Grazing animals at risk of eating the plant under dry pasture conditions should be either denied access to them or given adequate supplementary feed to reduce the probability of significant browsing of shrubs and trees.

Effective mechanical removal of the plant is hindered by its capacity to produce root suckers readily. Picloram + triclopyr (Access®) is registered for use on bitterbark as a basal bark or cut stump application using diesel as a carrier. Long-term control of root suckers in fallowed cultivation (> 3 years) is by applying picloram + triclopyr (Grazon® DS) as a 1:4 concentration in water using a blanket wiper in autumn. Adding glyphosate provides no advantage. Use 2% Grazon® DS (100 ml concentrate in 5 L water) to spot spray individual plants, thoroughly wetting all leaves and stem. Treated areas should not be cultivated for 6 months. These are registered uses of these herbicides. [Osten & McCosker 2002]

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Indole (pyrrolidinoindoline) alkaloids - calycanthine, chimonanthine, idiospermuline

Chemical structure:

The known toxic pyrrolidinoindoline alkaloids are

- calycanthine [CA] (Hamor *et al* 1960, Woodward *et al.* 1960)
- chimonanthine [CH]
- idiospermuline [ID] (Duke *et al.* 1995)

Sources:

Plants

Australia

Idiospermum australiense (Diels) S.T.Blake [= *Calycanthus australiensis* Diels] (ribbonwood) is a rare lowland rainforest tree of northern Queensland in the monotypic Family Idiospermaceae (Blake 1972). Its "seeds" are 3-6 cm in diameter and comprise naked embryos each with 3-4 massive fleshy cotyledons (Blake 1972). The "seeds" contain CA, CH, ID (Duke *et al.* 1995).

New Guinea

Bhesa archboldiana (Merr. & Perry) Ding Hou [= *Kurrimia archboldiana* Merr. & Perry] is a large rainforest tree of New Guinea in the Family Celastraceae. Its bark contains CA (Culvenor *et al.* 1970).

North America

Calycanthus L. (2 species - Mabberley) are shrubs in the Family Calycanthaceae distributed in southern USA

Calycanthus fertilis Walt. ("bubby" or strawberry bush; Carolina, hairy or smooth allspice; sweet or pale sweet shrub; Indian toothpick) There is field evidence of its toxicity in Tennessee USA (Beasley *et al.* 1997).

Calycanthus floridus L. (Carolina allspice) – CA
Calycanthus glaucus Willd. - CA
Calycanthus occidentalis Hook. & Arn. (Californian allspice)
Meratia praecox [*sic*] in the Family Asteraceae – CA [isolated from seeds by Manske 1929]
China
Chimonanthus Lindley (6 species - Mabberley) are shrubs in the Family Calycanthaceae restricted to China; some species are cultivated
Chimonanthus fragrans Lindley – CH
Chimonanthus praecox (L.) Link - CA
Amphibians
South America
skin of the dendrobatid frog *Phyllobates terribilis* (*q.v.*)

Organ systems affected: CNS

Toxicity:

Idiospermum australiense : "Seeds" are toxic to cattle. There is one natural poisoning case on record from the Daintree region of north Queensland in August 1971 in which 6 cattle died (Blake 1972). Feeding experiments with sheep and calves have confirmed the toxicity of the "seeds". Lethal single doses of cotyledons PO in sheep and a calf were 5-6 g/kg (Hall 1971, 1974, 1975).
Calycanthus fertilis : Seeds are "reputed to be poisonous to cattle in Tennessee" (Chesnut 1898).
Calycanthus floridus: Bradley & Jones (1963) reported strychnine-like signs in cattle after consumption of the plant in the field in Georgia USA and in a dog fed seeds experimentally at 6.25 g/kg.

Mode of action: (Duke *et al.* 1995)

Conditions of poisoning:

Idiospermum australiense : Affected cattle ate "seeds" in large amounts from the ground under mature trees (Blake 1972).

Clinical signs:

Idiospermum australiense (Clague 1971, Hall 1971, 1974, 1975):
sudden deaths
powerful muscle contractions & excitement followed by collapse and death
hypersensitivity to external stimuli (time from dose to onset in experimental ruminants was 3-4 hr)
exaggerated reflexes
tetanic spasms

Calycanthus fertilis (Beasley *et al.* 1997):
sudden deaths
muscle fasciculation
recumbency with tetanic spasm stimulated by sensory input (cf. strychnine poisoning)
injected sclera, pupil dilation
tachypnoea

Pathology:

Idiospermum australiense (Clague 1971, Hall 1971, 1974, 1975):
Numerous "seeds" in rumen contents (> 1kg in the field case)
haemorrhages in epicardium, rumen, abomasum and upper small intestine with free blood in the lumen of the small intestine in one case

Calycanthus fertilis (Beasley *et al.* 1997):
Clinical chemistry normal
No lesions in organs
seed pods and seeds in rumen contents

Diagnosis:

syndrome + access + seeds in rumen
differential diagnoses include strychnine poisoning (hypersensitivity leading to tetanic spasms) and arsenic poisoning (alimentary haemorrhages)

Therapy:

Calycanthus fertilis (Beasley *et al.* 1997): Place cattle in quiet stall and institute a regimen of minimal stimulation. Dose PO with activated carbon. Sedate with chloral hydrate IV @ 50-70 mg/kg to effect or pentobarbitone IV @ 30 mg/kg to effect.

Prevention & control: deny access

References:

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Taxine diterpenoid alkaloids

Core data

Common sources: Taxus spp. (yew trees)
Animals affected: horses, cattle (and others)
Mode of action: inhibition of cardiac depolarisation
Poisoning circumstances: access to trees in gardens or to garden waste
Main effects: sudden death
Diagnosis: plant in mouth/stomach
Therapy: prompt activated charcoal + atropine
Prevention: deny access

Chemical structure:

Taxine is a complex mixture of at least 11 **diterpenoid alkaloids** (Cooper & Johnson 1998). Taxine A is a minor component of the mixture with **taxine B** and **isotaxine B** being the major toxic components (Kite *et al.* 2000, Wilson *et al.* 2001).

Paclitaxel (taxol), the mitotic spindle poison used to treat human ovarian neoplasms, also has cardiotoxic effects at high dose (Wilson *et al.* 2001).

Other toxic constituents of *Taxus* spp. are traces of taxiphyllin, a cyanogenic glycoside, and a volatile oil (oil of yew) thought to be responsible for irritation of the alimentary tract seen in some cases.

Sources:

***Taxus* spp.** (**yew** trees) – 6-7 species in the genus; dioecious (sexes separate) evergreen coniferous trees (gymnosperms), native of the northern hemisphere temperate zone (mostly)

[species associated with recorded poisoning cases = #]

***Taxus baccata*#** (common yew, English yew, Irish yew) native of Europe, cultivated in Australia (many cultivars available, all female) (Samuel 1993)

Taxus cuspidata# (Japanese yew) native of Asia, cultivated in Australia

Taxus brevifolia# (Californian yew) native of North America (exploited as a source of paclitaxel (Taxol) and docetaxel (Taxotere), used for therapy of ovarian neoplasia in humans)

Taxus canadensis# (American yew) native of North America

Taxus mairei (= *T. chinensis, T. sumatrana*) (Chinese yew)

Taxus x *media* (= *T. baccata* x *T. cuspidata*)

Toxicity:

horses, cattle most commonly affected

sheep and deer are more resistant, with some evidence available of browsing of yew trees without effect by these species

cases of poisoning have been recorded in sheep, goats, deer, kangaroos, a dog, emus, pheasants and canaries (Cooper & Johnson 1998), llamas, chinchillas (Wilson *et al.* 2001), but not in parrots ingesting yew clippings (Wilson *et al.* 2001).

all parts of yew trees are toxic except the brightly-coloured aril (scarlet when ripe) around the seed

dried plant retains toxicity

most toxic in winter

oral lethal doses: horses 0.5-2.0 g/kg, pig 3.0 g/kg, ruminants 1.0-12 g/kg,

total lethal dose of fresh plant: horse 100-200 g, cattle about 500 g, sheep 100-200 g, pig 75 g, fowl 30 g

Mode of action: alkaloids believed to inhibit cardiac muscle Ca and Na pumps (Panter *et al.* 1993)

Conditions of poisoning:

- access to gardens containing cultivated yews
- access to garden waste containing prunings from yews

Clinical signs:

- **sudden death** (even in the act of chewing the plant) is the most common outcome; time from ingestion to death may be as little as 5 minutes; most deaths occur within the first 6 hr after ingestion, however, signs in cattle may not occur for up to 2 days
- trembling, muscle weakness, dyspnoea & collapse
- cardiac arrhythmia (tachycardia then bradycardia, heart block) is believed to be the cause of death
- ± abdominal pain, pupil dilation, diarrhoea, vomiting and convulsions
- mildly-affected animals may survive

Pathology:

- plant in mouth and rumen/stomach
- **no significant lesions**
- ± gastric congestion (if death delayed for a few hours)

Diagnosis:

- access + sudden death + plant demonstrated in mouth/stomach [more likely in ruminants than monogastrics]
- assay of stomach contents for taxine alkaloids or other chemical markers of *Taxus* spp. is available in some labs; stomach contents preserved with ethanol are suitable for some assays
- assay methods reported include
  - HPLC/mass spectrometry to detect intact taxine alkaloids (Kite *et al.* 2000)
  - direct insertion probe mass spectrometry (Smith 1989) and GC/MS (Lang *et al.* 1997) to detect β-methylamino-β-phenyl-α-hydroxy propionic acid, the major breakdown fragment of taxine alkaloids.
  - thin layer chromatography to detect taxol (Panter *et al* 1993)
  - phloroglucindimethylether (3,5-dimethoxyphenol) detection (Musshoff *et al.* 1993)

Therapy:

- **prognosis grave** unless very early therapeutic intervention, however, not all cases are invariably fatal (Hu276, Cooper & Johnson 1998)
- prompt detoxication (rumenotomy with removal of contents and/or oral activated charcoal + cathartic) (Casteel & Cook 1985)
- prompt atropine administration (*vs.* cardiodepressant effects); IV lignocaine has been used against arrhythmia in human cases (von Dach & Streuli 1988)
- support for respiration

Prevention & control: deny access to susceptible animals

References: Hu276, Os393

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☑ Theobromine (a xanthine alkaloid)

Core data

Common sources: chocolate
Animals affected: dogs
Mode of action: interference with the electrical activity of cardiac myocytes
Poisoning circumstances: rapid consumption of large amounts of confectionary dominated by chocolate
Main effects:

- sudden death, myocardial necrosis
- pancreatitis

Diagnosis: syndrome + possible assay of stomach contents
Therapy: decontamination + antiarrhythmic drugs
Prevention: deny access

See Human Foods & Beverages under Chocolate (theobromine)
Xanthines include caffeine (1,3,7-trimethylxanthine), theobromine (3,7-dimethylxanthine) and theophylline (1,3-dimethylxanthine)

☑ Swainsonine [an indolizidine alkaloid]

Core data

Syndrome names: pea-struck, locoism, Darling pea poisoning
Common sources:

- *Swainsona* spp. (Darling peas) in Australia
- *Astragalus* spp. & *Oxytropis* spp. (locoweeds) in North America

Animals affected: horses > ruminants
Mode of action: inhibits α-mannosidase → acquired lysosomal storage disease
Poisoning circumstances:

- after droughts & floods, *Swainsona* spp. shoot from perennial rootstock
- large intake > 2 weeks for horses, > 4 weeks for ruminants
- some selectively graze *Swainsona* spp. (addiction?)

Main effects:

- weight loss, incoordination, erratic behaviour
- fine cytoplasmic vacuolation of neurones, viscera and lymphocytes

Diagnosis: pathology
Therapy: nil; remove from source
Prevention:

- graze infested pasture up to 2 (horses) or 4 weeks (non-pregnant ruminants)
- spell all stock 4 weeks before re-exposure to plants

> "Of the Darling Pea, Mr. Wm. Nepean Hutchison says stock readily devour it, and it takes but little to drive them perfectly silly."

> Frederick Manson Bailey (Colonial Botanist) & Patrick Robertson Gordon (Chief Inspector of Stock) (1887) *Plants Reputed Poisonous and Injurious to Stock.* James C. Beal, Government Printer, William Street, Brisbane. p.25.

The identity of swainsonine as the cause of plant-associated neurological syndromes of livestock in both Australia and North America was in part the result of collaboration between scientists in both continents beginning about the time of the first Australia-United States Symposium on Poisonous Plants held in Ames, Iowa (Hartley 1978).

Syndrome names:

Swainsonine alone:

- **pea-struck**
- Darling pea poisoning
- **locoism** [USA]

Swainsonine + altitude causing congestive heart failure [USA] (James *et al.* 1983, 1986, 1991a,b):

- high mountain disease
- brisket disease

Chemical structure:

Swainsonine is a polyhydroxylated indolizidine alkaloid (Fellows 1986), first isolated and characterised from *Swainsona canescens* in Australia (Dorling *et al.* 1993). It was subsequently identified in *Astragalus lentiginosus* in North America (Molyneux & James 1982).

Plant Sources:

Family Fabaceae

- *Swainsona* spp. (**Darling peas**) in Australia (Colegate *et al.* 1991)
- *Astragalus* spp. & *Oxytropis* spp. (**locoweeds**) in North America (Molyneux & James 1991); *Oxytropis sericea* (white locoweed) is associated with high mountain disease in USA (James *et al.* 1983, 1986, 1991a,b).

Swainsona spp.:

Swainsona is confined to Australasia with 84 species are known in Australia and 1 in New Zealand (Thompson 1993). Seven species have been associated with poisoning of livestock to date, namely:

Swainsona brachycarpa Benth. (small-flowered Darling pea) - south-eastern Q, north-eastern NSW. In grassland and woodland, often associated with rocky sites and loamy soils.

Swainsona canescens (Benth.) F.Muell. (grey Swainson pea) [includes *S. canescens* var. *horniana* J.Black] - widespread in desert regions of WA, western SA and NT in red sandy soil, often in shrubland or mallee woodland.

Swainsona galegifolia (Andr.) R.Br. (smooth Darling pea) - In coastal areas and on the Dividing range from tropical Q to northern Vic, extending inland to the plains, especially in NSW. In light or heavy soil in a variety of habitats from grasslands and woodlands to rainforest margins.

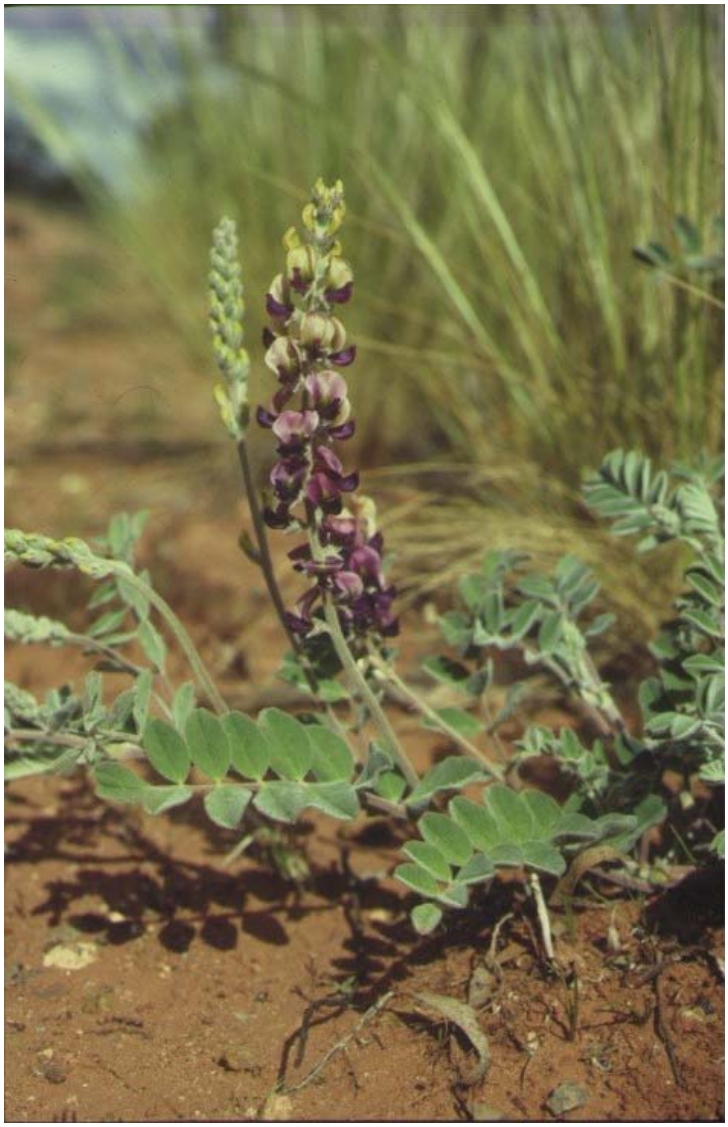
Swainsona greyana Lindl. (hairy Darling pea) - Almost limited to the heavy grey soils of the banks and flats of the lower Murray River and Darling River and major tributaries. Q, NSW, V

Swainsona luteola F.Muell. (dwarf Darling pea) - Widespread in south-eastern Q, especially west of the Dividing Range, and in central NSW, and in north-western Vic. Usually in rich heavy soil in open grassland.

Swainsona procumbens (F.Muell.) F.Muell. (Broughton pea) - Widespread in inland NSW, Vic and southern Q, and in south-eastern SA. Usually in heavy soils prone to waterlogging.

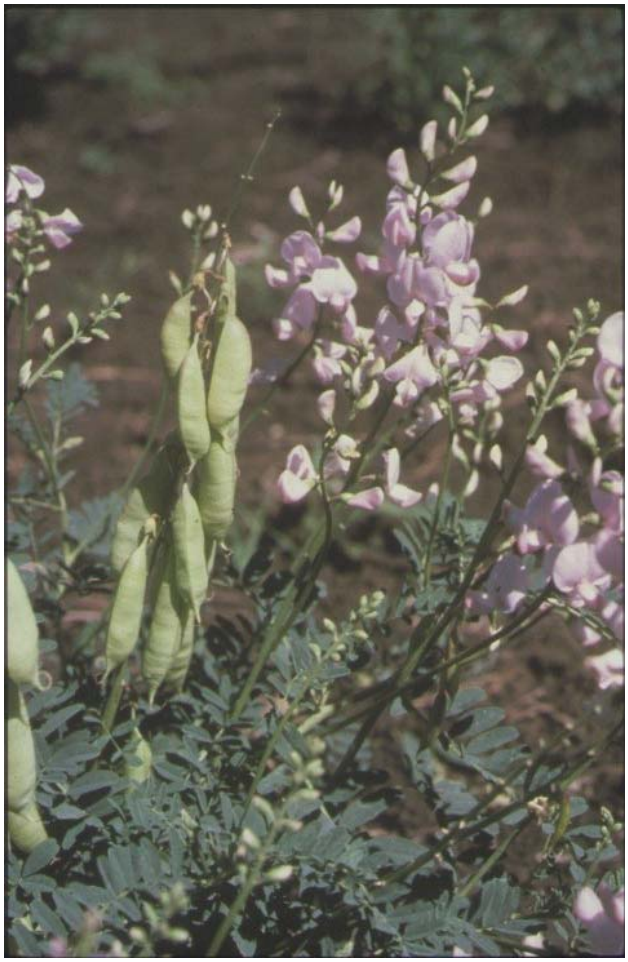
Swainsona swainsonioides (Benth.) A.Lee ex J.Black (downy Swainson pea) - Widespread in south-eastern Q, inland NSW, and northern Vic, and in north-eastern SA with an isolated occurrence in the north-west. In heavy red or black soils on plains.

Swainsona canescens (grey swainson pea) whole plant [RAM Photos]



Swainsona canescens (grey swainson pea) flowering twig [RAM Photos]

Swainsona galegifolia (smooth Darling pea) - flowering and fruiting plant [RAM Photo]



Swainsona galegifolia (smooth Darling pea) - flowers & seed pods [RAM Photo]

Family Convolvulaceae

Ipomoea spp. (*q.v.*)

Family Malvaceae

Sida carpinifolia which causes acquired α-mannosidosis in goats in Brazil (Driemeier *et al.* 2000; Colodel *et al.* 2002)

Fungal Sources:

Rhizoctonia leguminicola a pathogen of *Trifolium pratense* (red clover) in North America (Schneider *et al.* 1983).

Metarhizium anisopilae (Hino *et al.* 1985)

Toxicity:

Swainsonine:

Horses are about twice as susceptible as **ruminants** to swainsonine. The syndrome was first experimentally reproduced in Australia by feeding sheep with *Swainsona galegifolia* in New South Wales (Martin 1897). Field and/or experimental toxicity has been reported in the following animal species with the following *Swainsona* species:

- **horses**: *Swainsona canescens* (Hooper & Locke 1979); *Swainsona luteola* (O'Sullivan & Goodwin 1977); *Swainsona brachycarpa* (O'Sullivan & Goodwin 1977)
- **sheep**: *Swainsona canescens* (Gardiner *et al.* 1969, Dorling *et al.* 1978); *Swainsona galegifolia* (Martin 1897, Laws & Anson 1968); *Swainsona luteola* (Cleland & McDonald 1917, Laws & Anson 1968); *Swainsona procumbens* (Hurst 1942)
- **cattle**: *Swainsona canescens* (Hooper & Locke 1979); *Swainsona galegifolia* (Huxtable & Gibson 1970, Hartley & Gibson 1971); *Swainsona swainsonioides* (Everist 1981)
- **honey bees**: *Swainsona galegifolia*

Sheep experimentally dosed with 0.2 mg swainsonine/kg or greater (in *Oxytropis sericea*) for 30 days gained less weight than controls and had histological lesions; histological lesions occurred when tissue swainsonine concentrations were about 150 ng/g (Stegelmeier *et al.* 1999).

Swainsonine + altitude:

- cattle
- disease reproduced by feeding swainsonine at high altitude (2000-3000 m)

Mode of action:

Swainsonine:

Swainsonine poisoning is an **acquired lysosomal storage disease**. Swainsonine **inhibits lysosomal α-D-mannosidase and Golgi mannosidase II** and causes accumulation of large quantities of oligosaccharides composed of mannose & N-acetylglycosamine in lysosomes (Tulsiani *et al.* 1982). This appears histologically as fine cytoplasmic vacuolation. In cases including abortion, swainsonine causes lysosomal storage disease in the uterus, placenta & foetus.

Swainsonine + altitude:

The pathogenesis of this condition is not understood. Pulmonary vascular changes, damage to innervation to the heart, damage to neurones of the respiratory centre of the medulla may be involved in exacerbating hypoxia and pulmonary hypertension.

Conditions of poisoning:

Swainsonine:

Swainsona plants are palatable. Poisoning occurs when they make up a large proportion of the diet for a prolonged period. A rule of thumb for length of exposure to *Swainsona* before clinical signs appear is **> 2 weeks intake for horses, > 4 weeks intake for ruminants**.

Suitable conditions for abundant *Swainsona* growth can occur

- in spring (September-November) in years with below-average summer rain and when winter rain has been enough to stimulate growth of *Swainsona* from its rootstocks (Everist 1947).
- after drought-breaking rain
- floods may stimulate abundant *Swainsona* growth during autumn-winter

Some animals selectively graze *Swainsona* plants and are reported to develop a craving, actively seeking them among the available forage (Hartley 1978). This has been described as addiction, but the aptness of this term for this behaviour is not established. Addiction is defined as the overwhelming desire or need to continue the ingestion of a xenobiotic even when such use has deleterious physical, psychological or social manifestations (Hodgson *et al.* 1998).

Swainsonine + *altitude*:

Cattle in North America grazing mountain pastures (2000-3000 m high) infested with *Oxytropis sericea* (white locoweed) have a much higher prevalence of congestive heart failure (high mountain or brisket disease) than those not exposed to the plant.

Clinical signs:

Swainsonine: (Hartley & Gibson 1971, Hartley 1978, Locke *et al.* 1980)

- **weight loss** (some sheep progress to emaciation and death without nervous signs)
- staring eyes
- head shaking or tremor
- head pressing
- **incoordination**, paddling gait
- muscle tremor
- hyperexcitability, **erratic manic behaviour** when handled (particularly in horses)
- difficulty with prehension & mastication
- infertility
    - ± abortion in cattle [more commonly reported in North America than Australia] – affected cattle in Australia have a poor breeding record, tend to abort at *ca.*5-7 months and full term calves are non-viable. Abortion plus skeletal malformations occur in both sheep & cattle in North America. Abortions or terata are not reported in sheep in Australia.
    - prolonged oestrus cycles in cattle (from mean 19 days to mean 34 days) with infertility (Panter *et al.* 1999)
    - testicular degeneration in rams (Panter *et al.* 1989)

Swainsonine + *altitude*:

Right-sided heart failure

- depression
- diarrhoea
- oedema of submandibular space and brisket
- dyspnoea
- distended jugular veins
- weakness

Pathology:

Swainsonine:

- no specific lesions at necropsy; emaciation
- yellow discoloration of brainstem in chronically-emaciated sheep
- **cytoplasmic vacuolation** of circulating **lymphocytes** (Huxtable & Gibson 1970)
- fine **cytoplasmic vacuolation** of **neurones** and **visceral parenchyma** (liver, kidney, pancreas, thyroid, placenta)
- persistent eosinophilic spheroids in axons

Swainsonine + *altitude*:

- right ventricular hypertrophy and dilation
- ascites, hydrothorax
- passive venous congestion of liver
- neurovisceral foamy cytoplasmic vacuolation

Diagnosis:

Swainsonine:

- access + pathology
- differential diagnosis in horses includes *Indigofera linnaei* poisoning and pyrrolizidine alkaloidosis

Swainsonine + altitude:

syndrome + plant access on high mountain pasture + pathology

Therapy:

Swainsonine:

There is no specific therapy. Removal from the swainsonine source leads to gradual recovery unless animals are severely or chronically affected.

Swainsonine + altitude:

Remove from high pastures and plant access

Prevention & control:

Swainsonine:

Non-pregnant cattle and sheep can be grazed on infested pasture for up to 4 weeks without serious poisoning and horses for 2 weeks. Spell all stock for 4 weeks before re-exposure to plants. Increased stocking rates for shortened times have been effective at reducing losses from white point locoweed (*Oxytropis sericea*) in USA (Ralphs *et al.* 1984). Herbicides have been used successfully against locoweeds in USA, but soil seed reservoirs and long seed viability prevent their long term use (Ralphs & Ueckert 1988). Ionophore growth promotants appear not to potentiate the effects of swainsonine (Whittet *et al.* 2002).

Swainsonine + altitude:

Avoid plant access at high altitude.

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*Swainsonine + calystegines (*Ipomoea *spp.)*

Chemical structure:

Swainsonine = a polyhydroxylated indolizidine alkaloid

Calystegines = polyhydroxylated *nor*tropane alkaloids

Calystegines have been isolated previously from *Calystegia sepium* and *Convolvulus arvensis* (both in Family Convolvulaceae) and *Solanum dimidiatum* and *Solanum kwebense* (Molyneux 1990); they are a class of polyhydroxy alkaloids, the *nor*tropanes, with potent glycosidase inhibitory properties (Molyneux *et al.* 1993)

Sources:

***Ipomoea* spp.**

- *Ipomoea* sp. aff. *calobra* (Weir vine) [DM117] Australian native plant confined to the Maranoa district, Q; some dispute over taxonomy – possibly closely related to isolated populations of *Ipomoea polpha* in the Atherton district, Q and central Australia (Molyneux *et al.* 1995)
- *Ipomoea carnea* ssp. *fistulosa* native to the Americas → clinical cases in Mozambique, Sudan, India, Indonesia (Idris *et al.* 1973, de Balogh *et al.* 1999)

- *Ipomoea muelleri* (poison morning glory) Australian native plant widespread in tropics (WA, NT, Q)

Toxicity:

Ipomoea sp. aff. *calobra*

- sheep, cattle, horses
- toxins = swainsonine (indolizidine alkaloid) + (probably) calystegines (nortropane alkaloids); both glucosidase inhibitors (Molyneux *et al.* 1995)

Ipomoea carnea

- goats
- 1.5 kg fresh weight/goat/day for up to 3 months (Idris *et al.* 1973)
- toxins = swainsonine + calystegines B_2 and C_1 (de Balogh *et al.* 1999)

Ipomoea muelleri

- sheep in WA (Gardiner *et al.* 1965)
- toxin unidentified (currently suspected swainsonine + calystegines)

Mode of action:

- acquired lysosomal storage disease through inhibition of enzymes of carbohydrate metabolism
- swainsonine inhibits α-mannosidase (see above)
- calystegines inhibit β-glucosidase and α- and β-galactosidase
- inhibition of α- and β-galactosidase → phenocopies of human genetic lysosomal storage defects Gaucher's disease and Fabry's disease respectively.

Conditions of poisoning:

Ipomoea sp. aff. *calobra*

- spring rains → rapid growth of vines from underground tubers, other feed scarce
- prolonged intake (4-5 weeks) → poisoning

Ipomoea carnea

- village goats with limited nutritional sources

Ipomoea muelleri

- consumption of plants which become abundant after wet season rains; plants grow in seasonally-flooded clay soils in open grassland (Everist 1981)

Clinical signs:

Ipomoea sp. aff. *calobra*

- ↓ body condition
- `star-gazing'
- blindness
- head pressing
- muscle tremor
- ↑ urine frequency & volume
- death from starvation/thirst/misadventure

Ipomoea carnea (de Balogh *et al.* 1999)

- ataxia
- head tremors
- nystagmus
- hyperaesthesia
- high-stepping gait
- death within a few weeks

Ipomoea muelleri (Everist 1981)

- steady loss of weight
- hind limb dysfunction: driven sheep have a "jerky" gait and tire easily with dyspnoea
- knuckling of hind feet
- posterior ataxia (swaying, incoordinated)

Pathology:

Ipomoea sp. aff. *calobra*

- nephrosis
- cytoplasmic vacuolation of neurones, persistent spheroids in axons (cerebellum particularly susceptible)

Ipomoea carnea

- cytoplasmic vacuolation of neurones, spheroids in some axons (cerebellum particularly susceptible)

Diagnosis: pathology

Therapy: nil
Prevention & control: deny access for prolonged periods
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*Calystegines (***nor***tropane alkaloids) - probable aetiology of* **Solanum** *spp.-associated cerebellar degeneration*

Syndrome names:

maldronksiekte (cattle, South Africa) (= mad -drunk disease)
vallendesiekte (cattle, South Africa) (= epilepsy)
crazy cow syndrome (west Texas)

Chemical structure:

The neurotoxins responsible are **unknown**. Signs are not consistent with the known effects of glycoalkaloids of *Solanum* spp. Bourke (1997) suggested β-carboline alkaloids as possibly involved. The pathology observed suggests that an acquired lysosomal storage disease may underlie the syndrome. In this vein, Riet-Correa *et al.* (1983) suggested a possible gangliosidosis. **Calystegines** (*q.v.*) have been isolated from some of the plants involved (*Solanum dimidiatum* and *Solanum kwebense* (Molyneux 1990)), are potent inhibitors of glucosidase enzymes and appear, at least in part, to be the most likely known causative toxins for this syndrome.

Sources & toxicity:

particular *Solanum* spp. involved

Solanum kwebense (rooibessie, bitterappel); southern Africa; cattle (Pienaar *et al.* 1976); horses, donkeys, goats claimed to be affected by farmers, but unconfirmed

Solanum fastigiatum (jurubeba); Brazil; cattle (Riet-Correa *et al.* 1983; Zambrano *et al.* 1985)

Solanum bonariensis (Uruguay); cattle (Riet-Correa *et al.* 1983)

Solanum dimidiatum Raf. [= *S. carolinense* L.] (potato weed, western horsenettle); north America; cattle (Menzies *et al.* 1979). Naturalised in Australia in the Bundaberg area of Queensland, associated with sugar cane (Purdie *et al.* 1982). No poisoning cases recorded in Australia.

Solanum cinereum (Narrawa burr); Australia; goats (Bourke 1997), sheep & horses (Dodd 1922, 1923, Hurst 1941); not closely-related to other native Australian *Solanum* spp. (Purdie *et al.* 1982)

All parts of the plants are probably toxic

The syndrome has been reproduced by feeding experiments in cattle with *S. kwebense* (Pienaar *et al.* 1976), with *S. dimidiatum* (Menzies *et al.* 1979) and with *S. fastigiatum* (Riet-Correa *et al.* 1983) and in sheep with *S. fastigiatum* (Zambrano *et al.* 1985); feeding experiments with *S. kwebense* in a donkey, 2 goats and a sheep were negative (Pienaar *et al.* 1976)

Mode of action:

undetermined

Conditions of poisoning:

cattle browsing *S. kwebense* in north-western Transvaal; cases more common in dry years; overgrazed pastures with *S. kwebense* replacing the more usual forage (Pienaar *et al* 1976)

cattle grazing fruiting *S. dimidiatum* during late summer and winter in North America; youngest affected cattle 6 months old (Menzies *et al.* 1979)
cattle grazing *S. fastigiatum*-infested pastures in South America (Riet-Correa *et al.* 1983)
goats grazing weed-infested pastures for several months (12-18 months) in Australia ; youngest affected goat 6 months old (Bourke 1997)

Clinical signs:

Cattle (Pienaar *et al.* 1976, Menzies *et al.* 1979, Riet-Correa *et al.* 1983):
affected animals behave normally until disturbed, raising the muzzle also precipitates an episode
then severely-affected animals had **transient episodes** comprising
rigid neck with extended head
nystagmus, staring eyes
falling to the side or backwards with muscle tremors, opisthotonus
struggle to regain feet
generalised muscle tremors
rapid recovery; secondary trauma from effects of sudden falling (broken horns, broken teeth and jaws, bruising of mouth, brisket & legs)
less severely-affected animals had
swaying of head from side to side, "star-gazing"
lateral head tilt
broad-based stance
ataxia
muscle tremors
hypermetria
syndrome irreversible
death from the condition is rare; usually associated with misadventure (affected animals are "accident-prone") for example, drowning is common

Goats (Bourke 1997)
paresis; mild in forelimbs, marked and intermittent in hindlimbs; worst when starting to walk or turning
wide-based stance
incoordinated gait
disturbed equilibrium
mild tremor of head, neck and body
mild hypermetria of forelimbs
abnormal head orientation ; either extended or tilted
nystagmus
intensity of signs can be enhanced by raising the head backwards until the animal fell over or by holding the animal in lateral recumbency for several minutes, then suddenly releasing it
syndrome is chronic non-progressive but irreversible

Pathology:

Cattle (Pienaar *et al.* 1976, Riet-Correa *et al.* 1983)
cerebellar atrophy at necropsy
paucity or absence of Purkinje cells in cerebellar cortex
foamy **cytoplasmic vacuolation** of remaining Purkinje cells
swollen axons of Purkinje cells
ultrastructural studies revealed numerous whorled membranous bodies in Purkinje cell cytoplasm (Riet-Correa *et al.* 1983)

Goats (Bourke 1997)
cerebellum: deficit of grey matter compared with white matter; lesion only in cerebellum; other organs normal at necropsy
brain weights and cerebellar weights similar to normal goats
absence of Purkinje cells in many cerebellar folia
some remaining Purkinje cells degenerative and some with fine foamy cytoplasmic vacuolation
similar but milder vacuolation in hippocampus and choroid plexus
proximal portions of Purkinje cell axons swollen and degenerate ("torpedoes")
limited spheroidal neuroaxonal dystrophy scattered through brain
mild Wallerian degeneration in white matter of cerebellum and spinal cord

Diagnosis: syndrome + plant access
Therapy: nil
Prevention & control:
prevent access over a prolonged period
reduce or eliminate factors leading to increased population density of the causative plants in pasture, such as overgrazing

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☑ *Indole alkaloids of* Phalaris *spp (phalaris) - Phalaris staggers*

Core data

Syndrome names: Phalaris staggers
Common sources: Phalaris aquatica
Animals affected: sheep, cattle
Mode of action: methyl tryptamine & β-carboline alkaloids act on serotonergic receptors in brain and spinal cord neurones
Poisoning circumstances:

- access to lush new growth
- alkaloid content boosted by N fertilizer, heat, shade (cloud)
- onset may be delayed weeks-months

Main effects:

- hyerexcitable state exacerbated by forced exercise
- tremors, limb paresis, recumbency with vigorous struggling
- consciousness retained
- cattle-dysphagia

Diagnosis:

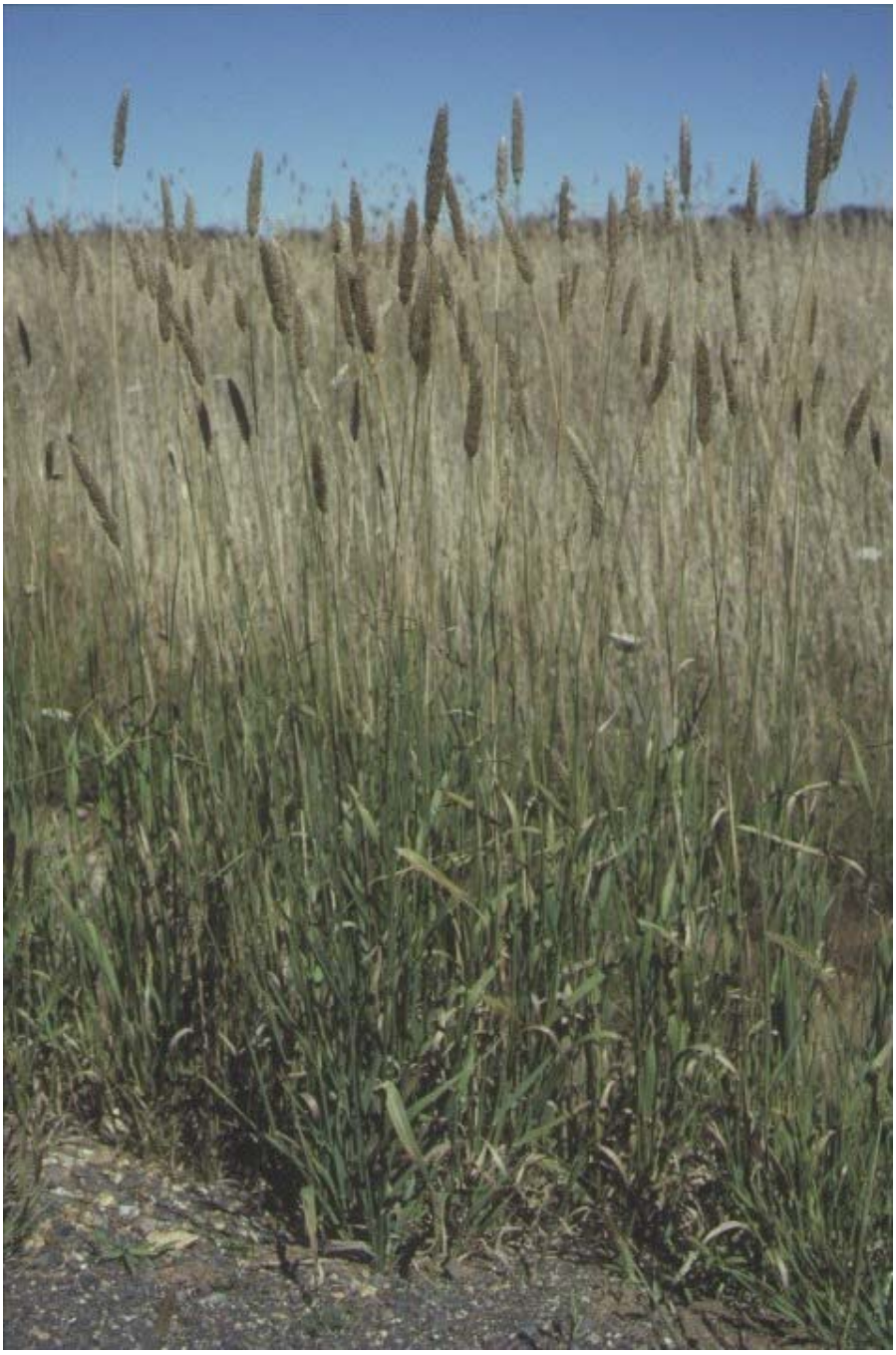
- syndrome
- CNS, kidney pigmentation

Therapy: gentle removal from source
Prevention: cobalt bullets before access to pasture

See notes on *Phalaris aquatica* sudden death (*q.v.*) and *P. coerulescens* (*q.v.*)
Syndrome name: **Phalaris staggers**
Chemical structure:
- toxins:
methylated tryptamine alkaloids (dimethylated indole alkylamines) → rapid effects
ß-carboline (indole) alkaloids - cumulative & → persistent effects (Allen & Holmstedt 1980)

Sources: temperate zone pasture grasses
- ***Phalaris aquatica*** (Australian phalaris, Toowoomba canary grass, *P. tuberosa*)

- *Phalaris arundinacea* (reed canary grass)
- *Phalaris canariensis* (canary grass)
- *Phalaris angusta* – South America (Gava *et al.* 1999)
- *Phalaris brachystachys* – Europe
- *Phalaris caroliniana* – North America
- *Phalaris minor* – North America



Flowering / seeding *Phalaris aquatica* (Australian phalaris, Toowoomba canary grass) [RAM Photo]

Toxicity:

- **sheep**, cattle
- Australia, South Africa, New Zealand
- California (East & Higgins 1988)
- syndrome is tremorgenic & paretic, **not** usually convulsive

The literature does not record cases of phalaris staggers in horses. One probable case has been seen in horses which grazed a newly established *Phalaris aquatica* pasture in the Hunter Valley of New South Wales in the autumn of 1989, the first clinical signs (described by a lay observer as staggers) being seen within a week of first exposure (RA McKenzie, unpublished data 1990, cited in McKenzie 1994)

Mode of action:

- direct action of phalaris alkaloids on serotonergic receptors in specific brain and spinal cord nuclei → ↑ response to excitatory inputs
- clinical syndrome in sheep reproduced by IV 5-methoxy dimethyl tryptamine (Bourke *et al.* 1990*)*.

Conditions of poisoning:

- access to lush new growth (young plants more toxic)
- pasture is more toxic in the morning than in the afternoon
- factors promoting alkaloid content: high soil N, high temperatures, shading (e.g. foggy or cloudy weather), frosts
- soil type appears to influence prevalence of disease, but not related to cobalt

Clinical signs:

- C.A. Bourke (NSW Agriculture) produced a revised clinical interpretation of the syndrome compared with the previous one by Gallagher
- very variable clinical signs & clinical course (days → weeks → months)
- onset may be delayed for weeks or months after access starts or may be delayed until weeks after access ceases
- signs exacerbated by forced exercise

Sheep (Bourke et al. 1990)

- **hyperexcitable**; fully conscious, struggle vigorously to regain feet if recumbent
- **tremors** of head (sometimes body); twitching of lips, tail, ears; head shaking, nodding
- **limb paresis**: kneeling, walking on the knees, knuckling of fetlock joints, falling to sternal or lateral recumbency,
- **disturbed equilibrium** / incoordination: wide-based stance, splaying of digits, crossing of limbs during motion, falling over
- **hypermetria** of thoracic limbs
- **bounding, hopping or jumping movements** (both pelvic limbs moved together)
- muscle asynergy: limb stiffness, stilted or rigid gait, segmented execution of movements
- proprioceptive deficit: retarded correction of foot placement after fetlock flexion

- sheep recumbent for long periods (10-14 days) may be comatose or may convulse

Cattle

- **hyperexcitable** → difficult to handle
- mild incoordination
- mild hindlimb paresis
- **difficulty with chewing & swallowing**, inappetence (cranial nerve involvement)
- → weight loss, ↓ weight gain
- tongue protrusion (normal muscle tone & strength)
- saliva drooling

Pathology:

- **greenish pigmentation** in CNS (brain, spinal cord & dorsal root ganglia) and kidney medulla = indole-like pigments
- pigments thought to indicate neurones affected by the syndrome, but pigments themselves not responsible for dysfunction; stored in lysosomes
- CNS pigment distribution: neurones in brain stem nuclei (thalamus to caudal medulla), cerebellum and in spinal cord dorsal root ganglia and dorsal and ventral horn cells

Diagnosis:

- access + syndrome, pathology

Therapy: gentle removal to safe pasture

Prevention & control:

- **cobalt bullets** placed in rumen at start of each grazing season (probably → microbial detoxication)

- low-alkaloid cultivars of *P. aquatica* have been developed, but clinical disease has occurred on these. Reduction of dimethyl tryptamines may result in increases in β-carbolines and a shift in syndrome presentation to more chronic forms.
- immunoassay (ELISA) developed for tryptamine toxins and used in plant breeding program (Skerritt *et al.* 2000)

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McKenzie RA (1994) *Plant Poisonings of Horses in Australia.* Proceedings of the 16th Bain-Fallon Memorial Lectures, Australian Equine Veterinary Association, Artarmon, Sydney, pp. 1-56.
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ß-carboline alkaloids [indole alkaloids] – Coonabarabran staggers

Syndrome names: Coonabarabran staggers
Chemical structure:
ß-carboline alkaloids
Sources: *Tribulus terrestris* (caltrop) (*q.v.*)
Toxicity:
progressive **irreversible** nervous dysfunction
described only in sheep in NSW
Mode of action: undescribed (CNS effect)
Conditions of poisoning: hot, dry weather after drought-breaking storms → dense growth
Clinical signs:
duration 1-15 months
mild hindquarter incoordination
hindquarters lean to one side consistently → move on a diagonal
forelimb involvement → difficulty standing
death from thirst/starvation or misadventure
Pathology:
± demyelination of some peripheral nerves
± Wallerian degeneration of spinal cord white matter
± neurogenic degeneration skeletal muscles
Diagnosis: syndrome + access
Therapy: nil
Prevention & control: deny access to dense *T. terrestris* populations
References:

Se138
Allen JRF, Holmstedt BR (1980) The simple β-carboline alkaloids. *Phytochem.* **19**:1573-1582.

☑ *Piperidine, pyridine (nicotine) & quinolizidine alkaloids* - Conium, Lupinus, Nicotiana *teratogens*

Core data

Syndrome names: crooked calf disease
Common sources:

- *Conium maculatum* (hemlock) – coniine (piperidine)
- *Lupinus* spp. (lupins) – anagyrine (quinolizidine), ammodendrine (piperidine)

Animals affected: cattle, sheep, goats, pigs
Mode of action:

- induced ↓ foetal movement *in utero*

Poisoning circumstances:

- intake by pregnant females
- foetus susceptible at particular times during gestation

- anagyrine only affects cattle

Main effects:

- arthrogryposis
- palatoschisis

Diagnosis: syndrome + access
Therapy: nil
Prevention: deny pregnant stock access

Syndrome names: **crooked calf disease** (North American cattle + *Lupinus* spp.)
Chemical structure:

teratogens: anabasine (pyridine), coniine, ammodendrine (piperidines), anagyrine (quinolizidine)

Sources:

plant sources associated with disease (natural/experimental):
Family Apiaceae
Conium maculatum (hemlock - contains coniine) (Panter & Keeler 1989)
Family Fabaceae
Lupinus spp. (lupins - 14 species contain anagyrine, 1 contains ammodendrine); cultivated
Family Solanaceae
Nicotiana tabacum (tobacco - contains anabasine); cultivated in Australia (decreasingly)
Nicotiana glauca (tree tobacco - contains anabasine); naturalised in Australia (Purdie *et al*. 1982)

Plant genera with species yielding piperidine alkaloids with the apparent structure for teratogenicity: *Ammodendron*, *Carica*, *Cassia*§, *Collidum*, *Conium*§, *Dichroa*, *Duboisia*§, *Genista*§, *Hydrangea*§, *Liparia*, *Lobelia*§, *Lupinus*§, *Nicotiana*§, *Pinus*§, *Prosopis*§, *Punica*, *Sedum*, *Withania* (§ = in Australia)

Toxicity:

pigs, cattle, sheep, goats affected in **North America**
pigs/*Nicotiana tabacum* stalks
cattle/*Lupinus* spp. → `crooked calf disease'

Mode of action: (Panter *et al.* 1991, Panter 1993)

induced ↓ foetal movement *in utero*
direct relationship between degree of reduced foetal movement and severity of arthrogryposis and cleft palate
cleft palate from non-movement of tongue/jaws.
foetus susceptible at particular times during gestation

Cattle: 40-70 days palate, limbs, spine, neck
Pig: 30-41 days palate; 40-53 days forelimb, spine, neck; 50-63 days hindlimb
Sheep: 30-60 days palate, limbs, spine, neck
Goat: 35-41 days palate; 30-60 days palate & limbs

diurnal duration of reduced foetal movement influences severity of abnormalities

Conditions of poisoning:

intake by pregnant females
Lupinus spp. containing anagyrine are only teratogenic in cows - apparent inherent metabolic difference from other animals

Clinical signs: congenital deformity
Pathology: **congenital arthrogryposis** (± brachygnathia, palatoschisis, torticollis, scoliosis, lordosis)
Diagnosis: pathology + access
Therapy: nil
Prevention & control: deny access at susceptible times of gestation
References:

Se34
Finnell RH, Gay CC, Abbott LC (1991) Teratogenicity of rangeland *Lupinus*: the crooked calf disease. Chapter 2 in Keeler RF, Tu AT (eds) *Handbook of Natural Toxins. Vol.6. Toxicology of Plant and Fungal Compounds.* Marcel Dekker, Inc., New York. pp. 27-39.

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Purdie RW, Symon DE, Haegi L (1982) Solanaceae. *Flora of Australia* **29**:1-208

Pyridine (nicotine) and piperidine alkaloids

Sources:

Australian plant sources

Family Solanaceae – pyridine (nicotine) alkaloids

Nicotiana spp. [DM91] - 17 native tobaccos (including *N. velutina, N. megalosiphon, N. suaveolens*), 2 introduced (*N. glauca, N. tabacum*) - widespread in inland Australia (Purdie *et al.* 1982). Local abundance of species in Central Australia harvested by Australian aboriginal people for production of pituri quids (*Nicotiana gossei, N. excelsior, N. rosulata, N. benthamiana* in descending order of popularity) is positively correlated with recent burning of the vegetation as fire stimulates germination of dormant seeds (Latz 1995).

Duboisia hopwoodii (pituri) - arid zone from western Queensland border to Indian Ocean (Bancroft 1872, 1877, 1879, Purdie *et al.* 1982). Plants in Queensland and Western Australia contain mostly nicotine, while those in Central and southern Australia contain mostly nor-nicotine (Barnard 1952). Pollen is reputed to form a deadly poison for the enemies of Central Australian aboriginal people (Latz 1995).

Family Apiaceae (Umbelliferae) – piperidine alkaloids

Conium maculatum (hemlock)

Mode of action:

nicotine and related pyridine alkaloids act on various neuro-effector junctions; piperidine alkaloids similarly

the alkaloids have both stimulant and depressant phases → confusing clinical signs

Toxicity & Conditions of poisoning:

plants unpalatable

Nicotiana spp.

hungry travelling cattle & sheep

dry months (Aug-Nov)

access to green *Nicotiana* plants after crossing dry/bare stock routes

Duboisia hopwoodii

grazing horses, cattle, sheep, goats, camels

root suckers harvested in hay

used by Central Australian aboriginal people to poison waterholes for the capture of emus (Latz 1995)

C. maculatum containing coniine → poisoning of cattle, pigs very similar to nicotine alkaloids. Sheep resistant.

Clinical signs:

unwillingness to move, incoordination

muscle tremor, weakness

pupil dilation

recumbency, paddling → paralysis

± diarrhoea

Pathology:

alimentary tract congestion in cases with diarrhoea
Diagnosis: access + clinical signs
Therapy:
no specific therapy recommended
affected animals left undisturbed often recover
Prevention & control: prevent access
References: Se80

Bancroft J (1872) The pituri poison. *J. Qd. Phil. Soc.* 1-2. [cited by Hurst 1942]
Bancroft J (1877) Pituri and *Duboisia. J. Qd. Phil. Soc.* 3-13. [cited by Hurst 1942]
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Barnard C (1952) The *Duboisias* of Australia. *Economic Botany* **6**:3-17.
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☑ Erythrophleum *spp. (diterpenoid alkaloids & cinnamic acid derivatives)*

Core data

Common source: Erythrophleum chlorostachys (Cooktown ironwood); northern Australia
Animals affected: cattle, horses + others
Poisoning circumstances:

- all parts of plant toxic
- suckers most hazardous

Main effects:

- sudden death
- cardiac arrhythmia

Diagnosis: plant access
Therapy: no specific therapy

Chemical structure:

Highly cardiotoxic **diterpenoid alkaloids** cassaidine, cassaine and eryhtrophleguine (6α-hydroxycassamine) have been isolated from bark of *Erythrophleum guineense* and reported to have digitalis-like action on the heart (Ruzicka 1940, Turner 1966, Thorell 1968 [all cited by Harborne & Baxter 1996])

Petrie (1921a,b) and Petrie & Priestly (1921) isolated 0.002% alkaloid in leaf and 0.03% alkaloid in seed of *E. chlorostachys* and compared it to that from *E. guineense* ("erythrophleine") by tests in frogs and dogs. The alkaloid was not characterised further.

Griffin *et al.* (1971) extracted 0.5% crude alkaloid from leaf of *E. chlorostachys* from Mareeba, northern Queensland. Four compounds were identified in this crude extract: β-dimethylaminoethyl cinnamate, *N*-2hydroxyethyl-*N*-methylcinnamamide, *N*-2-hydroxyethyl-*N*-methyl-*trans*-*p*-hydroxycinnamamide and *N*-hydroxyethylcinnamamide. It was considered that the corresponding esters were more likely to be the actual compounds present in the original leaf. The toxicity of these compounds were not reported. Samples of *E. chlorostachys* leaf from Darwin and Cooktown contained only the diterpene ester alkaloids found in other *Erythrophleum* spp. Thus there appear to be two chemical races of *E. chlorostachys* present in Australia. Only the diterpenoid alkaloids are recorded as toxic. No toxicity testing is recorded for the cinnamic acid derivatives.

Loder *et al.* (1972) reported the diterpenoid alkaloids norcassamidide and norcassamidine from the bark of *Erythrophleum chlorostachys*.

Loder (1975) reported the diterpenoid alkaloid norerythrostachaldine (19-oxonorcassaidine) from the bark of *Erythrophleum chlorostachys.*

Sources:

- toxic species occur in Australia, Africa and South-east Asia (Hauth 1974). Mabberley (1997) indicates that 9 species of *Erythrophleum* occur in the Old World tropics (Africa 4, Madagascar 1, Eastern Asia & Malesia 3, Australia 1). Species with animal/human toxicity cases on record = ♥
- Australia
    - ***Erythrophleum chlorostachys*** (ironwood, Cooktown ironwood) ♥ [DM147] Family Caesalpiniaceae [Leguminosae] - tree widespread in tropical Australia north of 20°S latitude (through about Townsville), mainly on sandstone country (Hall 1964), usually found in open eucalypt forest, woodland or savannah (Ross 1998)

Erythropleum chlorostachys (Cooktown ironwood) whole tree (left); leaf (right). Note the unequal amount of tissue on either side of the midribs of the leaflets. [RAM Photos]

- Africa
    - *Erythrophleum africanum* ♥
    - *Erythrophleum gabunense*
    - *Erythrophleum guineense* (= *E. suaveolens*) ♥
    - *Erythrophleum ivorense* ♥
    - *Erythrophleum lasianthum* ♥
    - *Erythrophleum le-testui*
    - *Erythrophleum micranthum*
    - *Erythrophleum purpurascens*
- Madagascar
    - *Erythrophleum couminga*
- South-eastern Asia (Indochina, Thailand, Borneo)
    - *Erythrophleum angustifolium*
    - *Erythrophleum cambodianum*
    - *Erythrophleum fordii*
    - *Erythrophleum succirubrum* ♥
    - *Erythrophleum teysmannii*

- *Erythrophleum unijugum*

Toxicity:

***Erythrophleum chlorostachys*:**

Cattle, sheep, goats, horses & camels have been reported to be poisoned by *E. chlorostachys* (Bailey 1900, Hall 1964)

Rock ringtail possums (*Petropseudes dahli*) are reported to use *E. chlorostachys* leaf as a major food source in the Kakadu region of the Northern Territory (Runcie 2000)

Dried leaves of *E. chlorostachys* retain their toxicity (Hall 1964)

Toxic doses of *E. chlorostachys* determined experimentally:

- calf: 170 g leaf caused death within 24 hrs (Anon 1948)
- steer (295kg): 113 g dried leaf caused death in 30 hrs, and steer (227 kg): 57 g dried leaf caused severe non-fatal illness (LG Newton & MD McGavin, unpublished data 1953;cited by Hall 1964)
- horse: <55 g dried leaves caused death within 24 hr (Anon 1948)
- horse: 45 g dried leaves caused death within 50 hrs, and 23 g dried leaves caused non-fatal illness (LG Newton & MD McGavin, unpublished data 1953;cited by Hall 1964)

Other *Erythrophleum* spp.

Some African species have been used as ordeal poisons as part of the indigenous judicial system. Species reported toxic in Africa include *E. guineense, E. africanum, E. ivorense* and *E. lasianthum* (Nwude & Chineme 1981).

E. guineense (Nwude & Chineme 1980) and *E, africanum* (Nwude & Chineme 1981) have been confirmed as fatally toxic to sheep in feeding experiments at doses of 0.25 – 8.0 g leaf/kg (*E. guineense*) and 2.0 – 4.0 g leaf/kg (*E. africanum*).

Mode of action: unknown

Conditions of poisoning by *E. chlorostachys*:

- all plant parts are highly toxic, but young leaves of **suckers** are the most hazardous because they are most accessible to grazing livestock (Bailey 1900, Hall 1964)
- cattle travelling on the Murranji stock route in the Northern Territory have been poisoned when feed is scarce (Anon 1952)
- bulls exposed to branches caught in the sides of motor trucks traversing an overgrown road in northern Queensland died soon after unloading (Hall 1964)
- horses tied up under *E. chlorostachys* trees in Croydon, north Queensland (Hall 1964)
- cattle introduced to the "Top End" of the Northern Territory for live export to Asia (Thompson 1999)

Clinical signs:

Effects are similar to acute cardiac glycoside poisoning.

Erythrophleum chlorostachys:

Clinical signs common to all affected species (Hall 1964):

- anorexia
- mucosa pallor
- loud & irregular heart sounds
- diarrhoea with blood
- apparent disturbed vision (staring eyes)
- frequent contraction of abdominal muscles
- dyspnoea (terminal)

Additional signs seen in horses: profuse sweating, periodic extrusion of the upper and lower lips (Hall 1964)

Additional signs seen in sheep: groaning, apparent blindness, some develop diarrhoea (Bailey 1900)

Other *Erythrophleum* spp. (African) produce (depending on dose) sudden death, depression, teeth grinding, profuse diarrhoea, trembling, recumbency, dyspnoea in sheep (Nwude & Chineme 1980, 1981). *E. succirubrum* seeds have caused poisoning of children in Thailand, resulting in vomiting, diarrhoea, tachypnoea and cardiac arrhythmia (Echeverria *et al.* 1986).

Pathology (Hall 1964):

- recognisable leaves (or other plant parts) present in rumen contents (whole in some cases) (Bailey 1900)
- gastrointestinal congestion

- ± haemorrhagic colitis
- subepicardial, subendocardial and myocardial haemorrhage

Diagnosis: history of access + sudden death + identify plant in ingesta

Therapy:

- nil recorded
- possibly worth applying therapy used for cardiac glycosides (activated charcoal, fluids, antiarrhythmic drugs)

Prevention & control: prevent access

References: Se50 - note incorrect spelling

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Ross JH (1998) *Erythrophleum. Flora Aust.* **12**:70-72.

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Thorell A (1968) *Acta. Chem. Scand.* **22**:2835 [cited by Harborne & Baxter 1996]

Turner RB (1966) *J. Am. Chem. Soc.* **88**:1766. [cited by Harborne & Baxter 1996]

Diterpenoid alkaloids - Delphinium *spp.*

Plant sources: *Delphinium* spp. (larkspurs) in North America

Ruminants mostly affected

Sudden death or syndrome dominated by weakness, dyspnoea, collapse.

Therapy: physostigmine IV, IP or SC @ 0.04-0.08 mg/kg (Pfister *et al.* 1994)

References:

Pfister JA, Panter KE, Manners GD, Cheney CD (1994) Reversal of tall larkspur (*Delphinium barbeyi*) poisoning in cattle with physostigmine. *Vet. Human Toxicol.* **36**:511-514.

Tropane alkaloids [scopolamine (=hyoscine), hyoscyamine, atropine and others]

Sources:

Family Solanaceae

**Atropa belladona* (deadly nightshade) – very rare in Australia, native of Europe

Hyoscyamus spp.

**Hyoscyamus niger* L. (black henbane) - rare in Australia, native of Europe

**Hyoscyamus albus* L. (white henbane) - rare in Australia, native of Europe

Duboisia spp. (corkwoods) (Purdie *et al.* 1982)

Duboisia leichhardtii (corkwood) [DM152]

Duboisia myoporoides (corkwood) [DM152] (Barger *et al.* 1937, 1938)