Macrozamia miquelii leaf showing the distinctive features of the genus. Note the lack of midribs in the pinnae (leaflets) and the pigmented/pale patches at the base of the pinnae. [RAM Photo]

Mature female cones of *Macrozamia miquelii* before break-up and dispersal of the orange-coloured seeds. [RAM Photo]

Macrozamia moorei mature plant in natural habitat - the largest species of this genus. [RAM Photo]

Cultivated mature female specimen of *Macrozamia lucida*, a medium-sized example of this genus. Note the mature female cone containing red seeds. [RAM Photo]

Mature plant of *Macrozamia heteromera* in natural habitat, an example of a small member of this genus [RAM Photo]

***Lepidozamia* spp.** (tropics, subtropics - Q, NSW)

2 species, both in Australia (Hill 1995, 1998; Jones 1993; Hill & Osborne 2001)

Lepidozamia peroffskyana Regel contains hepatotoxins (Gobé & Pound 1985) but is not known to have caused neurotoxicity; distributed in south-eastern Qld and north-eastern NSW from the ranges north-west of Brisbane to the Manning River district; cultivated

Lepidozamia hopei (W.Hill) Regel is not recorded as toxic; distributed in rainforest and wet sclerophyll forest in north-eastern Qld from Rockingham Bay to the Bloomfield River; cultivated

Family Stangeriaceae

***Bowenia* spp.** (tropics – Q)

2 species, both in Australia (Hill 1995, 1998; Jones 1993; Hill & Osborne 2001)

Bowenia serrulata (W.Bull) Chamb. (Byfield fern [*sic*]) (Hall & McGavin 1968, Seawright *et al.* 1998b) - distributed in central coastal Qld around Byfield, north-east of Rockhampton; cultivated

Bowenia spectabilis Hook. Ex Hook.f. (zamia fern [*sic*]) - distributed in and around rainforests in north-eastern Qld on the coast and ranges from Cardwell to Cooktown with a population in the McIlwraith Range on Cape York Peninsula. The population near Tinaroo Dam, previously considered a possible separate species, is included in this taxon. Cultivated. - has been associated with field cases in cattle

Mature *Bowenia serrulata* plant in natural habitat, with leaves sprouting from the underground trunk. [RAM Photo]

America

Poisonings on Caribbean islands (Dominican Republic, Puerto Rico) and in Florida involve *Zamia* spp. probably including *Zamia integrifolia* L. f. in Aiton (coontie, Florida arrowroot), *Zamia pumila* L. and *Zamia portoricensis* Urban (e.g. Mason & Whiting 1968, Reams *et al.* 1993). Cycads in the genera *Ceratozamia, Chigua, Dioon, Microcycas* and *Zamia* occur variously in Mexico and in Central and South America and many contain MAM glycosides, but no posterior ataxia syndrome has been recognised in livestock except those noted above.

Japan

Poisonings on southern islands of Japan involve the local species *Cycas revoluta* (sago "palm") (Kobayashi *et al.* 1983, Yasuda *et al.* 1985)

Africa

Cycads in the genera *Encepalartos* and *Stangeria* occur and contain MAM glycosides, but no posterior ataxia syndrome has been recognised in livestock.

For recent detailed information on cycad biology, see the books of Norstog & Nicholls (1997) and Jones (1993, 2002) and the Proceedings of the 4 International Conferences on Cycad Biology (Stevenson 1990, Stevenson & Norstog 1993, Vorster 1995, Chen 1998)

Toxicity:

The syndrome is seen in **cattle** and rarely sheep. Water buffalo (*Bubalus bubalis*) are reported as susceptible (Anon. 1988)

Australian native birds (e.g. emus, cockatoos) and mammals (brush-tailed possum, chudich *Dasyurus geoffroyi*) are agents of seed dispersal and consume the sarcotesta of *Macrozamia*

seeds, suggesting an innate resistance to toxicity (Burbidge & Whelan 1982, Ladd *et al.* 1993). *Rattus fuscipes* is a consumer of *Macrozamia communis* seed (Ballardie & Whelan 1986).

MAM is carcinogenic (see references below)

Humans and cycads

Indigenous peoples in several parts of the world have used and do use parts of cycads as the basis of food and beverages after processing to reduce or remove toxins. Documentation of indigenous cycad use includes that of Thieret (1958) and Whiting (1963), and in Australia, Beaton (1982), Horsfall (1987) and Beck (1993). Beaton (1982) notes the use of seeds of *Macrozamia* spp. and *Cycas* spp. Horsfall (1987) notes the use of *Cycas media* seed*, Lepidozamia hopei* seed and *Bowenia spectabilis* subterranean parts ("rhizomes") by north-eastern Queensland aboriginal people. Beck (1993) notes the use of seeds of *Cycas* spp. including *C. angulata* and (probably) *C. armstrongii* in northern Australia.

A group of neurological syndromes in humans on Guam in Micronesia (Mariana Islands) has been postulated as associated with the use of seeds from the local cycad species, *Cycas micronesica* (formerly classified in *C. circinalis*), as food by the local Chamorro people. The syndrome complex has close similarities with amyotrophic lateral sclerosis (ALS), Parkinson's disease (parkinsonism) and dementia and is called the amyotrophic lateral sclerosis / parkinsonism-dementia complex (ALS/PDC). The causal connection between cycads and the syndrome complex, known locally as lytico-bodig, is unproven and other putative causes (all also unproven) include slow viruses and mineral toxicities. For a popular account of this phenomenon, see Sacks (1996). Seawright *et al.* (1995) studied the possible involvement of cycad pollen in ALS/PDC.

Mode of action: undetermined

Conditions of poisoning:

substantial intake of **young fronds** or of **seeds**

Macrozamia moorei (Carnarvon Ranges, Queensland): The average cattle morbidity/mortality is 1% of the herd, rising to 3-5% in particular years, with greater prevalences (up to 40-50%) in certain paddocks. Seasonal conditions predisposing to higher overall prevalence appear to be good rains in summer followed by none through autumn and winter. This reduces pasture nutrients at the same time that the cycads produce a heavy seed crop. The seeds fall from the cones beginning in autumn when pasture quality is rapidly declining. Clinical cases begin to occur in winter. (John Watkins, grazier, Springsure district, personal communication 28 August 2001)

young green fronds become available after plants and their habitat is burnt and little other feed is available. These leaves are palatable to cattle (Wesley-Smith 1973, Everist 1981)

Macrozamia moorei (Carnarvon Ranges, Queensland): Morbidity/mortality of the order of 50% has been reported after a herd was exposed to young regrowth after a bushfire (John Watkins, grazier, Springsure district, personal communication 28 August 2001).

experimentally, cattle have to be fed the plants daily for several months (at least 50 days) before clinical signs develop (Seawright *et al.* 1998b)

Clinical signs:

The syndrome is **irreversible** (in contrast with *Xanthorrhoea* poisoning - *q.v.*) and is a manifestation of **proprioceptive dysfunction** causing

- an irregular stiff goose-stepping gait in the hindlimbs
- knuckling of the hind fetlocks
- muscle atrophy in the hindquarters
- and finally, posterior paralysis

Experimental *Macrozamia* toxicity. Note drooping of hindquarters and knuckling of hind fetlocks. [WTK Hall photograph]

Pathology:

Degeneration of spinal cord white matter occurs in the fasciculus gracilis, dorsal spinocerebellar & corticospinal tracts.

Chronic liver damage (fibrosis, enlarged hepatocytes) may result in ataxic cattle from the action of MAM

Diagnosis: access + pathology

Therapy: nil

Prevention & control:

Effective and practical prevention and control measures can be difficult to achieve under Australian conditions. In summary, **preventing access of livestock to plants with fruit or young fronds** underlies all measures. Plants may be killed with herbicides or physically removed from pastures and cattle may be excluded from dense populations by strategic fencing. Where neither option is economical or practical, attempts can be made to reduce intake of cycads by providing supplementary feeding. The efficacy of this latter approach has not been scientifically assessed.

Be aware of the **legal requirements for the conservation of cycads**. Consult the nature conservation authorities at state government level for the precise legal obligations of landholders and others under each jurisdiction throughout Australia. For example, in Queensland, the *Nature Conservation Act 1992* and the associated schedules in the *Nature Conservation Regulation 1994* both apply. See these for precise interpretations in a given situation. In summary, all species of *Cycas, Lepidozamia, Macrozamia* and *Bowenia* (including any naturally occurring hybrids), which are not listed as rare or threatened [Schedules 1, 2, 3 & 4 - Presumed extinct, Endangered, Vulnerable and Rare respectively] are listed in Schedule 5 as common plants in trade. Clearing on state land (leasehold land) requires a tree clearing permit under the *Land Act 1962* and also under the *Nature Conservation Act 1992*. A single (tree clearing) permit will suffice provided consultation between the Department of Environment and the Department of Natural Resources has been undertaken. On private land (freehold land) there are no controls on destruction *except* for those species listed in Schedules 1, 2, 3 & 4. This means that all except the rare or threatened species can be destroyed but can also be harvested and sold under commercial harvesting licences. If applications for selective clearing are made because of toxicity to stock and cannot

be accommodated by either of the above options then consideration can be given for issuing a damage mitigation permit for rare or threatened species. If the applicant can demonstrate a lack of other options, the plants may be destroyed or may be salvaged by the holder of a harvesting licence.

Cycad-free reserves
Where cycad populations are widespread and dense, consider removing cycads from suitable small paddocks to be **reserved** for use in seasons when the plants are producing fresh leaves or fruit and other feed is scarce. Careful removal and use of the cycads for horticultural / landscape architectural purposes is the preferred option for disposal (see legal considerations above).

Herbicide control (all data in this section from Vitelli 1993)
Chemical control of cycads is difficult. Reshooting from the base of the plant will occur if control techniques are not thorough. Effective herbicides include the following

Herbicide	Application rate	Carrier	Application Method	Efficacy
Access®	1:100	Diesel	Foliar spray	100%
Garlon 600®	1:30	Diesel	Foliar spray	100%
Brush-Off®	30g/100L	Water + wetting agent	Foliar spray	100%
Arsenal®	1:50	Water	Foliar spray	90%
Tordon 50D ®	1:3	Water	Injection*	70%
Tordon 75D ®	1:4	Water	Injection*	

* Inject 1ml per cut into the growing point

In northern Australia, best results are achieved by **integrating fire with chemical application**. The area to be treated is burnt in August-September [destroys existing fruit, interrupts cycad fertilisation, stimulates young leaf growth] then actively-growing plants are sprayed overall with herbicide.

Supplementary feeding of at-risk cattle (all data in this section from Laing 1993)
Cattle producer experience in northern Queensland suggests supplementation reduces the incidence of cycad poisoning cases; *scientific evaluation is required*. Recommendations are:

Wet season: lactating cows 10 g P/day, dry stock 5g P/day; deliver as loose mixes in roofed feeders or as blocks

Dry season, adequate standing feed: urea blocks and loose mixes including P @ 150g protein/adult/day, 75-100g protein/weaner/day [Urea is 46% nitrogen, or 288% protein equivalent (46 x 6.25). Thus 150 g protein would equate to 150 x 1/6.25 = 24 g N = 24 x 100/46 = 52.2 g urea.]

Dry season, inadequate standing feed: feeding urea and loose mixes may encourage cycad consumption, thus add energy to supplements – suitable & economical supplements include M8U* + P, M8U + 4% protein meal +P, M3U + 8% protein meal + P

[*M8U is molasses with 8% urea (usually fed *ad libitum*), i.e., for every 100 kg molasses there is inclusion of 8 kg urea - not strictly 8% urea as 8 in 108 kg, but for producers it is easier for them to calculate how much urea to add based on weight of molasses in tank, in this way. Similarly M3U is molasses + 3% urea - 3 kg urea to every 100 kg molasses. The former mixture is less palatable and thus the cattle eat less of it voluntarily. The 4 or 8% protein meal referred to are feed sources such as cottonseed meal which appear to give some benefit over and above that of urea.]

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Nervous – Ataxia – Family Amaranthaceae

Gomphrena celosioides *(gomphrena weed, soft khaki weed)*

Unknown neurotoxin

Sources: *Gomphrena celosioides* (gomphrena weed, soft khaki weed) in Family Amaranthaceae

Toxicity:

- horses
- toxin unknown

Mode of action: undescribed

Conditions of poisoning:

- coastal Queensland
- horses confined to pasture dominated by *G. celosioides*.

Clinical signs:

- depression
- posterior ataxia
- toe dragging
- difficulty turning
- progressive → all 4 limbs affected → difficulty standing, fall easily

Pathology: no lesions reported

Diagnosis: syndrome + access

Therapy: nil

Prevention & control: deny access to dense populations of *G. celosioides*

References: Se58, DM95

Newton LG (1952) *Gomphrena celosioides* - a plant causing ataxia in horses. *Aust. Vet. J.* **28**:151-154.

Nervous – Ataxia – Family Asclepiadaceae

Hoya australis *(hoya, wax flower)*

Chemical structure:

Neurotoxin unknown: ? cynanchosides

Sources:

Hoya australis (hoya, wax flower) [DM118] which has 5 subspecies according to Forster & Liddle (1996) who nominate *H. australis* ssp. *australis* as toxic to livestock.

Hoya is a large genus of climbing (twining) plants of over 300 species (Forster *et al.* 1999). The 6 native and 1 naturalised Australian species are described by Forster & Liddle (1996) . 37 species commonly cultivated in Australia are described by Forster *et al.* (1999).

Toxicity:

- cattle, sheep in inland Queensland
- guinea pigs fed fresh leaf developed 'paralysis' to varying degrees after one to two days and either died or recovered (Bailey 1915)
- 2 sheep drenched with fresh leaf at 17 and 29 g/kg respectively and 1 sheep drenched with juice expressed from leaf at 34 g leaf/kg died in 2 days (Legg & White 1939a,b)
- toxic dose for calves of whole plant (leaf + stem) was 0.8% of body weight (8 g/kg); toxic dose of stem for calves was 0.2-0.4% of body weight (2-4 g/kg) for calves and sheep (Hall 1969)
- toxin does not appear to be water-soluble (Hall 1969)
- dried leaf appears to lose toxicity (Hall 1969)

Mode of action: undetermined

Conditions of poisoning:

- access to plants in softwood scrubs (depauperate or "dry" rainforests) during drought conditions
- plants are succulent and can act as a water source for cattle

Clinical signs:

- incoordination → collapse of forequarters or hindquarters
- muscle tremors
- frequent kneeling
- knuckling of fetlocks
- recumbency
- clonic or tetanic spasms aggravated by stimulus
- nystagmus

Pathology: no significant lesions reported

Diagnosis: access + syndrome

Therapy: mildly-affected animals recover unaided

Prevention & control: deny access to large populations of plant

References:

DM118, Ev102
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Nervous – Ataxia – Family Asteraceae

☑ Hypochoeris radicata – *presumptive cause of `Australian' stringhalt*

Core data

Common sources: syndrome associated with *Hypochoeris radicata*

Animals affected:

- horses
- large horses more susceptible

Mode of action: distal axonopathy of longest nerves
Poisoning circumstances: pasture often dominated by *H. radicata*
Main effects:

- exaggerated uncontrolled hock flexion progressing to inability to move
- laryngeal dysfunction
- muscle atrophy (hind limbs)

Diagnosis: syndrome
Therapy: time + phenytoin
Prevention:

- improve pasture
- reduce *H. radicata* density

Syndrome names: Australian stringhalt
Chemical structure:

The neurotoxin responsible for the syndrome is **unknown**.

Sources:

Numerous cases are associated with pastures heavily infested with ***Hypochoeris radicata*** (cats ear, flat weed, false dandelion) [Family Asteraceae] [DM66]. Not all cases are associated with this plant, there being some association with *Taraxacum officinale* (dandelion) (Araya *et al.* 1998), *Malva parviflora* (marsh mallow), other plants or no particular plant species.

Toxicity:

Horses, particularly larger individuals and Thoroughbreds and draft breeds, are affected. In a study group in Victoria, 84% of affected animals were over 15 hands high (P. Huntington, personal communication 3 November 2000). Cases are reported from Australia, New Zealand (Cahill *et al.* 1985), North America (Galey *et al.* 1991, Gay *et al.* 1993) and South America (Araya *et al.* 1998).

Classically, multiple cases occur in a herd (10-15% of individuals) with onset during summer-autumn [January-March] in southern Australia, then spontaneous recovery. Single cases are common

Attempted reproduction by feeding *H. radicata* (Seddon & Belschner 1926; Blythe *et al.* 199?) has been unsuccessful to date. The size of horses used for experimental work with this syndrome may be a critical factor, with the largest horses yielding the greatest probability of success.

Mode of action:

distal axonopathy of peripheral nerves (↑ size of horse → ↑ susceptibility)

Conditions of poisoning:

larger adult horses (foals can be affected)
onset in late summer to autumn in southern Australia (winter rainfall zone)
pasture dominated by *H. radicata* in many cases
cases have been reported from southern Chile where *H. radicata* was absent, but *Taraxacum officinale* (dandelion) was present (Araya *et al.* 1998)

Clinical signs:

- sudden **exaggerated uncontrolled hock flexion**, usually bilateral. Affected horses are unable to back. Severity of signs vary from day to day, being most severe in cold weather and when the horses is excited. Severely-affected cases are unable to move except by "bunny hopping". Many horses have a progressive deterioration for a few weeks, then a period of stability followed by gradual recovery. (P. Huntington, personal communication 3 November 2000)
- **muscle atrophy** in hind legs, particularly in the digital extensor group (gaskin) with many horses having wastage of the thighs and between the hindlegs. In some cases (usually Draft horse breeds) there is gait abnormality and muscle atrophy in forelimbs (P. Huntington, personal communication 3 November 2000).
- **laryngeal asynchrony & hemiplegia**/paralysis (later onset, longer persistence than gait abnormality) → less vocalisation than normal, muted/hoarse

Pathology:

distal axonopathy of longest nerves in the body (recurrent laryngeal, peroneal) (Cahill *et al.* 1986)
neurogenic atrophy of associated muscles (Cahill *et al.* 1986)

Diagnosis:

clinical syndrome

differentiated from "classical" stringhalt (Crabhill *et al.* 1994) by having multiple cases and spontaneous recovery

Therapy/Management:

untreated recovery occurs from a few days to 18 months (median 6-12 months).

remove horses from the paddock and hand feed or increase hand feeding while leaving the horses in the paddock

phenytoin in severe cases → ↓ clinical severity, ↓ recovery time [15 mg/kg *per os* each morning for 15 days with a 2nd dose each evening] (Huntington *et al.* 1991)

horses that need to be moved/transported should be sedated as this tends to reduce the severity of the signs (P. Huntington, personal communication 3 November 2000)

various other therapy has been used including dosing with the vitamin B group (including thiamine), vitamin E and magnesium, but there are no published data with which to evaluate their effectiveness (P. Huntington, personal communication 3 November 2000).

surgery (as used to treat classical stringhalt) is of questionable value, given the excellent long-term prognosis without intervention.

Prevention & control:

reduce *H. radicata* population density (mechanical removal, herbicides)

actively improve pastures

References:

DM66

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Tyler CM, Davis RE, Begg AP, Hutchins DR, Hodgson DR (1993) A survey of neurological disease in horses. *Aust. Vet. J.* **70**:445-449. [10 cases recorded; no data on exposure to *H. radicata* or other plants]

Nervous – Ataxia – Family Fabaceae

Chamaecytisus proliferus *(tagasaste)*

Syndromes:

Two syndromes are reported to follow consumption of this plant

- congenital leucoencephalopathy of calves
- a staggers syndrome in cattle

Chemical structure:
- toxin unknown; quinolizidine alkaloids possibly involved

Sources:
- *Chamaecytisus proliferus* ssp. *palmensis* [= *Cytisus proliferus*] (tagasaste, tree lucerne, escobon [Spain]); Family Fabaceae; endemic to the Canary Islands (Francisco-Ortega *et al.* 1991)
- used as a fodder shrub in Australia and New Zealand (Francisco-Ortega *et al.* 1991)

Toxicity:

Congenital leucoencephalopathy:
- cattle (neonates)
- Western Australia (West Midlands district); several herds affected (Forshaw 1999, Norris 1999)

Staggers:

A staggers syndrome in cattle grazing tagasaste is recognised in Western Australia; literature reports are scanty and somewhat conflicting, namely
- no gross or microscopic lesions have been found; cattle usually recover if left alone despite continuing to graze tagasaste (Forshaw 1999)
- some cattle have died and have had severe kidney damage and fatty change in livers (Main 1994)

Mode of action: unknown

Conditions of poisoning:

Congenital leucoencephalopathy:
- dams browsing tagasaste in late pregnancy (Norris 1999)

Clinical signs:

Congenital leucoencephalopathy:
- >60% calves may be affected (Norris 1999)
- hypermetria of forelimbs
- mild-to-severe hindlimb ataxia
- muscle tremors of hindlimbs, worsening with exercise
- ± unable to stand
- sucking reflex, fear, blink and withdrawal reflexes all present
- death within 4-5 days of birth

Pathology:

Congenital leucoencephalopathy:
- histological lesions of **CNS white matter** (Main 1998): vacuoles and faintly eosinophilic plaques 40-50 μm in diameter (H&E), plaques slightly pink (PAS) or non-staining (LFB) with special stains; some plaques contained peripheral nuclei resembling those of oligodendroglia; plaques contained normal axon segments demonstrated in LFB-Holmes silver stained sections; occasional microglial phagocyte in empty axonal tubes but no other inflammation; glial cell reaction limited to occasional isolated dense shrunken microglial nuclei near injured axons
- moderate-severe lesions of white matter in internal capsule, midbrain, medulla and cerebellum; severity increasing towards the mid and hindbrain
- lesions present in optic tracts and chiasma, but not optic nerves or retina
- severe lesions in spinal cord white matter, but not cauda equina or peripheral nerves
- lesions similar to those of progressive ataxia of Charolais cattle in which signs occur at 8-24 months of age and progress over 1-2 years(Blakemore & Palmer 1974)
- ultrastructure of CNS lesions (Forshaw 1999): plaques = intramyelinic expansions containing numerous vesicular membranous profiles and myelin bodies dispersed throughout a granular matrix possibly containing remnants of microtubules; remnant myelin sheaths around these foci and very thin with only a few lamellae. Around the plaques, numerous glial cells and their processes are similarly affected with numerous vesicular profiles; oligodendrocytes and possibly astrocytes affected. Some smaller diameter myelinated fibres have periaxonal vesiculation, apparently from the inner lamellae of myelin sheathes. Some brains have focal plaques of disorganised and tangled myelin.

Diagnosis:

Congenital leucoencephalopathy:

syndrome + dam access

Therapy: nil

Prevention & control:

Congenital leucoencephalopathy:

deny access or supplementary feed dams in late pregnancy??

References:

Blakemore WF, Palmer AC (1974) Progressive ataxia of Charolais cattle associated with disordered myelin. *Acta Neuropathol.* **29**:127-139.

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Nervous – Ataxia – Family Iridaceae

Romulea rosea *var.* australis *(onion grass, Guildford grass)*

Syndrome names:

- "romulosis"
  - infertility & abortion; testicular hypoplasia (?)
  - "autumn staggers" or "onion grass staggers" – ataxia & paralysis
- phytobezoars

Toxin:

The toxin responsible for romulosis is unknown. The leaf-spot fungus *Helminthosporium biseptatum* was suspected of involvement in romulosis and feeding trials with mice and guinea pigs demonstrated depressed fertility (Fisher & Finnie 1967), but this could not be confirmed later (Culvenor 1974).

Source:

Romulea rosea var. *australis* [= *R. longifolia, R. bulbocodium*]; Family Iridaceae (*not* a grass); Widespread naturalised exotic weed in southern Australia (Cook 1986).

Toxicity:

Romulosis (reproductive effects)

Sheep are affected. About 18 incidents were recorded during 1946-57 (Gorrie 1962).

Romulosis (ataxia/paralysis)

Sheep are affected. Affected flocks have reported more than 10% of sheep with the nervous syndrome (Galvin 1998).

Phytobezoars

Horses, cattle and sheep are affected. Reported to form phytobezoars in the stomach of horses and the abomasum of cattle and sheep in Victoria (Eckel 1964, Salisbury 1967, Pitt 1976, Clem & Johnston 1977) and New South Wales (Helms 1901).

Circumstances of poisoning:

Romulosis (reproductive effects)

Sheep: Summer-mated ewes grazing *R. rosea*-dominant pastures in late summer and autumn (McDonald & Eckel 1968). McDonald & Eckel (1968) suggested that prolonged ingestion of dry onion grass and its seed may underlie the disease.

Romulosis (ataxia/paralysis)

Sheep grazing *R. rosea*-dominant pastures in autumn. Onion grass as a significant if not dominant component of pasture following drought (Rifkin 1984).

Phytobezoars

Plants dominating available pasture in dry autumns in Victoria, leading to intestinal obstruction in the following spring when pasture is lush (Eckel 1964, Pitt 1976, Clem & Johnston 1977)

Clinical signs:

Romulosis (reproductive effects)

- Infertility manifest as sporadic very low lambing percentages associated with antenatal and perinatal mortality (McDonald 1966, McDonald & Eckel 1968). Lamb-marking rates can be down to 1-2% (Gorrie 1962, Eckel 1964), 10% (Galvin 1998). Recovery does not always occur in the subsequent season (Eckel 1965).
- mostly mid-term abortion (Gorrie 1962)
- vulvitis (Eckel 1964)
- testicular hypoplasia has been associated with onion grass in one report (Galloway *et al.* 1992).

Romulosis (ataxia/paralysis)

"Autumn staggers": sheep have developed a "spread-eagle" attitude when attempting to walk. Intermittent staggers and collapsing have been followed by death in up to a week (Eckel 1964). Involuntary flexion of fetlocks, followed by increasingly severe ataxia, walking on the knees, sternal recumbency, lateral recumbency and then death from intercurrent disease of misadventure if animals are not individually cared for (Rifkin 1984). Signs include knuckling of the forelimbs and hindlimbs (due to peripheral nerve damage), which progresses until sheep eventually become recumbent. There have been reports of sheep recovering following 7-12 weeks nursing, but many die in the meantime (Galvin 1998).

Phytobezoars

Signs of alimentary tract blockage: depression & anorexia, ruminal atony, dehydration, scanty inspissated faeces to no faeces (Spalding 1973, Rendell 1991). Colic is not a feature of the disease in cattle (Spalding 1972).

Pathology:

Romulosis (reproductive effects)

fluid accumulation noted in non-pregnant uteri (Gorrie 1962)

Romulosis (ataxia/paralysis)

Histological lesions attributed to this disease included degeneration of myelin with axonal swelling and fragmentation, particularly of motor nerve roots. Peripheral nerves were less affected. There was usually a light infiltration of lymphocytes (rarely more than one cell layer deep) about blood vessels in the brain stem. There was mild swelling of axons and nerve sheaths in the ventro-medial columns of the spinal cord white matter. In more chronic cases, macrophages containing golden-brown lipochrome pigment were found in perivascular spaces in the brain stem, cerebellar white matter and spinal cord. Rarely, there was also fine vacuolation of the white matter of the cerebrum, brain stem and cerebellum. (Rifkin 1984)

Phytobezoars

Bezoars in stomach/abomasum or blocking the tract downstream of these sites.

Diagnosis:

Syndrome + association with a pasture dominated by the plant.

Therapy:

Romulosis (reproductive effects)

Nil

Romulosis (ataxia/paralysis)

If affected sheep are nursed through the most severe phase of gait derangement lasting a few weeks, then subsequent recovery is reported to be very rapid and virtually complete in 3-5 days (Rifkin 1984).

Phytobezoars

Purgatives (Spalding 1972).

Surgical removal from valuable animals. Right flank laparotomy in cattle is usually successful if the animal is not recumbent at presentation (Rendell 1991). Rendell (1991) considered conservative medical treatment only delayed inevitable surgery, thus worsening the ultimate prognosis.

Control:

Reduce population density of *R. rosea* in pasture

References:

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Nervous – Ataxia – Family Liliaceae

Trachyandra *spp.*

Core data
Common sources: *Trachyandra divaricata , Trachyandra laxa*
Animals affected: sheep, horses
Mode of action: acquired lysosomal storage disease
Poisoning circumstances: pastures on sandy soil dominated by plant forcing large intake
Main effects:

- ataxia, posterior paresis
- lipofuscin accumulation in neurones

Diagnosis: histopathology
Therapy: nil
Prevention: deny access

Syndrome names: branched onion weed poisoning
Chemical structure:
- toxin/s unknown

Sources:
Trachyandra Kunth - about 45 species in Africa (Obermeyer 1962)
- ***Trachyandra divaricata*** (Jacq.) Kunth (branched onionweed)
  - native to southern Cape Province, South Africa (Hewson 1987)
  - in Australia naturalised in sandy soil in coastal south-western WA, on Eyre Peninsula and in the Flinders Ranges of SA and in the Sydney region of NSW (Hewson 1987)
- *Trachyandra laxa* in South Africa

Toxicity:
- sheep, horses
- cases in Australia (WA), South Africa

Mode of action:
apparent acquired lysosomal storage disease
severe neuronal lipofuscinosis is not invariable and cannot easily be related to the clinical deficits (Huxtable *et al.* 1999)

Conditions of poisoning:
- pasture on sandy soils dominated by the plants → stock forced to eat them during dry summer months

Clinical signs:
- onset after 4-6 weeks access

- lethargy, reluctance to move
- stand with arched back
- muscle tremor
- **ataxia**
- posterior paresis, **crouching stance → dog-sitting**
- recumbency

Pathology:
- no lesions at necropsy
- intense **lipofuscin pigment granule deposition in neurones** (brain, cord, ganglia)
- spheroids in spinal cord grey matter and brain stem
- axonal degeneration, demyelination, lipid storage by Schwann cells (Huxtable *et al.* 1999)

Diagnosis: histopathology
Therapy: no effective therapy
Prevention & control: deny access to dense plant populations
References: Se131

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Nervous – Ataxia – Family Mimosaceae

Prosopis juliflora *(mesquite) – neuronal vacuolation in cranial nerve nuclei*

Chemical structure:
- toxin unknown; piperidine alkaloids identified in seeds (Ahmad *et al.* 1978) but their relationship with this syndrome is unknown
- calystegines?

Sources:
- *Prosopis juliflora* (Sw.) DC var. *juliflora* (mesquite, algaroba, Cloncurry prickle bush, kiawe bean [Hawaii]) native of the Americas, introduced to Hawaii, Africa (Sudan), introduced to Australia where it is a weed of arid areas. [? = *Prosopis pallida* (Willd.) Kunth (Algaroba mesquite) (van Klinken & Campbell 2001)]

Note that species of *Prosopis* in Australia include *P. glandulosa* var. *glandulosa* (honey mesquite) and *P. velutina* [= *P. flexuosa*] (velvet mesquite, Quilpie mesquite, Quilpie algaroba) as well as *P. juliflora* var. *juliflora*, all of which are very similar and have been considered as one species by some authors (Perry 1998). Distinguishing features are: *P. juliflora* has soft herbaceous leaflets which appear somewhat curled or corrugated when dry; the internodes of the pinnae axes of *P. glandulosa* are considerably longer than those of the other two species and *P. velutina* has hairy pods (Perry 1998).

- used as feed for ruminants, pigs, poultry, rabbits, humans

Toxicity:
- cattle > goats (Tabosa *et al.* 2000)
- pods toxic when fed in large amounts for a long period
- cases recorded from Texas (Dollahite & Anthony 1957), New Mexico (Kingsbury 1964), Hawaii (Adler 1949, Hendershot 1946), Peru, Brazil (Tabosa *et al.* 2000)

Mode of action: undetermined
Conditions of poisoning:
- consumption of large amounts of the pods for a long period (8-12 months)

Clinical signs (Kingsbury 1964, Tabosa *et al.* 2000):
- profuse foamy salivation

- continuous chewing; tilting of the head during chewing
- rumen stasis, ↓ frequency of eructation → cessation of eructation
- **atrophy of masseter muscles**
- weight loss → emaciation
- abnormal jaw and tongue function
    - involuntary tongue movements
    - tongue protruding between the lips at rest
    - yawning
    - dysphagia (swallowing impaired)
- submandibular oedema (transitory)
- ± anaemia
- ± muscle tremors of the head and mandible, twitching of lips

Pathology:

- rumen full of mesquite pods & seeds
- denervation atrophy of masseter, temporal, hyoglossus, genioglossus, styloglossus, medial pterygoid, lateral pterygoid and mylohyoid muscles: marked variation in muscle fibre diameter, some myofibre degeneration, some fibrous replacement
- spongiosis & gliosis of CNS
- **trigeminal motor nuclei neuronal lesions**
    - loss of Nissl substance
    - **fine cytoplasmic vacuolation** of pericaryon or one pole of cell
- trigeminal ganglion lesions
    - loss of neurones
    - proliferation of satellite cells & neuronophagia
- Wallerian degeneration in trigeminal & mandibular nerves

Diagnosis:

- prolonged access + pathology
- differentiate from transmissible spongiform encephalopathies, lysosomal storage diseases, swainsonine & calystegine intoxication

Therapy: nil

Prevention & control:

diet should not exceed 50% *P. juliflora* pods for prolonged periods
plant control: fire (Campbell & Setter 2002)

References:

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Nervous – Ataxia – Family Proteaceae

Macadamia *spp. seeds/kernels (dogs)*

Chemical structure: Unknown toxin or toxins

Sources:

Seven *Macadamia* species (Family Proteaceae) are endemic in eastern Australia and 1 in Sulawesi, Indonesia (Gross 1995). *Macadamia tetraphylla, M. integrifolia* and hybrids (Macadamia nut tree, Queensland nut tree, bopple nut tree) are cultivated in Australia, Hawaii and elsewhere for the edible kernels of their seeds. Some kernels of these species contain traces of cyanogenic glycosides (Everist 1981; Orchard & Wilson 1999). *M. ternifolia* and *M. whelanii* kernels contain significant amounts of cyanogenic glycosides, are bitter and thus not currently

utilised as human food (Everist 1981). *M. whelanii* kernels are used by Aboriginal people after processing (Gross 1995).

Toxicity:

Dogs (no age or sex predisposition). Cases have been reported in Hawaii (Gfeller & Messonnier 1998) , Australia (McKenzie *et al.* 2000), USA (Hansen *et al.* 2000) and South Africa (C. Botha, personal communication 2002). Some dogs appear not to be susceptible, but this could be that the dose was insufficient to produce toxicity.

- unknown toxin responsible for syndrome below
- toxic dose estimated from clinical cases as
  - 0.7-4.9 g kernels/kg (mean 3.0 g/kg); kernel mean mass 2.5g; toxic dose range for 20 kg dog = 5-40 kernels (McKenzie *et al.* 2000)
  - 2.2-62.4 g kernels/kg (mean 11.7 g/kg) (Hansen *et al.* 2000)
- experimental intoxication induced by 20 g/kg PO: 4 dogs; depression, weakness (rear > forelimbs), vomiting (2 dogs), recumbency, mild hyperthermia but no joint pain induced (Hansen *et al.* 2000)

Mode of action: unknown

Conditions of poisoning:

- seeds of trees cultivated for human food toxic to dogs
- avid consumption of kernels or whole seed by dogs
- roasted kernels retain toxicity (including *Macadamia* butter made from crushed dry-roasted kernels)

Clinical signs (McKenzie *et al.*2000, Hansen *et al.* 2000):

- posterior paresis, ataxia
- muscle tremor (weakness)
- depression
- recumbency
- ± joint pain & swelling
- ± vomiting
- ± hyperthermia

Pathology: no lesions described

Diagnosis: access and syndrome

Therapy:

- no specific therapy; **signs resolve spontaneously in 12-24 hr**
- NSAIDs may be indicated for joint pain

Prevention & control: prevent excess kernel consumption

References:

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Nervous – Ataxia – Family Xanthorrhaceae

☑ Xanthorrhoea *spp. (grasstrees) – posterior ataxia syndrome*

Core data

Syndrome name: wamps

Common sources: Xanthorrhoea spp.

Animals affected: cattle

Mode of action: toxin unknown

Poisoning circumstances:

- consumption of flower spikes
- onset may be delayed weeks after removal from source

Main effects:

- reversible posterior ataxia

- urinary incontinence

Diagnosis: syndrome + access to flower spikes
Therapy: remove from source + supportive measures
Prevention: deny access to flowering plants

Syndrome name:

- **wamps** – onomatopoeia (pronunciation imitates sound of cattle falling and striking the ground)
- coastal disease
- wallum disease - refers to the geographical zone of occurrence in coastal sandy heath land with vegetation containing *Banksia aemula* (wallum banksia)

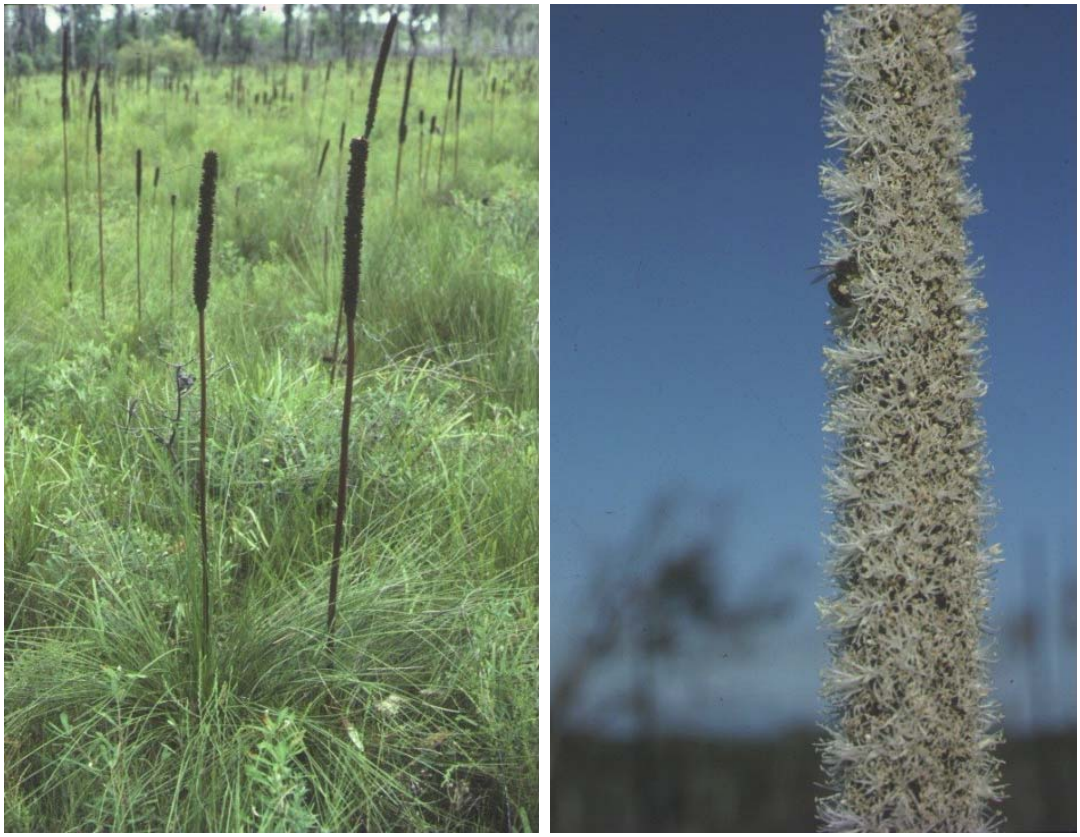
Neither coastal disease nor wallum disease is quite accurate as cases occur in inland areas as well.

Sources:

Family Xanthorrhoeaceae (Bedford *et al.* 1986)

- 28 species of ***Xanthorrhoea* (grasstrees, black boys)** are endemic to Australia (Bedford 1986), 4 have been associated with poisoning incidents, namely
  - ***Xanthorrhoea johnsonii*** (northern forest grasstree - Q) [DM60]
  - ***Xanthorrhoea fulva*** [= *X. resinosa, X. hastile*] (swamp grasstree - Q) [DM60]
  - *Xanthorrhoea australis* (yacca - Tas)
  - *Xanthorrhoea quadrangulata* or *Xanthorrhoea semiplana* (SA)

Mature *Xanthorrhoea johnsonii* (northern forest grasstrees) in natural habitat. [RAM Photo]



Mature *Xanthorrhoea fulva* (swamp grasstrees) in natural habitat with spent flower spikes (left), and a flowering spike to show the detail of the flowers (note honey bee for scale) [RAM Photos]

- *Lomandra longifolia* (spiny-headed mat-rush) and *Lomandra leucocephala* ssp. *leucocephala* (woolly mat-rush, irongrass) have been suspected of producing a very similar syndrome in cattle (RA McKenzie, unpublished data) but experimental confirmation of toxicity is lacking. There are 50 species of *Lomandra*, all native to Australia (Lee & Macfarlane 1986)

Cultivated flowering *Lomandra longifolia* (spiny-headed mat rush). [RAM Photo]

Toxicity:
- **cattle** in coastal Q, NSW, Tas

Mode of action: toxin unknown

Conditions of poisoning:
- **flower spikes** most toxic, but leaves have caused cases
- delayed onset after access may be up to 10 weeks

Clinical signs:
- syndrome **reversible** (cf. cycad poisoning)
- weight loss
- **posterior ataxia** (consistent sideways lurching of hindquarters to one particular side in an individual case)
- tail held high
- **urinary incontinence**
- fall easily, have difficulty rising

Pathology:
- ± degeneration of spinal cord, brain stem and cerebellar white matter. Lesions are usually slight. (RA McKenzie, unpublished data)

Diagnosis: syndrome + access

Therapy:
- remove from source & hand feed
- recovery in 2-3 weeks in most cases

Prevention & control: deny access to flowering plants

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Se149
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Nervous – Ataxia – Family Zygophyllaceae

Tribulus micrococcus *(yellow vine)*

Chemical structure:
- toxin unknown

Source:
Family Zygophyllaceae
Tribulus micrococcus (yellow vine)

Flowering and fruiting *Tribulus micrococcus* [RAM Photo]

Toxicity:
- sheep. NSW.
- clinically similar to `Coonabarabran staggers', but transient, **reversible**

Mode of action: unknown
Conditions of poisoning:
Clinical signs:
- hindlimbs move in lateral arcs, toes dragged
- fetlocks knuckle, hocks flexed
- hindquarters dragged → recumbency

Pathology: not reported
Diagnosis: syndrome + access
Therapy: nil
Prevention & control: deny access to dense *T. micrococcus* populations
References: Se139

Nervous – Ataxia – Family Lamiaceae, Malvaceae, Poaceae

Other plant staggers syndromes – Stachys arvensis, Lamium amplexicaule, Malva parviflora, Echinopogon *spp.*

Syndrome names: staggers, shivers
Toxicity:
- toxins unknown

- sheep mostly affected; also horses, goats, cattle
- young animals appear most susceptible; suckling lambs and foals reported affected (Dodd & Henry 1922)

Conditions of poisoning:

- after access to large concentrations of certain plants
- syndrome more prevalent in spring following a mild winter when herbage is more lush than usual (Dodd & Henry 1922, Seddon & Carne 1926)
- has occurred when only dried herbage available as fodder (Dodd & Henry 1922)

Clinical signs (general; Dodd & Henry 1922):

- animals usually normal while grazing in home paddocks; signs precipitated during forced exercise (mustering, droving)
- sudden onset
- stiff gait in hind quarters
- posterior ataxia
- hunched back, outstretched neck
- sweating (horses)
- dyspnoea
- muscle tremor
- sternal recumbency, muscles flaccid
- tachycardia, hyperthermia (40-41^{o}C) even in cool weather conditions
- forcing affected animals to exercise further leads to death

Pathology:

- no significant lesions reported (Dodd & Henry 1922), but see below under individual plant accounts

Plants:

- ***Stachys arvensis*** (stagger weed, hedge nettle [UK]) [DM90]; Family Lamiaceae
  - plants carrying seeds are more toxic than immature plants or plants from which seed has been shed (Seddon 1925, Seddon *et al.* 1926)
  - myelopathy and peripheral neuropathy detected in sheep [20-80% of 1000 Merino wethers at Young, NSW, affected in 2 successive years after grazing stagger weed spray-topped 1 week previously; stilted gait, dropping of the hindquarters when moving, knuckling of hind feet, often found recumbent; deaths from dehydration or misadventure] (Philbey 1991, Philby *et al.* 2001)
- ***Lamium amplexicaule*** (dead nettle; henbit); Family Lamiaceae
  - "staggers" or "shivers" reported in valleys of Namoi, Gwydir and Peel Rivers in northern New South Wales since 1895 (Dodd & Henry 1922, McBarron 1977); recorded in Queensland (White 1920-21, 1921, 1925, 1935)
  - mature plant contaminating cereal stubbles poses greatest risk of toxicity (Everist 1981)
  - experimental feeding of sheep from locations where the syndrome was unreported + forced exercise reproduced the syndrome:
    - 4 ewes (with lambs) fed 10 kg fresh plant daily for 13 days (total 128 kg); lambs and ewes affected from day 6, control ewes (& their lambs) normal; signs persisted for 19 days after feeding ceased (Dodd & Henry 1922)
    - ewes each fed 2 kg fresh plant daily were affected after 30 days; their lambs, prevented from accessing the plant but suckled on the ewes twice daily, were unaffected (Seddon 1924)
- ***Malva parviflora*** (marsh or small-flowered mallow) [DM105]; Family Malvaceae
  - see notes on *Malva parviflora* under Skeletal muscle syndromes
  - experimental feeding of sheep from locations where the syndrome was unreported + forced exercise reproduced the syndrome (Dodd & Henry 1922):
    - 6 ewes (with lambs) fed 4 kg fresh plant/ewe/day for about 1 month; only lambs affected
    - 6 5-month-old sheep fed 33 kg fresh plant daily for 9 days; all affected
    - 4 ewes (with lambs) fed 12 kg fresh plant daily for 5 days: only lambs affected, controls (lucerne-fed) were normal
    - 2 ewes (with lambs) and 2 hoggets fed 2 kg wilted plant daily for 42 days (total 87kg); only lambs affected starting 24 days after feeding commenced

- 1 sheep fed 700 g seed daily for 6 days became affected
- 4 adult sheep fed 9.3 kg mature plant daily for 14 days (total 130 kg); 1 sheep affected on day 4, a second on day 11
- 4 adult sheep fed 8 kg flowering plant daily for 10 days (total 80 kg); 1 sheep affected from day 9, a second on day 10

- ***Echinopogon* spp.** [*E. caespitosus* and *E. ovatus*] (rough bearded grasses); Family Poaceae (Ev316)
  - cases in New England and at Yass, NSW (Seddon & Carne 1926, McBarron 1977)
  - clinically somewhat different from the above syndrome
  - experimental feeding of sheep reproduced the syndrome (Seddon & Carne 1926)

Diagnosis: syndrome + plant access + pathology?

Therapy:
- no specific therapy
- affected animals may recover completely after a period of rest

Prevention & control: prevent excessive intake of the plants

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EYE

Eye – Family Asteraceae

Helichrysum argyrosphaerum

Neurotoxin unknown

Sheep, cattle. Southern Africa (Namibia, north-western Cape Province)

Syndrome of cerebral oedema, optic nerve degeneration, retinal degeneration, skeletal muscle fibre necrosis. Cataracts seen in some cases

References:

Basson PA, Kellerman TS, Albl P, von Malitz LJF, Miller ES, Welman WG (1975) Blindness and encephalopathy caused by *Helichrysum argyrosphaerum* DC. (Compositae) in sheep and cattle. *Onderstepoort J. Vet. Res.* **42**:135-148.

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Eye – Family Myrtaceae

Rhodomyrtus macrocarpa *(finger cherry)*

Rhodomyrtus macrocarpa Benth. (finger cherry, native loquat, Cooktown loquat, wannakai, pool-boo-nong) (Family Myrtaceae) is a large shrub or small tree confined to the high rainfall areas of northern Queensland (Cook and North Kennedy pastoral districts) in rainforest as far south as the Herbert River. Its range extends to New Guinea. Seven other species of *Rhodomyrtus* occur in Queensland; 6 in the northern rainforests and one in the south-east. None of these are known to be toxic.

Fruits in large amounts are toxic to **humans**, causing sudden onset of **optic nerve atrophy** and **permanent blindness** within 24 hr of consumption. Mostly children but also adults have been affected. Deaths have been reported (D'Ombrain & Lucas 1944). Twenty seven soldiers were reported to suffer blindness after eating unspecified fruits in New Guinea (*Courier Mail*, Brisbane, 27 Dec 1945, p.2). Banfield (1910) stated that Aboriginal people and some Europeans habitually ate the fruit without ill effect. Cases were reported frequently before 1915 when the Queensland government circulated schools with a pamphlet illustrating the plant and warning of its potential toxicity. The probable first medical report was in 1894 of cases from around Cairns and cites FM Bailey, Colonial Botanist, as observing specimens that he identified as heavily infected with the fungus *Gloeosporium periculosum* forming yellow pustules on the fruit surface (Flecker 1944).

The exact circumstances and stage of maturity of the most toxic fruit are uncertain, except that toxicity follows consumption of a large quantity of fruit in a short time. Opinions are that

- only immature fruits (green or clear bright red) are toxic,
- only over-ripe fruits are toxic, or
- fruits are probably toxic only when mature or over-ripe and infected by the fungus *Gloeosporium periculosum* (Bailey 1895, 1900, 1912; Jarvis 1969)

Flecker (1944) reported toxicity from consumption of over-ripe fruits (2 cases) and immature fruits (2 cases) with one case consuming fruits of unspecified maturity. The first case described by Flecker (1944) was in 1892 in a 4-year-old girl who had eaten fruit from the ground beneath a bush (mature, mouldy fruit?) while her two siblings ate from the bush and were unaffected. The second case was in an 8.5-year-old boy who had two large meals of over-ripe fruit picked from a tree before sudden onset of blindness. The third and fourth cases were in girls 8-years-old who ate immature fruit because older children had eaten the mature ones. John Tonge (1947 unpublished letter in Queensland Herbarium files cited by Everist 1981) reported that one affected child ate green fruits while four others ate ripe fruits and were unaffected.

Poisoning of domestic animals is rare as access to plants rarely occurs. Plants do not persist when rainforest is cleared for pasture. Two female **calves** tethered to a plant at Malanda in July 1921were poisoned by eating leaves resulting in posterior paralysis in both, death in one and blindness in the other (White 1921). A **goat** habitually fed fruit in 1892 by the patient in the first of Flecker's cases became blind in 6 weeks (Flecker 1944).

Webb (1948) reported paralysis of the hindquarters in 2 guinea gips dosed PO with juice from the leaves (no dosage data was given).

The nature of the neurotoxin responsible is **unknown**. Rhodomyrtoxin, a complex benzofuran (a tetrahydroxydimethyldi*iso*valeryldibenzofuran) (Trippett 1957), proposed as 1,1'-(1,3,7,9-tetrahydroxy-2,8-dimethyldibenzofuran-4,6-diyl)-3,3'-dimethyldibutan-1-one (Sargent *et al.* 1983), has been isolated at up to a 1% yield from the ether-soluble fraction of an acetone extract of dried immature fruits which was toxic to mice (Trippett 1957). The PO LD_{50} of rhodomyrtoxin was 12 mg/kg in mice, but no details of toxicity were reported (Trippett 1957). ψ-rhodomyrtoxin, a dibenzofuran associated with a 2-methylbutyryl and an isovaleryl residue (Anderson *et al.* 1969), having the structure 1,1'-(1,3,7,9-tetrahydroxy-2,8-dimethyldibenzofuran-4,6-diyl)-2,3'-dimethyldibutan-1-one (Sargent *et al.* 1983), was also isolated from immature fruit (Anderson *et al.* 1969). ψ-rhodomyrtoxin produced no toxic effects on a rat PO at 13 mg/kg or a cat SC at 6 mg/kg, but unspecified toxic effects were seen in mice dosed at *ca.* 30 mg/kg (route not specified, but probably PO) (Anderson *et al.* 1969). No temporary or permanent blindness was caused in these animals, nor was any effect observed from direct administration to the eyes of a rabbit (no dose stated) (Anderson *et al.* 1969).

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White CT (1921) Two plants poisonous to stock. *Qd. Agric. J.* **16**:194-196.

Eye – Family Xanthorrhaceae

Xanthorrhoea johnsonii *(northern forest grasstree) - cataracts*

See posterior ataxia syndrome (above)
Cattle. Queensland
Chronic consumption
References: Se149, DM60

Seawright AA (1955) Wallum disease in north Queensland. *Technical Service Notes, Division of Animal Industry, Queensland Department of Primary Industries,* July 1955, pp.13-15.

ALIMENTARY

Alimentary – Family Anacardiaceae

Schinus *spp. (*q.v.*)*

Alimentary – Family Fabaceae

Castanospermum australe *(Moreton Bay chestnut, black bean)*

Core data

Common sources: Castanospermum australe
Animals affected: cattle, rarely horses
Mode of action: toxin unknown
Poisoning circumstances:
- large intake of ripe seeds
- usually during drought in winter

Main effects: haemorrhagic gastroenteritis
Diagnosis: syndrome + access to ripe seeds in large amounts
Therapy:
- remove from source
- rumenotomy
- rehydrate, adsorbent, demulcent

Prevention: deny access to ripe seeds during drought

Chemical structure:

The toxin responsible for poisoning of domestic animals and humans is **unrecognised** despite the isolation of a number of toxins from the plant.

Saponins have been isolated from seeds (Brunnich 1901, Simes *et al.* 1959). The unripe seeds fed to cattle by McKenzie *et al.* (1988) contained considerably less saponins than in the ripe seeds fed (GS Sidu, 1987, personal communication). The latter were more toxic.

Castanospermine, a polyhydroxylated indolizidine alkaloid isolated from *C. australe* seeds (Hohenschutz *et al.* 1981), inhibits bovine α-glucosidase, but has not produced clinical disease in cattle (generalised glycogenosis type II, Pompe's disease) consistent with a deficiency of this enzyme (Reichmann *et al.* 1987, 1989). The experiments of McKenzie *et al.* (1988) revealed equal depression of α-glucosidase activity in circulating lymphocytes and skeletal muscle tissue in cattle fed ripe and unripe *C. australe* seeds, but gastroenteritis only in the former, thus

indicating that castanospermine was present equally in both types of seeds, but was not correlated with the induction of alimentary pathology.
Australine (= 7a-epialexine), a second polyhydroxy indolizidine alkaloid from *C. australe* seeds, also inhibits α-glucosidase.

Other toxins isolated from seeds include 6-epicastanospermine (Nash *et al.* 1990b), 7-deoxy-6-*epi*-castanospermine (Molyneux *et al.* 1990), 3,8-di-*epi*-alexine (Nash *et al.* 1988), 1,7a-di-*epi*-alexine (Nash *et al.* 1990a), 7,7a-di-*epi*-alexine (Nash *et al.* 1990a), fagomine (Chen *et al.* 1990).

Sources:

- ***Castanospermum australe*** (Moreton Bay chestnut, black bean) DM150
- coastal Q & northern NSW

Toxicity:

- **cattle**, rarely horses (Maiden 1904), humans (Maiden 1904), pig (Maiden 1904), dog (Kessels 1991)
- cattle: **ripe seeds** are toxic **in large doses**. Hindmarsh & Hart (1937) and McKenzie *et al.* (1988) experimentally reproduced toxicity in cattle with ripe seeds. Reassessment of the report of Hindmarsh & Hart (1937) suggests that the seeds they called unripe were in fact ripe (McKenzie *et al.* 1988).
- cattle: experimentally, a toxic dose is 16 g ripe seeds/kg for 13-16 days; ripe seeds weigh 8-45 g (mean 30 g) (McKenzie *et al.* 1988); estimated 3-9 g ripe seeds/kg for 9 days (Hindmarsh & Hart 1937)
- cattle: no effect was produced by feeding 210 kg leaf to a 230 kg steer over 72 days (WTK Hall 1952 unpublished data)
- a dog (adult) developed signs of severe abdominal pain and diarrhoea within 12 hr of being seen chewing mature seeds; responded to treatment (Kessels 1991)
- Steyn & van der Walt (1941) produced no adverse effects in sheep fed a total dose of 140 g unripe seeds/kg over 3 days or a total dose of 50 g leaf/kg over 7 days or 100 g leaf/kg over 14 days.

Humans and *Castanospermum australe* seeds

Toxicity: Less than one raw seed can cause clinical signs and roasting does not prevent toxicity (Maiden 1904 citing TL Bancroft). Four RAAF personnel on a training exercise in the Numinbah Valley of south-eastern Queensland ate 0.5, 0.5, 2 and 4 raw seeds respectively at 1 pm on 30 April 1968. [Queensland Herbarium voucher specimen BRI 077001] Two hours later they all developed sudden and violent vomiting, severe abdominal cramps and dizziness and two collapsed. All were hospitalised but continued nauseous and vomiting. Antiemetic medication was given and 2 patients received pethidine. At 8 pm all 4 simultaneously developed severe profuse diarrhoea, with a positive test for blood in one case. All recovered overnight. (SL Everist personal communication to AA Seawright, 7 May 1968, citing the report of the medical officer at RAAF Amberley)

Detoxication: Aboriginal Australians traditionally used the seeds as a carbohydrate source, first processing them by maceration, then steeping in water for 8-10 days, then sun drying, roasting on hot stones, pounding into a meal and finally mixing the meal with water and baking it as thin cakes (Maiden 1904 citing C.Moore). Processing of seeds by northern Queensland aboriginal people involved either roasting or steaming before macerating and leaching in running water (Horsfall 1987). Soaking thin slices of seed in running water for 2 days followed by boiling in 3 changes of water for 45 min was insufficient to detoxify them which required maceration in a food processor, washing in running water for 14 days and baking resulting in a harmless but tasteless product (Cribb & Cribb 1974). An investigation of detoxification methods resulted in no ill effects on the experimenter after cooking, grating and soaking in water for 18 days while changing the water daily and then dry roasting, or grating and soaking in water for 26 days while changing the water daily or pounding and placing in running water for 4 days, then dry roasting (McBride 1997).

Mode of action:

- an unknown toxin is responsible for alimentary irritation (neither saponins nor castanospermine nor australine appear to be likely candidates). The large dose-response differences between ruminants and humans may relate to microbial detoxication in the rumen.
- castanospermine inhibits mammalian α-glucosidase, β-glucosidase & β-glucocerebrosidase (Saul *et al.* 1983, 1985) and insect glucosidases (Scofield *et al.* 1995a,b) and is suggested as a plant defense chemicals directed against seed feeding insects (Fellows *et al.* 1986). It also has antiviral properties against influenza virus (Pan *et al.* 1983) and against human immunodeficiency virus (HIV) - the cause of acquired immunodeficiency syndrome (AIDS) (Tyms *et al.* 1987, Walker *et al.* 1987).

Conditions of poisoning:

- livestock: large intakes can occur during **droughts** in **winter** when seeds have fallen from the trees

Clinical signs:

Cattle, horses

- **severe diarrhoea with dark faeces**
- dehydration
- ± sudden death (seen more often in horses)
- frequent straining to urinate and scanty dark urine was reported in one cow (Symons 1911)

Pathology (McKenzie *et al.* 1988):

- cytoplasmic vacuolation of 1-5% of lymphocytes in peripheral blood; cytoplasmic PAS-positive granules present in 78-92% of lymphocytes (15-20% positive in unexposed animals)
- **haemorrhagic gastroenteritis**
- ± focal myocardial necrosis
- ± nephrosis (vacuolation of renal cortical tubular epithelium, hyaline casts, dilation of collecting ducts)

Diagnosis: syndrome + access to ripe seeds in large amounts

Therapy:

- remove from source
- rumenotomy to remove bulk of *C. australe* seeds
- rehydrate + adsorbents + demulcents

Prevention & control: deny access to ripe seeds during drought

References:

Se28

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trihydroxypyrrolizidine [(2S,3R,4R,5S,6R)-2-hydroxymethyl-1-azabicyclo[3.3.0]octan-3,4,6-triol]. *Tetrahedron* **44**:5959-5964.

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Crotalaria aridicola & Crotalaria medicaginea (*Oesophageal ulceration of horses)*

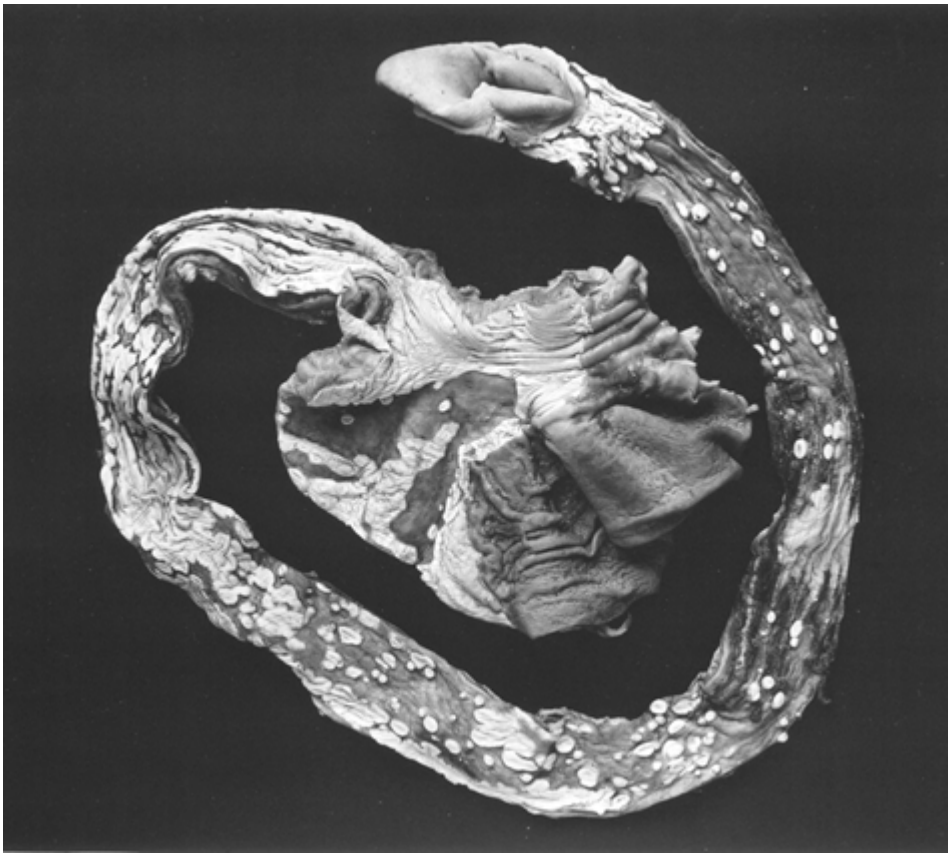
Core data

Common source: Crotalaria aridicola (North Qld), *Crotalaria medicaginea* (Capricornia)
Animals affected: horse
Mode of action: undetermined, toxin uncharacterised
Poisoning circumstances: exposed to abundant plant under poor pasture conditions
Main effects: oesophageal ulceration → regurgitation
Diagnosis: syndrome + plant access
Therapy: remove food from oesophagus, pass stomach tube (± under anaesthesia)
Prevention: deny access

Syndrome name:
Chillagoe horse disease
Chemical structure:
toxin unknown (pyrrolizidine alkaloids possible)
Sources:
syndrome associated with
Crotalaria aridicola (Chillagoe horse poison) [DM73] in northern Queensland
Crotalaria medicaginea (trefoil rattlepod) [DM74] in central Queensland. This species has a wide distribution across tropical Australia, but the syndrome is recognised only in the Capricornia region of Queensland

Mature flowering and fruiting *Crotalaria medicaginea* in natural habitat. [RAM Photos]

Toxicity:
horses
Mode of action: undefined
Conditions of poisoning:
exposure to large amounts of plant under poor pasture conditions predisposes
first exposure to onset about 4-5 weeks
Clinical signs:
lick the lips, drool saliva & grind teeth
drink water in small amounts
then vigorous swallowing attempts → regurgitation of ingest through nostrils
coughing
± swelling of neck above site of oesophageal obstruction
weight loss
dehydration → death
Pathology:
extensive **ulceration of mucosa of oesophagus** and cardia of the stomach



Horse oesophagus and stomach dissected to display their mucosal surfaces. Experimental intoxication with *Crotalaria medicaginea* from the Capricornia region, Queensland. The surviving normal areas of oesophageal mucosa are the raised pale sections among the darker ulcerated areas. Only the stratified squamous epithelium of the oesophagus and oesophageal part of the stomach is affected. [Original photograph 1953 by OH Brooks]

Diagnosis: plant access + syndrome + pathology
Therapy:
attempt to remove accumulated food and to pass a stomach tube (under anaesthesia if required) through which to feed and water the animal until the lesions heal
Prevention & control:
prevent access to plants (fence off if practical)
References: Se36

Alimentary – Family Phytolaccaceae

Phytolacca *spp.*

Phytolacca in Family Phytolaccaceae contains 25 species, mostly in tropical America, with 3 species naturalised in Australia (Hewson 1984).

Australia
Phytolacca americana (pokeweed, pigeon berry): weedy herb of disturbed soils; alimentary irritation of cattle in North America (Kingsbury & Hillman 1965, Barnett 1975)
Phytolacca dioica (packalacca, bella sombra): tree native to South America; cultivated for shade and fodder in south-eastern Australia, NSW, Vic; Ingestion of fruit & leaves by cattle and fowls in Australia associated with enteritis (Storie *et al.* 1992)
Phytolacca octandra (inkweed): weedy herb of disturbed soil; suspected of poisonings in Australia (Everist 1981)

Africa

Phytolacca dodecandra: alimentary irritation and lung damage in cattle in Africa (Mugera 1970)

References:

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Everist SL (1981) *Poisonous Plants of Australia.* 2nd ed. Angus & Robertson, Sydney. p. 577.
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Kingsbury JM, Hillman RB (1965) *Cornell Vet.* **55**:534
Mugera GM (1970) *Bull. Epizoot. Dis. Afr.* **18**:41
Storie GJ, McKenzie RA, Fraser IR (1992) Suspected packalacca (*Phytolacca dioica*) poisoning of cattle and chickens. *Aust. Vet. J.* **69**:21-22.

Alimentary – Family Poaceae

Avena sativa *(oats) – "red-tipped" or "rusty" oats crops*

Syndrome names:
oats sickness
green oats poisoning

Sources: stressed *Avena sativa* (oats) used as fodder crops

Toxicity:
- unknown toxin
  - possible phytoalexin produced in response to stress
  - possible hordenine (alkaloid) responsible for diarrhoea; dosing sheep orally with hordenine produces diarrhoea within 6 hr (CA Bourke, unpublished data cited by Anon 1997)
  - possible mycotoxicosis (Schneider *et al.* 1985)
- cattle, sheep susceptible
- cattle morbidity commonly 10-25%, but may be up to 90%
- mortality is rare

Mode of action:
- suspected depression of ruminal flora function; ruminal protozoal populations are severely depleted (Anon 1997)

Conditions of Poisoning:
lactating cows principally affected
ruminants grazed on forage oats crops under stress
crops had been previously grazed at least once (Anon 1997)
dry and frosty conditions predispose to incidents (Anon 1997)
grazing periods exceeding 2 hr predispose to incidents (Anon 1997)
identified stressors
- "red-tipped" oats – suspected nutritional deficiencies of N, P, Mg or Zn
- infection with barley yellow dwarf virus
- oats infected with the fungus *Drechslera campanulata* in southern Africa (Schneider *et al.* 1985)

crops implicated have had consistently small plant crude protein (4.4-8.1%) and acid detergent fibre (20.5-28.3%) concentrations (Anon 1997).

Clinical signs:

Cattle (Q, WA, NSW; several cases)
- sudden onset; recovery in 2-5 days
- reluctance to graze
- become difficult to handle; react suddenly & violently to noise
- depression
- **severe diarrhoea**; faeces may contain mucus and undigested feed (Anon 1997)
- loss of milk production / agalactia
- ± sudden collapse of hindquarters, recumbency
- ± death

Sheep (NSW; 1 case only)
- onset 3 hr after first exposure; recovery in 3-5 days
- depression, loss of appetite
- diarrhoea

Pathology:

Cattle

- ↓ plasma concentrations of P
- lesser decreases in plasma Ca, Mg
- ruminal acidosis: rumen pH of 5 or less and urine pH of less than 7; serum D-lactate concentrations increased (9.18-15.15 mmol/L in 3 cows; normal <0.4) (Anon 1997)
- no necropsy findings reported

Diagnosis:
- access to "red-tipped" oats + syndrome
- rule out nitrate-nitrite toxicity, hypomagnesaemia

Therapy:
- immediate removal of dairy herds from crop
- IV Ca & Mg suggested

Prevention/control ("red-tipped" oats): Thompson & Bywater (1987)
- apply increased rate of N fertilizer pre-planting (at least 50 units N)
- apply additional N at planting + P, S, K where deficiencies of these elements are identified
- or top-dress with N after first grazing
- or introduce a legume rotation (lab lab, chickpeas) with the oats crop
- or include a legume planting (medics, vetch) with the oats
- or plant alternative fodder crops (triticale, barley)

References:

Allen JG (1997) Oat sickness. Posting to VETTOX Internet Discussion Group

Anon. (1973) Toxicity in sheep grazing virus affected oats. *NSW Dept of Agriculture Veterinary Notes*

Anon. (1997) Two outbreaks of diarrhoea and agalactia in dairy cows grazing winter cereal crops in the Bega district. Unpublished report, Bega Rural Lands Protection Board. 8pp.

Grundon NJ (1983) Red-tipped oats: eastern Darling Downs, 1983. Unpublished report, Queensland Wheat Research Institute, DPI Queensland

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Thompson PJM, Bywater JW (1987) Red-tipped oats. *Qd. Agric. J.* May-June pp.165-170

☑ Pennisetum clandestinum *(kikuyu grass)*

Core data

Syndrome names: kikuyu poisoning of cattle
Common sources: Pennisetum clandestinum
Animals affected: cattle
Mode of action: unknown toxin and pathogenesis
Poisoning circumstances: ± related to pasture stress (e.g. insects)
Main effects:
- dehydration
- rumenitis
- nephrosis

Diagnosis: syndrome + access
Therapy: no specific therapy [?rehydration, as for ruminal acidosis]
Prevention: no specific measures recognised

Syndrome names: `**kikuyu poisoning' of cattle**
Chemical structure:
- unknown toxin
  - trichothecene mycotoxins suspected, but evidence inconclusive
  - considering the insect damage observations, a phytoalexin (*q.v.*) may be involved

Sources:
- ***Pennisetum clandestinum*** (kikuyu grass); cultivated pasture grass in subtropical and temperate climates; originating from eastern Africa

Toxicity:
- cases in New Zealand, South Africa, Australia (WA, NSW, Q)

Mode of action: unknown

Conditions of poisoning:
- cases have followed **pasture "stress"**:
  - insect larval (`army worm') damage to the grass
  - active growth of the grass following fertilisation/rain/irrigation after a long dry period
- significance of such factors is not understood

Clinical signs:
- depression, anorexia
- reluctance to move
- excessive salivation
- **sham drinking** (animals stand with head over water source, but do not drink)
- **dehydration** (haemoconcentration)
- **ruminal distension**
- tachypnoea
- constipation
- ataxia (high goose-stepping gait)
- sternal recumbency
- **high case fatality rate**

Pathology:
- dehydration (↑ PCV)
- ↑ serum urea & creatinine, ↓ serum Cl
- ruminal contents fluid (pH may be acid)
- ruminal and abomasal distension & hyperaemia
- histological lesions
  - **microvesication of forestomach mucosa** with neutrophil infiltration
  - **renal tubular necrosis**

Diagnosis: syndrome + access

Therapy:
- no specific therapy
- rehydration
- consider using therapy for ruminal acidosis

Prevention & control: no effective measures recognised

References:

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Gabbedy BJ, Gwynn R, Hopkinson WI, Kay BE, Wood PM (1974) Kikuyu poisoning of cattle in Western Australia. *Aust. Vet. J.* **50**:369-370.

ALIMENTARY – PHYTOBEZOARS

Alimentary – Phytobezoars – Family Ebenaceae

Diospyros virginiana *(persimmon) - phytobezoars (plant fibre balls): gastric or intestinal impaction/obstruction*

Horse; over-eating of fruit

Tannins are thought responsible for producing a coagulum in the stomach that entraps plant material (including persimmon seeds) and forms the basis of a phytobezoar (Cummings *et al.* 1997)

Clinical syndrome can include: Gastric impaction / duodenal obstruction, gastric ulceration with perforation, phytobezoars causing blockages in small intestine, intermittent refractory colic, weight loss

Persimmon consumption is thought to be the most common worldwide cause of phytobezoar formation in humans (Izumi *et al.* 1933, Dolan & Thompson 1979, Chisholm *et al.* 1992)

Diagnosis is facilitated by endoscopic examination of the stomach (Kellam *et al.* 2000)

Treatment with analgesics and laxatives is generally unsuccessful; gastrotomy may be indicated to remove the phytobezoar (Kellam 2000).

References:

Chisholm EM, Leong HT, Chung SC *et al.* (1992) Phytobezoar: an uncommon cause of small bowel obstruction. *Ann. Roy. Coll. Surg. Engl.* **74**:342-344.

Cummings CA, Copedge KJ, Confer AW (1997) Equine gastric impaction, ulceration, and perforation due to persimmon (*Diospyros virginiana*) ingestion. *J. Vet. Diagn. Invest.* **9**:311-313.

Dolan PA, Thompson BW (1979) Management of persimmon bezoars (diospyrobezoars). *South. Med. J.* **72**:1527-1528.
Honnas CM, Schumacher J (1985) Primary gastric impaction in a pony. *J. Am. Vet. Med. Assn.* **187**:501-502.
Izumi S, Isida K, Iwamoto M (1933) The mechanism of the formation of phytobezoars, with special reference to the persimmon ball. *Jap. J. Med. Sci. Biochem.* **2**:21-35.
Kellam LL, Johnson PJ, Kramer J, Keegan KG (2000) Gastric impaction and obstruction of the small intestine associated with persimmon phytobezoar in a horse. *J. Am. Vet. Med. Assoc.* **216**:1279-1281.
Morgan SE, Bellamy J (1994) Persimmon colic in a mare. *Eq. Pract.* **16**:8-10.
Wilson, RB, Scruggs DW (1992) Duodenal obstruction associated with persimmon fruit ingestion by two horses. *Eq. Vet. Sci.* **12**:26-27.

Alimentary – Phytobezoars – Family Iridaceae

Romulea rosea *var.* australis *(onion grass, Guildford grass) (*q.v.*)*

Alimentary – Phytobezoars – Family Ranunculaceae

Anemone patens *(pasque flower)*

Can dominate vegetation on the dry prairies of mid-western North America (Kingsbury 130)
Reported to form phytobezoars in intestinal tracts of sheep (Thomson RB, Sifton HB (1922) *A Guide to the Poisonous Plants and Weed Seeds of Canada and the Northern United States.* University of Toronto Press, Toronto. Cited by Kingsbury as Reference 1574)

MUSCLE

Muscle – Family Caesalpiniaceae

☑ Senna *spp. [*= Cassia *spp.]*

Core data

Common sources: Senna occidentalis
Animals affected: cattle (secondary – dogs)
Mode of action: undescribed; toxin uncharacterised
Poisoning circumstances: eating plants with seedpods
Main effects:
- skeletal muscle necrosis
- myoglobinuria
- diarrhoea

Diagnosis: pathology + access
Therapy: Se/vitamin E contraindicated
Prevention: deny access to plant with seedpods

Chemical structure:

Unidentified toxins cause the muscle damage.
Anthraquinone glycosides cause the diarrhoea.

Sources:

Family Caesalpiniaceae:

***Senna* spp.** - the generic name is taken from the Arabic sana for species which have leaves and pods with cathartic and laxative properties; about 62 species in Australia (Randell & Barlow 1998b). Toxicity appears to be confined to some of the introduced naturalised species.

Senna occidentalis (L.) Link [= *Cassia occidentalis* L.] (coffee senna, ant bush) [DM138] - probably native to the Americas, but now an aggressive pantropical weed (Randell & Barlow 1998b); toxicity recorded in North America and Australia (Rogers *et al.* 1979)

Senna obtusifolia (L.) H.S.Irwin & Barneby [= *Cassia obtusifolia* L.] (sicklepod, Java bean) - probably native to the Americas, but now of pantropical distribution (Randell & Barlow 1998b); naturalised in NT, SA and northern Qld; presumed toxicity case in horses in the Top End, NT (Andrew Mitchell, personal communication 2002)

Senna didymobotrya (Fresen.) H.S.Irwin & Barneby [= *Cassia didymobotrya* Fresen.] - native of northern Africa and widely cultivated in tropical areas around the world' sparsely naturalised in Qld and NSW (Randell & Barlow 1998b); suspected toxicity cases in goats (Law & McKenzie 1996) and cattle (Finster & McKenzie 2002)

Cassia spp. - only 3 species occur naturally in Australia - *C. fistula* L., *C. queenslandica* C.T.White and *C. brewsteri* (F.Muell.) Benth. (Randell & Barlow 1998a), none being recorded as toxic to domestic animals or humans, but they may have toxic potential

Cassia roemeriana (twin-leaf senna, two-leaved senna) [USA] (Rowe 1991)

Toxicity:

natural cases of poisoning in **cattle**, pigs (Colvin *et al.* 1986), horses

experimental intoxications in cattle, sheep, goats, rabbits and poultry (Haraguchi *et al.* 1998a,b)

Australia (Q), North America

seeds toxic; *S. occidentalis* >> *S. obtusifolia* (John Reagor, personal communication VETTOX 2000)

secondary poisoning of dogs that fed on meat from cattle affected by *C. occidentalis* toxicity (Q) (RM Hedlefs, personal communication 1987)

Mode of action: undescribed

Conditions of poisoning:

consumption of plants bearing mature seedpods (*S. occidentalis*), or whole plants ± green seed pods (*S. obtusifolia*)

Clinical signs:

muscle weakness

stiff → stumbling gait

recumbency, unable to rise or remain standing when lifted

red urine (**myoglobinuria**)

± sudden death

diarrhoea

Pathology:

↑↑↑ serum creatine phosphokinase & AST concentrations

skeletal & cardiac muscle pallor

necrosis of muscle fibres

pancreatic acinar cell necrosis was seen consistently in affected pigs (Colvin *et al.* 1986)

Diagnosis:

pathology + access

differential diagnoses include haemolytic diseases (babesiosis, Cu poisoning)

Therapy:

Se/vitamin E dosing → exacerbation !

Prevention & control: deny access to mature plants

References:

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Barth AT, Kommers GD, Salles MS, Wouters F, Lombardo de Barros CS (1994) Coffee senna (*Senna occidentalis*) poisoning in cattle in Brazil. *Vet. Human Toxicol.* **36**:541-545.

Colvin BM, Harrison LR, Sangster LT, Gosser HS (1986) *Cassia occidentalis* toxicosis in growing pigs. *J. Am. Vet. Med. Assoc.* **189**:423-426.

Faz EM *et al.* (1998) *Cassia occidentalis* toxicosis in heifers. *Vet. Human Toxicol.* **40**:307.

Finster DM, McKenzie RA (2002) Suspected *Senna didymobotrya* poisoning in cattle. Unpublished report, DPI Natural Toxins Database 5 August 2002.

Flory W, Spainhour CB, Colvin B, Herbert CD (1992) The toxicological investigation of a feed grain contaminated with seeds of the plant species *Cassia*. *J. Vet. Diagn. Invest.* **4**:65-69.

Haraguchi M, Gorniak SL, Calore EE, Cavaliere MJ, Raspantini PC, Calore NMP, Dagli MLZ (1998a) Muscle degeneration in chicks caused by *Senna occidentalis* seeds. *Avian Pathol.* **27**:346-351.

Haraguchi M, Calore EE, Dagli MLZ, Cavaliere MJ, Calore NMP, Weg R, Raspantini PC, Gorniak SL (1998b) Muscle atrophy induced in broiler chicks by parts of *Senna occidentalis* seeds. *Vet. Res. Commun.* **22**:265-271.

Kirby CJ, Rogers GM (1999) Sicklepod toxicity. *Compendium of Continuing Education for the Practicing Veterinarian (Food Animal Supplement)* February, S66-S79.

Law JA, McKenzie RA (1996) Suspected *Senna didymobotrya* poisoning in goats. Unpublished report, DPI Poisonous Plants Files.

Martin BW, Terry MK, Bridges CH, Bailey EM (1981) Toxicity of *Cassia occidentalis* in the horse. *Vet. Human Toxicol.* **23**:416-417.
McCormack JE, Neisler WE (1980) *Cassia obtusifolia* (sicklepod) toxicity in a dairy herd. *Vet. Med. / Small Anim. Practit.* **75**:1849-1851.
Nicholson SS, Thornton JT, Rimes AJ (1977) Toxic myopathy in dairy cattle caused by *Cassia obtusifolia* in greenchop. *Bovine Practitioner* **12**:120.
Parsons WT, Cuthbertson EG (2001) *Noxious Weeds of Australia.* 2nd edition. CSIRO Publishing, Melbourne, pp. 457-461.
Randell BR, Barlow BA (1998a) *Cassia. Flora of Aust.* **12**:75-80.
Randell BR, Barlow BA (1998b) *Senna. Flora of Aust.* **12**:89-138.
Rogers RJ, Gibson JA, Reichmann KG (1979) The toxicity of *Cassia occidentalis* for cattle. *Aust. Vet. J.* **55**:408-412.
Rowe LD (1991) *Cassia*-induced myopathy. Chapter 16 in Keeler RF, Tu AT (eds) *Handbook of Natural Toxins. Vol.6. Toxicology of Plant and Fungal Compounds.* Marcel Dekker, Inc., New York. pp. 335-351.

Muscle – Family Malvaceae

Malva parviflora *(mallow, marsh mallow, small-flowered mallow)*

Myotoxin unknown
Toxicity:
- sheep
- toxin unknown
- reproduced experimentally (Dodd & Henry 1922)

Conditions of poisoning:
- hungry sheep + large quantity of plant
- usually young sheep up to 2 yr-old, including lambs on ewes
- ewes and lambs affected (10 ewes + 6 lambs dead from a flock of 420 ewes) after grazing a pasture comprising 90% *Malva parviflora* and being driven (Main 2001)

Clinical signs:
- lameness, lag behind flock if mustered
- "staggers", incoordination
- muscle tremors
- ± recumbency
- morbidity high (20-50% of flock), death rare

Pathology:
- ↑ CPK , AST, urea, creatinine
- skeletal muscle oedema, degeneration ± necrosis (particularly in large muscle groups of hindlimbs)
- multifocal myocardial necrosis (Main 2001)
- ± myogobinuric nephrosis
- Vitamin E & Se concentrations normal
- no CNS lesions detected

Diagnosis: access + pathology
Therapy: Nil
Prevention & control: deny access to large amounts of the plant by hungry sheep
References:

Anon. (1988) Staggers in sheep associated with marshmallow [Cases from Coonamble, Wilcannia contributed by RVL, Orange] *The Veterinary Pathology Report* (ASVP Newsletter) No.19 p.12
Dodd S, Henry M (1922) Staggers or shivers in live stock. *J. Comp. Path. Therapeut.* **35**:41-61. [sheep natural & experimental cases with *Malva parviflora* and *Lamium amplexicaule*]
Glastonbury J (1999) *Malva parviflora,* marshmallow poisoning in sheep. *The Veterinary Pathology Report* (ASVP Newsletter) No.52 p.18
Main C (2001) Marshmallow toxicity in merino ewes and lambs. *Vet. Path. Report (ASVP Newsletter)* No.61 p.36.

Muscle – Family Sapindaceae

Atalaya hemiglauca *(white wood)* (q.v.)

THYROID

Thyroid – Family Poaceae

Pennisetum typhoides *(pearl millet seed) – goats; Africa*

Goats fed 1 g seed/kg/day (Gadir & Adam 1999); fermented or fermented processed millet did not produce goitre (Gadir & Adam 2000) but did cause illness
Humans in western Sudan eating pearl millet (local name dukhn) develop goitre; those consuming fermented millet (assida) do not (Gadir & Adam 2000).
References:

Abdel Gadir WS, Adam SEI (1999) Development of goitre and enterohepatonephropathy in Nubian goats fed with pearl millet (*Pennisetum typhoides*). *Vet. J.* **157**:178-185.
Abdel Gadir WS, Adam SEI (2000) Effects of pearl millet (*Pennisetum typhoides*), and fermented and processed fermented millet on Nubian goats. *Vet. Human Toxicol.* **42**:133-136.

BONE

Bone – Family Apiaceae

☑ Trachymene *spp. (wild parsnips)*

Core data

Syndrome names: bent-leg of lambs
Common sources: Trachymene spp. seedheads
Animals affected: sheep
Mode of action:

- undescribed; toxin uncharacterised
- toxin transfer through placenta & milk

Poisoning circumstances: pregnant or lactating ewes grazing *Trachymene* seedheads
Main effects:

- irregular epiphyseal plates
- deviation of carpal joints

Diagnosis: syndrome + access
Therapy: nil; some lambs recover
Prevention: deny pregnant or lactating ewes access to seedheads

Syndrome names:

Three distinct syndromes have been associated with sheep grazing *Trachymene* spp.

- **bent-leg of lambs**, bandy-leg of lambs
- sudden death in young sheep
- diarrhoea in young sheep

Chemical structure:

No toxin has been identified as responsible for any of the 3 syndromes.

Sources:

Family Apiaceae (Umbelliferae) (carrot family)

Trachymene spp. - about 30 species in Australia, those that have been associated with disease in livestock are

Trachymene ochracea (wild parsnip) [DM96]
Trachymene glaucifolia (wild parsnip)
Trachymene cyanantha (wild or blue parsnip) [DM97]

Toxicity:

- **sheep**
- south-western Queensland & north-eastern New South Wales

Mode of action:

- unknown toxin *in utero* or in milk → irregular retardation of growth of cartilaginous epiphyseal plates → altered angle of long bone articular surfaces
- foetal damage probably occurs after gestation day 35

Conditions of poisoning:

Bent-leg of lambs

pregnant or post-parturient ewes grazing **mature inflorescences with seeds**

ewes grazed on spring pastures spelled during winter → deformed lambs

ewes grazed on the same pastures over winter → normal lambs

deformed lambs most commonly born in spring after late summer or autumn rains

Sudden death

young sheep grazing green wild parsnips if stressed by forced exercise

Diarrhoea

young sheep grazing green plants

Clinical signs:

Bent leg of lambs

up to 25% of lambs deformed

lateral and medial deviation of carpal joints

? occasional → low lambing percentages (foetal resorption?) (Clark 1977)

posterior peromelia (deformity of the limbs) - reduction or absence of bones distal to metatarsals (Clark 1977)

Pathology:

Bent leg of lambs

irregularity of epiphyseal plates

Sudden death, Diarrhoea

No pathology has been described.

Diagnosis: syndrome + access

Therapy:

nil

a proportion of affected lambs fully recover

Prevention & control:

Bent leg of lambs

avoid pregnant or suckling ewe access to flowering and seeding plant

References:

Se133

Clark L, Carlisle CH, Beasley PS (1975) Observations on the pathology of bent leg of lambs in south-western Queensland. *Aust. Vet. J.* **51**:4-10.

Clark L (1977) *Trachymene* spp. and infertility in sheep. *Aust. Vet. J.* **53**:249-250.

SKIN AND APPENDAGES

Skin & Appendages – Family Anacardiaceae

Schinus *spp.*

Schinus areira [= *Schinus molle*] (pepperina tree): Some deaths are recorded in poultry after ingestion of the fruits and liver damage in fowls forced to eat the leaves for 2-3 weeks (McBarron 1977, p.180) and deaths in pigs which ate fruits and leaves (DPI Poisonous Plant Database record 1960). There is a comment on one of the files that fruits are toxic to calves, but there is no supporting evidence given. There are records of toxicity in children that have eaten fruits - leading to vomiting and diarrhoea (Watt & Breyer-Brandwijk 1962, p.51)

Schinus terebinthifolia (broad-leaved pepper tree, Brazilian pepper tree): Fruits are recorded as toxic to children (same reference as above) and the plant is recorded as producing contact dermatitis in humans (McBarron 1977, p.127; Mitchell & Rook 1979, p.88). The DPI Poisonous Plants Database has a record of a dog thought to have developed contact dermatitis from the plant.

Skin & Appendages – Family Apocynaceae

Parsonsia *spp. (*q.v.*)*

Skin & Appendages – Family Cupressaceae

Callitris *spp. (cypress pines)*

Sawdust from *Callitris intratropica* used as bedding for pigs near Cooktown, northern Queensland, during the wet season was associated with severe contact dermatitis affecting the feet and lower limbs of a sow and her litter, the udder of the sow and the mouths, prepuces and scrotums of the piglets suckling her. The lesions required differentiation from viral-induced vesicular diseases (J. Shield, unpublished data 1999).

The timber and sawdust from *Callitris columellaris* F. Muell. (white cypress pine) is reported by Bolza (1976) to cause "dermatitis, swelling of the eyelids, asthma, irritation of the nose and throat, nasal cancer and furunculosis" in humans with occupational exposure, but no references to source data are provided. Mitchell & Rook (1979) cite Cleland (1925) as reporting that the wood can produce dermatitis when cut in flower, but the original reference contains no data on this plant.

References:

Bolza E (1976) *Timber and Health.* CSIRO Division of Building Research. p. 18.

Cleland JB (1925) Plants, including fungi, poisonous or otherwise injurious to man in Australia. *Med. J. Aust.* **2**:443-451.

Mitchell J, Rook A (1979) *Botanical Dermatology. Plants and Plant Products Injurious to the Skin.* Greengrass, Vancouver. p.240.

Skin & Appendages – Family Fabaceae

☑Vicia *spp. (vetch toxicity)*

Core data

Common sources:

- *Vicia villosa* (woolly-pod vetch)
- *Vicia benghalensis* (Popany vetch)

Animals affected: cattle (mostly Angus + Friesian + relatives), rarely horses
Mode of action: suspected immune-mediated
Poisoning circumstances: grazing dense sward of mature plants
Main effects:

- dermatitis
- ill-thrift
- eosinophilic granulomas

Diagnosis: pathology + access
Therapy: nil
Prevention: nil

Syndrome names: **vetch toxicity**
Chemical structure:

- **toxin unknown**, but lectins (peptides) suspected on the basis of the type of lesion and the possibility that neoantigens (*q.v.*) may be involved

Sources:

- pasture legumes
- Family Fabaceae

only 2 species of *Vicia* have been associated with this syndrome, namely

- ***Vicia villosa*** ssp. *dasycarpa* (woolly-pod vetch)
- *V. benghalensis* (Popany vetch)

Toxicity:

- **cattle** (overwhelmingly Aberdeen Angus, Friesian and related breeds) and (rarely) horses
- disease reported from North & South America, South Africa & Australia (WA, NSW, Q)

- reproduced experimentally in USA.
- NSW cases: mean morbidity 7%, case fatality 69%.

Mode of action:

hypothesis of cause: hypersensitivity reaction to antigen in or associated with plants which is widely distributed in the body and persists → granulomatous response

Conditions of poisoning:

- intensive grazing of dense swards of mature plants

Clinical signs:

- **dermatitis** (unrelated to pigmentation) → patchy alopecia, pruritis
- **ill-thrift** - weight loss, ↓ milk production
- ± diarrhoea
- ± ocular discharge
- ± pyrexia
- ± dyspnoea
- ± melena, haematuria

Pathology:

- all lesions **eosinophilic granulomas**, often associated with blood vessels
- dermatitis (head, neck, shoulders, tail base, udder, perineum, ± trunk, ± limbs)
- pale foci in kidneys, heart
- enlarged adrenal glands & lymph nodes
- ± jaundice

Diagnosis:

- syndrome (pathology) + access
- differential diagnosis: photosensitisation, dermatomycosis, dermatophilosis
- similar syndrome seen in cattle fed citrus pulp (see below)

Therapy: nil

Prevention & control: no specific recommendations

References:

Se144; Cheeke 294

Harper PAW, Cook RW, Gill PA, Fraser GC, Badcoe LM, Power JM (1993) Vetch toxicosis in cattle grazing *Vicia villosa* ssp. *dasycarpa* and *V. benghalensis*. *Aust. Vet. J.* **70**:140-144.

Peet RL, Gardner JJ (1986) *Aust. Vet. J.* **63**:381

Skin & Appendages – Family Juglandaceae

Juglans nigra *(black walnut) – laminitis, horses*

Juglans nigra (black walnut, American walnut) is native hardwood tree of central and eastern North America.

Wood shavings of black walnut have been used as bedding for horses in North America.

Juglone (5-hydroxy-1,4-naphthoquinone) (Westfall *et al.* 1980) has been suspected as the toxin in black walnut responsible for laminitis in horses that are bedded on and eat wood shavings, but juglone is present in the fruit, bark and leaves, not the heartwood from which shavings originate (Minnick *et al.* 1987, True *et al.* 1978). Topical application of juglone to equine feet caused no signs (True & Lowe 1980). Large doses (up to 1g) of juglone PO caused mild signs of laminitis in 2 of 8 horses, but not to the degree expected from natural cases (True & Lowe 1980). In contrast. a juglone-free aqueous extract of black walnut timber PO caused laminitis consistent with that seen in natural cases in 8 of 10 experimental horses (Minnick *et al.* 1987).

Conditions of poisoning: Use of *fresh* black walnut wood shavings as bedding for horses. (Peterson 1984)

Clinical signs & pathology (Galey *et al.* 1991; Uhlinger 1989):

time of onset about 8-12 hrs after ingestion
mild sedation
anorexia
acute laminitis (clinically: lameness, foot pain detected by hoof testers, increased intensity of digital pulse, palpable warmth of feet; pathologically: congestion of dorsal laminae at necropsy, necrosis of laminae histologically in severe cases)
oedema of lower limbs
± abdominal pain (colic)
± respiratory distress

transient neutropaenia (4 hr after dosing) rebounding to neutrophilia with a left shift by 8-12 hr

Therapy:

remove bedding
phenylbutazone

Prevention:

aerating (forking over) black walnut wood shavings rapidly reduces toxicity (Peterson 1984).
sun-drying black walnut wood shavings for several weeks before use as bedding may significantly reduce toxicity (FD Galey, personal communication VETTOX Discussion Group 30 May 1996).

References:

Eaton S, Allen D, Eades SC *et al.* (1995) Digital Starling forces and hemodynamics during early laminitis induced by an aqueous extract of black walnut (*Juglans nigra*) in horses. *Am. J. Vet. Res.* **56**:1338-1344.
Galey FD, Beasley VR, Schaeffer D, Davis LE (1990) Effect of an aqueous extract of black walnut (*Juglans nigra*) on isolated equine digital vessels. *Am. J. Vet. Res.* **51**:83-88.
Galey FD, Twardock AR, Goetz TE, Schaeffer DJ, Hall JO, Beasley VR (1990) Gamma scintigraphic analysis of the distribution of perfusion of blood in the equine foot during black walnut (*Juglans nigra*)-induced laminitis. *Am. J. Vet. Res.* **51**:688-695.
Galey FD, Whiteley HE, Goetz TE, Kuenstler AR, Davis CA, Beasley VR (1991) Black walnut (*Juglans nigra*) toxicosis: a model for equine laminitis. *J. Comp. Path.* **104**:313-326.
MacDaniels LH (1983) Perspective on the black walnut toxicity problem: apparent allergies to man and horse. *Cornell Vet.* **73**:204-207.
Minnick PD, Brown CM, Braselton WE, MeerdinkGL, Slanker MR (1987) The induction of equine laminitis with an aqueous extract of the heartwood of black walnut (*Juglans nigra*). *Vet. Human Toxicol.* **29**:230-233.
Peterson DE (1984) Equine laminitis associated with black walnut toxicity. *Minnesota Vet.* **24**:38-43.
Ralston SL, Rich VA (1983) Black walnut toxicosis in horses. *J. Am. Vet. Med. Assoc.* **183**:1095.
Thomsen MEMcC, Davis EG, Rush BR (2000) Black walnut induced laminitis. *Vet. Human Toxicol.* **42**:8-11.
True RG, Lowe JE, Heissen J, Bradley W (1978) Black walnut shavings as a cause of laminitis. *Proc. Am. Assoc. Equine Practitioners* **24**:511-515.
True RG, Lowe JE (1980) Induced juglone toxicosis in ponies and horses. *Am. J. Vet. Res.* **41**:944-945.
Uhlinger C (1989) Black walnut toxicosis in ten horses. *J. Am. Vet. Med. Assoc.* **195**:343-344.
Westfall BA, Russell RL, Anyong TK (1980) A depressant agent from walnut hulls. *Science* **134**:1617-1619.

REPRODUCTIVE – ABORTION

Reproductive – Abortion – Family Asteraceae

Gutierrezia *spp. (snakeweeds)*

Syndrome name: snakeweed poisoning

Sources: *Gutierrezia* spp. (= *Xanthocephalum*); Family Asteraceae; North America

- *Gutierrezia microcephala* (threadleaf snakeweed)
- *Gutierrezia sarothrae* (broom snakeweed)
- alternative common names: broomweed, perennial snakeweed, slinkweed, turpentine weed

Toxicity:

- cattle, sheep
- toxin(s) responsible is unclear; candidates include saponins, terpenes, flavonoids, resins
- toxicity variable in experiments: abortion resulted after cattle consumed >450 kg fresh foliage during 117 days or 9 kg during 7 days

Circumstances of poisoning:

- plants widespread in North America but generally unpalatable; toxicity reported in livestock on dry ranges in Texas, New Mexico, Arizona, California, Colorado, Idaho and northern Mexico (Kingsbury 1964)
- toxicity follows consumption by livestock under poor nutritional conditions or adverse weather conditions
- plants more toxic when on sandy rather than loamy soil and when rapidly growing (Dollahite & Anthony 1957)

Clinical signs & Pathology:

- abortion or birth of weak calves
- retained placenta
- hepatic damage with cholestasis
- nephrosis
- impaired reproductive function (not dependant on overt toxicity)

References:

Dollahite JW, Anthony WV (1957) Poisoning of cattle with *Gutierrezia microcephala*, a perennial broomweed. *J. Am. Vet. Med. Assoc.* **130**:525-530.

Kingsbury JM (1964) *Poisonous Plants of the United States and Canada.* Prentice-Hall Inc., Englewood Cliffs, New Jersey. p. 406-408.

Smith GS, Ross TT, Flores-Rodriguez GI, Oetting BC, Edrington TS (1991) Toxicology of snakeweeds, *Gutierrezia microcephala* and *G. sarothrae*. Chapter 23 in James LF, Evans JO, Ralphs MH, Child RD (eds.) *Noxious Range Weeds.* Westview Press, Boulder, Colorado. pp.236-246.

Iva angustifolia *(narrowleaf sumpweed)*

Iva angustifolia (narrowleaf sumpweed); Family Asteraceae; North America
Reported to cause abortions in cattle (Murphy *et al.* 1984)
References:

Murphy MJ *et al.* (1984) *Proc. Ann. Meeting, Amer. Assoc. Vet. Lab. Diagnosticians* **26**:161. [cited by Blood DC, Radostits OM, Blood DC (1989) *Veterinary Medicine.* 7th ed., Bailliere Tindall, London. p.1351]

Tanacetum vulgare *(tansy)*

Tanacetum vulgare [= *Chrysanthemum vulgare*] (tansy); Family Asteraceae
Outmoded medicinal herb used mainly as an anthelmintic
Recorded as abortifacient in women (Bown 1995)
Abortion in cattle has been reported in Pennsylvania (Gress 1935)
References:

Bown D (1995) *Encyclopedia of Herbs and their uses.* RD Press, Surrey Hills, Sydney (Dorling Kindersley, London) p.359-360.

Gress EM (1935) Poisonous plants of Pennsylvania. *Pennsylvania Dept. Agric. Bull.* **18**(5) [cited by Kingsbury 437]

Reproductive – Abortion – Family Brassicaceae

Berteroa incana *(hoary alyssum)*

Berteroa incana (hoary alyssum); Family Brassicaceae; North America (not recognised as naturalised in Australia)
Toxicity

- horses: abortions have been associated with syndrome of distal limb oedema (Ellison 1992), fever and laminitis are also seen with this syndrome (Geor *et al.* 1992)
- horses: abortions associated with additional signs of colic, moderate-profuse bloody diarrhoea, haemoglobinuria (Hovda & Rose 1993)
- toxin unknown; SMCO (*q.v.*) could be involved

Conditions of poisoning:

- contamination of fodder hay (*Medicago sativa*; alfalfa/lucerne)

References:

Ellison SP (1992) Possible toxicity caused by hoary alyssum (*Berteroa incana*). *Vet. Med.* **87**:472, 474-475.

Geor RJ, Becker RL, Kanara EW, Hovda LR, Sweeney WH, Winter TF, Rorick JK, Ruth GR, Hope E, Murphy MJ (1992) Toxicosis in horses after ingestion of hoary alyssum. *J. Am. Vet. Med. Assoc.* **201**:63-67.

Hovda LR, Rose ML (1993) Hoary alyssum (*Berteroa incana*) toxicity in a herd of broodmare horses. *Vet. Human Toxicol.* **35**:39-40.

Raphanus raphanistrum *(wild radish)*

Raphanus raphanistrum (wild radish, jointed charlock); Family Brassicaceae (Hewson 1982)
Toxin unknown, probably SMCO (*q.v.*)
Cattle grazing the plant in Queensland developed haemoglobinuria (Parkinson & Sutherland 1954, R.A. McKenzie, unpublished data)
Cattle forced to eat a large quantity of plant in Western Australia were intoxicated, some aborting; signs were loss of condition, decreased appetite, lethargy, mucus-coated droppings progressing to watery blood-stained diarrhoea, encrusted noses, brisket oedema; necropsy findings were jaundice, haemoglobinuria, liver damage including fatty change and fibrosis, splenomegaly (Craig 1955)
Reference:

Craig J (1955) Unpublished report, Western Australian Department of Agriculture. [cited by Gardner CA, Bennetts HW (1956) *The Toxic Plants of Western Australia.* Western Australian Newspapers Ltd., Perth. pp.32-34.]

Hewson HJ (1982) Brassicaceae. *Flora of Australia* **8**:231-357.

Parkinson B, Sutherland AK (1954) *Aust. Vet. J.* **30**:232.

Reproductive – Abortion – Family Fabaceae

Ateleia glazioviana *(timbo de Palmeira)*

Ateleia glazioviana (timbo de Palmeira); Family Fabaceae; South America
Associated with abortions in cattle in Rio Grande do Sul, Brazil; experiments with plant extracts in rats induced embryonic mortality (Marona *et al.* 1992, Langeloh *et al.* 1992)
References:

Langeloh A, Maidana-Leguizamon F, Dalsenter P (1992) [Abortive and infertility potential of Brazilian plants which occasionally contaminate pasture of cattle and other grazing animals of economical importance.] *Pesq. Vet. Bras.* **12**:11-18. [Abstract 2657 *Vet. Bull.* 1993]
Marona HRN, Langeloh A, Schenkel EP (1992) [Abortion caused by *Ateleia glazioviana* (Leg. Papillionideae) in rats.] *Pesq. Vet. Bras.* **12**:81-83. [Abstract 6744 *Vet. Bull.* 1993]

Trigonella foenum-graecum *(fenugreek)*

Trigonella foenum-graecum (fenugreek); Family Fabaceae
Toxin unknown
Abortions in ewes grazing lush fenugreek carrying numerous green seedpods in a barley crop on the Darling Downs (Q) in August 1999; strong smell of curry from the foetuses; infectious causes of abortion ruled out (Taylor JD & Gibson JA, unpublished data, DPI Queensland files, 1999).
Fenugreek seed has been used as an abortifacient and to induce/hasten childbirth in humans (Watt & Breyer-Brandwijk 1962; Bown 1995; Chevallier 1996).
References:

Bown D (1995) *Encyclopedia of Herbs and their uses.* RD Press, Surrey Hills, Sydney (Dorling Kindersley, London) p.364.
Chevallier A (1996) *The Encyclopedia of Medicinal Plants.* Dorling Kindersley, London. p.276.
Watt JM, Breyer-Brandwijk MG (1962) *Medicinal and Poisonous Plants of Southern and Eastern Africa.* 2nd edition, E & S Livingstone Ltd., Edinburgh. pp.666-667.

Reproductive - Abortion – Family Iridaceae

Romulea rosea *var.* australis *(onion grass, Guildford grass)* (q.v.)

Reproductive – Abortion – Family Lamiaceae

Mentha longifolia *(horse mint)*

Mentha longifolia (horse mint); Family Lamiaceae; Europe
Ingestion of large amounts associated with abortion of cows in Austria (Richter 1966)
References:

Richter HE (1966) [Damage caused by the etheric oils of *Mentha longifolia* (abortion in cows).] *Weiner Tierärztliche Monatsschrift* **53**:201-202. [cited by Cooper MR, Johnson AW (1998) *Poisonous Plants and Fungi in Britain. Animal and Human Poisoning.* 2nd ed., The Stationery Office, London. p.126.

Mentha satureioides *(native pennyroyal)*

Mentha satureioides (native pennyroyal, squeejit); Family Lamiaceae; Australia
Toxin unknown
Associated with late abortions of thin foals and small under-developed placentas in mares on the eastern Darling Downs (Hall WTK & Pascoe RR, unpublished data, DPI Queensland files, 1968; Gibson JA & McKenzie RA, unpublished data, DPI Queensland files, 1996)
Suspected of causing abortion in cattle in Queensland (White 1934, 1936)
References:

White CT (1934) *Annual Report of the Government Botanist, Queensland for 1933-34.*
White CT (1936) *Annual Report of the Government Botanist, Queensland for 1935-36.*

Salvia coccinea *(red salvia, Texas sage)*

Salvia coccinea (red salvia, Texas sage); Family Lamiaceae
Native to North America, naturalised in eastern coastal NSW and Qld
Toxin unknown
Reported associated with abortions in cattle in NSW (Hurst 1942), Qld (Everist 1981) and Hawaii (Arnold 1968)

Feeding of pregnant cows resulted in abortions in 2 separate experiments; infectious causes of abortion were not detected (Chester RD, unpublished report, Queensland Department of Agriculture and Stock, 1943; Beiers P, unpublished report, Queensland Department of Primary Industries, 1964)

References:

Arnold HL (1968) *Poisonous Plants of Hawaii.* Charles E. Tuttle Co., Rutland, Vermont. [cited in McBarron 1977]
Everist SL (1981) *Poisonous Plants of Australia.* Angus & Robertson, Sydney. p.383-385.
Hurst E (1942) *The Poison Plants of New South Wales.* NSW Poison Plants Committee, Sydney. p.345-346.

Reproductive – Abortion – Family Ranunculaceae

Ranunculus repens *(creeping buttercup)*

Ranunculus repens (creeping buttercup); Family Ranunculaceae; distribution includes Europe, South America, Australia

Abortions in cattle in Chile associated with consumption of *R. repens* constituting 15-20% of herbage in pasture; abortions over 3 years, peaking in November (Morales 1989)

Toxin unknown

References:

Morales H (1989) [Abortions in a dairy herd in the VIII region of Chile attributed to the consumption of creeping buttercup (*Ranunculus repens* L.).] *Archivos de Medicina Veterinaria* **21**:163-166. [Abstract 5077 *Vet. Bull.* 1990]

Reproductive – Abortion – Family Sterculiaceae

Mansonia altissima *(African redwood) – wood shavings*

Mansonia altissima (African redwood); Family Sterculiaceae

Abortion in pigs exposed to bedding containing wood shavings of this tree (Betrocchi & Dovadola 1967, Bertschinger & Lott-Stolz 1970)

Contact irritation causing ulcers on mouth and skin + anorexia, diarrhoea, prostration & death: calves (Vacirca *et al.* 1970), pigs, dogs (Venturoli & Quadri 1969)

References:

Hu249 citing:
Bertocchi D, Dovadola E (1967) *Nuova Vet.* **43**:459-462.
Bertschinger HU, Lott-Stolz G (1970) *Schweizer Arch. Tierheilk.* **112**:641-651.
Vacirca G, Agosti M, Crotti S (1970) *Atti. Soc. Ital. Sci. Vet.* **24**:461-463.
Venturoli M, Quadri E (1969) *Nuova Vet.* **45**:71-81.

Reproductive – Abortion – Family Verbenaceae

Verbena *spp.*

Sources: Family Verbenaceae; naturalised weeds in Australia
Verbena officinalis (common vervain)
Verbena bonariensis (purple top)

Toxicity:

Verbena officinalis contains a bitter crystalline glycoside, verbenalin (= cornin from *Cornus florida*), reported to produce uterine contraction (Watt & Breyer-Brandwijk 1962)

Verbena officinalis & *Verbena bonariensis* have been suspected of causing abortions in cattle in Queensland (Everist 1981)

References:

Everist SL (1981) *The Poisonous Plants of Australia.* 2nd edition, Angus & Robertson, Sydney. p.748-749.
Watt JM, Breyer-Brandwijk MG (1962) *The Medicinal and Poisonous Plants of Southern and Eastern Africa.* 2nd edition, Livingston, Edinburgh. p.1054.

Reproductive – Abortion – Family Apiaceae, Asteraceae, Fabaceae, Poaceae

Other plant toxins associated with abortion

Bicknell (1990) lists the following plants and fungi (additional to those listed above in these notes) as causing abortions in cattle in North America:

- *Gutierrezia microcephala* (broomweed) Asteraceae
- *Gutierrezia sarothrae* (broomweed) Asteraceae

- *Conium maculatum* (hemlock) Apiaceae
- *Solidago ciliosa* (goldenrod) Asteraceae
- *Sorghum almum* Poaceae
- *Trifolium subterraneum* Fabaceae
- *Xanthium strumarium* (cocklebur) Asteraceae

References:

Bicknell EJ (1990) Abortion caused by pine needles and other plants. Chapter 27 in Kirkbride CA (ed) *Laboratory Diagnosis of Livestock Abortion.* 3rd edition., Iowa State university Press, Ames. pp.165-169.

REPRODUCTIVE – INFERTILITY

Reproductive - Infertility – Family Iridaceae

Romulea rosea *var.* australis *(onion grass, Guildford grass)* (q.v.*)*

Reproductive – Infertility – Family Simmondsiaceae

Simmondsia chinensis *(jojoba) meal - poultry*

Simmondsia chinensis (jojoba [Note: in Spanish, j is pronounced as an h], goat nut, pig nut) is an evergreen dioecious shrub originating in the arid lands of North America. Seeds yield oil similar in quality to sperm whale oil. Plantations of the shrubs are drought tolerant when established. Seeds contain about 50% oil. Oil has potential as fuel, lubricant, cosmetic base *etc.*

Source: **meal** after extraction of oil

Toxicity:

- toxin(s) unknown, mode of action unknown
- poultry (broiler breeders)
- meal inclusion rates of 2.5 – 4.0% caused non-maturation of the oviducts in broiler breeders

Conditions of poisoning:

- meal included in poultry rations as an autonomous feed intake restriction agent to control broiler breeder body weight

Clinical signs: **complete absence of egg production**

Pathology: very small oviducts, ovulation normal

References:

Jackson DL, Jacobs SWL (1985) *Australian Agricultural Botany.* Sydney University Press, Sydney. p.185

Vermaut S, Onagbesan O, Bruggeman V, Verhoeven G, Berghman L, Flo G, Cokelaere M, Decuypere E (1998) Unidentified factors in jojoba meal prevent oviduct development in broiler breeder females. *J. Agric. Fd. Chem.* **46**:194-201.