrite is usually <30%, even in fatal poisonings; IV methylene blue is *not* an effective therapy for NPA or NPOH poisoning in ruminants

Mode of action:

- NPA or a metabolite of NPA is the primary toxic agent, not nitrite
- NPA inactivates
 - succinate dehydrogenase (mitochondrial enzyme of the citric acid cycle, essential for respiration), binding irreversibly to the enzyme
 - fumarase (associated with the citric acid cycle)
 - isocitrate lyase (involved in the glyoxylate cycle)
- → reduction of ATP synthesis; tissue hypoxia (energy deficiency)

Conditions of poisoning:

- overgrazed or early spring pastures dominated by *Astragalus* spp.
- lactating ruminants more susceptible
- *Astragal miser*: rainfall can cause sudden increases in toxicity; most toxic before flowering; toxin concentrations approach zero with advancing stages of growth if dry conditions persist

Clinical signs:

Acute intoxication

- rapid onset, death within 4 to 24 hrs after ingestion
- **sudden death**
- dyspnoea
- cyanosis
- paresis, collapse
- \pm excitability if stimulated

Chronic intoxication

- weight loss
- **dyspnoea, nasal discharge, roaring sound**
- poor exercise tolerance
- poor hair coat
- **hind limb paresis**, staggering
- **knuckling of fetlocks**
- stress may precipitate death
- some affected animals recover, some remain ill for months
- sheep: respiratory signs predominate over nervous signs

Pathology:

Acute intoxication (Willaims & James 1978)

- pulmonary oedema, hydrothorax, tracheal petechiae
- methaemoglobinaemia

Chronic intoxication (ruminants) (James *et al.* 1980)

- congested liver
- ulceration of cardiac region of abomasum
- **pulmonary emphysema**, bronchoconstriction \pm bronchopneumonia
- **Wallerian degeneration of spinal cord & peripheral nerve white matter**
- focal brain haemorrhage

Diagnosis:

- access + syndrome + pathology
- assay methods available for miserotoxin, NPA and NPOH in plants, e.g. HPLC, Fourier transform infrared spectroscopy [FT-IR] (Schoch *et al.* 1998)

Therapy:

- no effective specific therapy available
- methylene blue controls methaemoglobinaemia, but does not prevent death (except in rabbits)
- thiamine IM suggested (adult cattle 400 mg. sheep 100 mg), but efficacy is doubted

Prevention & control:

- deny access to or limit intake of hazardous plant species, particularly to lactating ruminants
- *Astragalus miser* clipped early in spring had reduced toxicity – suggests early grazing may significantly reduce poisoning hazard
- herbicides: phenoxyherbicides & triclopyr effective against *Astragalus miser*
- ruminal metabolism modification
 - modification of the rate of ruminal detoxication of NPOH to prevent poisoning has been demonstrated by priming the rumen with doses of the stable sodium salt of nitroethane (20mg/kg/day), a non-toxic analogue of NPOH, to promote the appropriate bacterial populations; the improved capacity is transferable to other ruminants (Majak & Pass 1989, Majak *et al.* 1998)
 - a Gram-positive bacterium capable of rapidly metabolising NPOH has been isolated from rumen contents and is to be developed as a rumen inoculant to help prevent intoxication by speeding up adaptation of the ruminal flora to dietary nitro-compounds; preliminary field experiment is promising (Anderson *et al.* 1998)

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☑ Aliphatic nitro compound(s) (probable aetiology) - *Indigofera linnaei* (Birdsville indigo) – Birdsville horse disease

Core data

Syndrome name: Birdsville horse disease

Common sources: *Indigofera linnaei*

Animals affected: horse

Mode of action:

- undefined
- toxin possibly a nitrotoxin

Poisoning circumstances: *Indigofera linnaei* major part of diet

Main effects:

- lethargy
- progressive incoordination
- spinal cord white matter degeneration

Diagnosis:

- access + syndrome
- differentiate from swainsonine poisoning

Therapy: gelatine drench

Prevention:

- feed supplementation
- deny access to dense plant stands

Syndrome names:

Birdsville horse disease

A similar clinical syndrome, called “grove disease”, is reported in horses grazing *Indigofera spicata* in Florida (Morton 1989).

Chemical structure:

The neurotoxin responsible for this syndrome is unknown, but is **possibly a nitrotoxin** yielding 3-nitropropionic acid (3-NPA) (*q.v.*), *not* the non-protein amino acid indospicine (*q.v.*) (Majak *et al.* 1992). The total NPA content of *I. linnaei* is 0.13-0.16% (Murray *et al.* 1965, Majak *et al.* 1992).

Majak *et al.* (1992) isolated from *I. linnaei* 3 3-nitropropanoyl esters of D-glucose. These are 1,2,6-tri-*O*-(3-nitropropanoyl)- β -D-glucopyranose (karakin), 3,4,6-tri-*O*-(3-nitropropanoyl)- α -D-glucose (previously undescribed) and 2,3,4,6-tetra-*O*-(3-nitropropanoyl)- α -D-glucose (previously described from *Indigofera suffruticosa*). Their toxicity has not been established, but their yielding of 3-NPA on hydrolysis suggests potential toxicity.

Sources:

Indigofera linnaei Ali [= *I. enneaphylla* L.] (Birdsville indigo, nine-leaved indigo) [DM78]

widespread in northern Australia (Q, NT, WA). The plant dominates vegetation in some parts of inland Australia under some circumstances.

Indigofera spicata (creeping indigo) is suspected of causing a similar syndrome (“grove disease”) in horses in Florida (see the account of indospicine)



Indigofera linnaei (Birdsville indigo). [RAM Photo]



Indigofera linnaei (Birdsville indigo); flowers and pods. [RAM Photo]

Toxicity:

Horses only.

Experimentally, a toxic dose of *I. linnaei* was 4.5 kg/horse/day for 2 weeks or more.

Mode of action:

Undefined. NPA (*q.v.*) inhibits succinate dehydrogenase and other mitochondrial enzymes essential to respiration.

Conditions of poisoning:

Poisoning occurs when *I. linnaei* forms a **major part of the diet** of horses. Poisoning occurs mostly in western Q, northern SA & NT, but rarely in other locations (Carroll & Swain 1983). Cases occur in November-March when rain stimulates the growth of *I. linnaei* from its rootstock, but when there is insufficient to cause significant other plant growth.

Clinical signs:

inappetence

lethargy (stand with eyes closed for long periods)

↓ body weight

progressive gait incoordination

forced exercise → head & tail held high, front feet lifted high

hindquarters droop, flexed hocks → **toe dragging** → excessive toe wear

difficulty with turning

± spin on front feet in tight circles

if ridden → sudden loss of control of hindquarters → collapse

± bilateral ocular discharge

± stomatitis

± roaring in recovered horses

Pathology: No lesions of significance in the nervous system or musculature have been described in the published literature (Bell & Hall 1952, Rose *et al.* 1951). Clinical signs suggest spinal cord damage, a possible peripheral neuropathy or both. A recent case in 2 Australian Stock horses examined histologically revealed mild Wallerian degeneration of the lateral and ventral spinal cord white matter columns (digestion chambers with microglial phagocytes and some swollen axons, best seen in longitudinal rather than transverse sections), but no degenerative or inflammatory lesions of peripheral nerves (McKenzie RA & Berry JE, unpublished data 2000, 2001).

Diagnosis:

plant access + syndrome

differentiate from *Swainsona* poisoning and pyrrolizidine alkaloidosis

Therapy:

Mild cases commonly recover unaided if removed from access to *I. linnaei*. Gelatine in warm water by stomach tube @ 450 g/day, 3 days → apparent improvement (Hooper *et al.* 1971)

Prevention & control:

feed supplements (lucerne hay, peanut meal, cotton seed meal reported effective)

? fence off large concentrations of plants

? graze large concentrations of plants with goats/sheep

References:

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Cynanchosides

Syndrome name: Cynanchosis (South Africa)

Chemical structure:

Seven pregnane glycoside neurotoxins have been isolated from African species of *Cynanchum* (Family Asclepiadaceae) (Steyn 1989) and the toxins in African species of *Sarcostemma* are thought to be closely related to these (Kellerman *et al.* 1988).

Sources:

Family Asclepiadaceae

Australia

Sarcostemma brevipedicellatum [= *Sarcostemma australe*] (caustic vine, caustic creeper, pencil caustic) [DM120] (Bailey 1900)

South Africa

Sarcostemma viminale (L.) R.Br. (caustic bush, caustic creeper, melktou, spantoumelkbos)

Cynanchum spp. (monkey rope, klimop, bobbejaantou)

Cynanchum africanum R.Br.

Cynanchum ellipticum (Harv.) Dyer [= *C. capensis* Thunb.]

Cynanchum obtusifolium L.f.

Toxicity:

sheep, horses

Mode of action: undescribed

Conditions of poisoning (Australia):

dry conditions, other feed scarce

recently-burnt areas, fresh shoots of *Sarcostemma*, no other feed

Clinical signs:

restlessness

staggering, muscle tremor, collapse → lateral recumbency

hypersensitivity

recurrent tetanic seizures, opisthotonus

limb paddling

saliva drooling

several days, paralysis → death

Pathology: no significant lesions reported

Diagnosis: access + syndrome

Therapy: nil reported

Prevention & control: deny access

References:

Bailey FM (1900) Plants reputed poisonous to stock. *Sarcostemma australe* R.Br., or caustic vine. *Qd. Agric. J.* 7:259. [cited by Hurst 1942]

Erasmus GL (1983) *Cynanchum* gifstowwe geidentifiseer. *Newsletter, Onderstepoort Veterinary Research Institute*, 1 (3):10 [cited by Kellerman *et al.* 1988, p.52.]

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