PLANTS WITH UNKNOWN OR UNCHARACTERISED TOXINS

[Plants are listed by syndrome/organ system affected, then by plant family]

SUDDEN DEATH / HEART

Sudden death / Heart – Family Apocynaceae

Parsonsia *spp.*

Woody climbing vines in eastern Australia; Family Apocynaceae
Toxicity appears related to higher-than-normal intake and/or little alternative feed being available.
Toxin unknown; cardiac glycosides are suspected due to other sources of these compounds in Family Apocynaceae, but no direct evidence is known
Parsonsia eucalyptophylla (gargaloo, monkey rope, silk pod) is commonly eaten as drought fodder by sheep and cattle without ill effect; attempted toxicity feeding trials with cattle were negative (Everist 1981, 1985)
- has been associated with **sudden deaths** in sheep (2 incidents) and cattle (1 incident) in western New South Wales; attributed to cyanogenic glycosides (McBarron 1978)
- has been associated with deaths in bulls under drought conditions in November near Roma in southern Queensland; lung congestion and haemorrhage and interstitial oedema of the heart were seen [Unpublished records, DPI Poisonous Plants Database; Newman RD (1994) personal communication]

Parsonsia straminea (monkey rope, silk pod)
- has been associated with deaths in cattle in south-eastern Queensland on 2 occasions, both in July under poor pasture conditions; mild liver damage was noted in one case [Unpublished records, DPI Poisonous Plants Database, Cavanagh GM (1999) personal communication; Tanner, S (2000) personal communication]
- associated with ataxia and teeth grinding with myocardial degeneration and cerebral oedema in Eastern Grey kangaroos in an enclosure in south-eastern Queensland in January (J Hanger, ND Sullivan, RA McKenzie, unpublished data DPI Poisonous Plant Database 2001)
- caused 1 incident of severe **skin irritation** in humans (Hunter 1997)

References

Everist SL (1981) *Poisonous Plants of Australia.* revised edition, Angus & Robertson, Sydney. p.87
Everist SL (1985) *Use of Fodder trees and Shrubs.* revised edition, Queensland Department of Primary Industries, Brisbane. p.51.
Hunter JT (1997) Severe blistering caused by *Parsonsia straminea* (R.Br.) F.Muell. (Apocynaceae) at Girraween National Park. *Qd. Naturalist* **35** (1-3):28-29.
McBarron EJ (1978) *Poisonous Plants of Western New South Wales.* Department of Agriculture, New South Wales, Sydney. p.79.

Sudden death / Heart – Family Poaceae

Phalaris aquatica *poisoning – sudden death*

Sudden death syndrome recognised as distinct from staggers syndrome
Toxin probably N-methyl tyramine
See notes on *Phalaris* under alkaloids
References: Se99

Skerritt JH, Guihot SL, McDonald SE, Culvenor RA (2000) Development of immunoassays for tyramine and tryptamine toxins of *Phalaris aquatica* L. *J. Agric. Food Chem.* **48**:27-32.

Phalaris coerulescens *poisoning (horses)*

Sources:
- *Phalaris coerulescens* (blue canary grass)
- naturalised pasture grass dominant in restricted area of central Victoria; present in V, NSW, NT

Toxicity:

- only horses reported affected
- cases on one Victorian property with pasture dominated by *P. coerulescens* 1988-93, on an adjacent property in 1998 and on a South Australian property in 1998 (Colegate *et al.* 1999)
- toxin unknown; tetrahydro-β-carbolines (also in *P. aquatica*), oxindoles (coerulescine, horsfiline) & a furanobisindole (phalarine and its hydroxy- and methoxy-analogues) have been isolated (Colegate *et al.*1999)

Mode of action: unknown

Conditions of poisoning:

- pasture dominated by *P. coerulescens* (newly shooting)
- onset after at least 22 days grazing

Clinical signs:

- spontaneous wild galloping, collapse followed by death within 10 minutes

Pathology: no lesions detected

References:

Anon. (1998) Horse deaths on blue canary grass pasture. *Aust. Equine Vet.* **16**:100-101.

Colegate SM, Anderton N, Edgar J, Bourke CA, Oram RN (1999) Suspected blue canary grass (*Phalaris coerulescens*) poisoning of horses. *Aust. Vet. J.* **77**:537-538.

Sudden death / Heart – Family Sapindaceae

Atalaya hemiglauca *(whitewood)*

Syndrome names:

- whitewood poisoning (horses)
- Alexandria disease (cattle, NT) (Rose 1951)

Source:

Atalaya hemiglauca (F.Muell.) F.Muell. ex Benth. (whitewood) (Family Sapindaceae) is a small tree to 6 m tall growing inland in all Australian states except Victoria and Tasmania, but mostly in Qld, NT and the Kimberley region of WA, in dry open mixed forests, at the margin of brigalow scrub and in deserts, in various soil types (Reynolds 1985). There are 8 other species of *Atalaya* in Australia, with others in Africa, Indonesia and New Guinea (Reynolds 1985).

Toxicity:

The toxin(s) responsible is(are) **unknown**.

Horses are susceptible to poisoning, but **field cases are apparently rare**. It is rarely suspected of poisoning cattle (Rose 1951; Gardner & Bennetts 1956; RA McKenzie unpublished data), but is usually regarded as good fodder for livestock (Reynolds 1985). Field cases in horses were attributed to *A. hemiglauca* in the Kimberley region during the later 1990s (TF Jubb, personal communication). Putative cases in cattle in the records of QDPI do not have a consistent syndrome with nephrosis (1987 Richmond district) and hepatic fatty change (1991 Cloncurry district) recorded, but no myocardial lesion detected

Fruiting specimens are regarded as the most toxic, with **toxicity concentrated in the fruit**. Experimentally fruits/seeds have reproduced poisoning when fed to horses. CSIR experiments 1928-1932: One horse died in 3 days when fed approximately 0.4 g dried fruits/kg/day; 3 horses died in 28 days when fed 0.06-0.13 g/kg/day; 2 horses died in 56 days when fed 0.06 g/kg/day; one horse died in 179 days when fed 0.03 g/kg/day; one horse remained healthy when fed 0.03 g/kg/day for 65 days (Murnane 1953). Northern Territory Animal Industry Branch experiments 1951: one horse died in 9 days when fed approximately 0.9 g seeds/kg/day; 2 horses fed approximately 0.3 g fruit/kg/day for 3 and 6 days died respectively 9 and 10 days after feeding started; one horse fed 0.09 g fruit/kg/day for 174 days was unaffected except for slight transient jaundice; another fed 0.09 g fruit/kg/day developed signs including muscular weakness, eyelid oedema and myoglobinuria and was killed 176 days after feeding started (McConnell & Barnes 1956).

Mature leaves are regarded as non-toxic, but **young shoots may poison horses and cattle** (Gardner & Bennetts 1956). Dried mature leaves fed to 2 horses at approximately 0.2 g/kg/day and 1.0 g/kg/day respectively for 190 days caused no illness (Murnane 1953). Horses that browse even small amounts of young shoots are reported to become excited and gallop madly, then either die from misadventure or recover fully (Gardner & Bennetts 1956).

Murnane & Ewart (1928) blamed *A. hemiglauca* for Kimberley horse disease (*q.v.*), which is now known to be a form of pyrrolizidine alkaloidosis commonly caused by *Crotalaria crispata* (*q.v.*) or *Crotalaria retusa* (*q.v.*). Their feeding trials in the Kimberley region of WA were compromised by the use of local horses which may have been subclinically affected by pyrrolizidine alkaloidosis. Later experiments (Murnane 1953) used plant from The Kimberley region fed to horses from Victoria.

Conditions of poisoning:

Poisoning in the Kimberley and in NT occurs during September-November, suggesting involvement of young shoots and seeds (Rose 1951, Gardner & Bennetts 1956). Flowering is during September and early October with fruit present during October-November (Gardner & Bennetts 1956). Fruit is available under natural conditions for about 6 weeks each year, but are **reported as unpalatable to horses** (!) (McConnell & Barnes 1956).

Young shoots are reported to be very palatable to horses (Gardner & Bennetts 1956).

Clinical signs (horses); not all horses have all signs

restlessness (Murnane 1953)

sweating (Whittem 1968)

muscle weakness; shivering; swaying or staggering gait; leaning over the forelegs (Murnane 1953, McConnell & Barnes 1956)

apparent inability to swallow (Murnane 1953)

head pressing (Murnane 1953)

oedematous swelling of the head in some animals; eyelids and supraorbital fossae (Murnane 1953, McConnell & Barnes 1956); suggestive of heart failure correlated with pathological findings

colic (McConnell & Barnes 1956, Whittem 1968)

recumbency & death preceded by violent running movements (Murnane 1953)

jaundice (McConnell & Barnes 1956)

myoglobinuria (red or brown urine) (Peterson 1952, Murnane 1953, McConnell & Barnes 1956)

Pathology (horses) (Murnane 1953, Gardner & Bennetts 1956, McConnell & Barnes 1956)

subendocardial, myocardial and diaphragmatic haemorrhages (Murnane 1953, McConnell & Barnes 1956, Whittem 1968)

skeletal muscle pallor and petechiation (McConnell & Barnes 1956, Whittem 1968)

excess volume of pleural and peritoneal fluid in some animals (Murnane 1953, McConnell & Barnes 1956, Whittem 1968)

myoglobinuria (very dark brown urine in bladder) (Murnane 1953, McConnell & Barnes 1956)

focal **degeneration & necrosis of skeletal & cardiac muscle** (McConnell & Barnes 1956, Whittem 1968)

hepatocyte degeneration (Whittem 1968); periacinar hepatocyte necrosis (McConnell & Barnes 1956)

nephrosis (hyaline [myoglobin?] casts in renal tubules (Peterson 1952)

References:

Gardner CA, Bennetts HW (1956) *Toxic Plants of Western Australia.* Western Australian Newspapers Ltd., Perth. pp.133-135.

McConnell JD, Barnes JE (1956) The toxicity of the fruits of *Atalaya hemiglauca* ("whitewood") for horses. *Aust. Vet. J.* **32**:74-76.

Murnane D (1929) *J. Coun. Sci. Industr. Res. Aust.* **2**:110

Murnane D (1953) The toxicity of *Atalaya hemiglauca* ("whitewood") for horses. *Aust. Vet. J.* **29**:188-190.

Murnane D, Ewart AJ (1928) *Kimberley Horse Disease. (Walk-about Disease). Report of co-operative work carried out by the Department of Agriculture of Western Australia and the CSIR.* Commonwealth of Australia Council for Scientific and Industrial Research Bulletin No.36. 61pp.

Peterson JE (1952) WA Department of Agriculture unpublished records cited by Gardner & Bennetts (1956).

Reynolds ST (1985) Sapindaceae. 4. Atalaya. *Flora of Australia* **25**:12-18.

Rose AL (1951) Personal communication to Gardner & Bennetts (1956).

Whittem JM (1968) Experimental whitewood, *Atalaya hemiglauca*, poisoning in the horse. *Aust. Vet. J.* **44**:426.

LUNG

Lung – Family Asteraceae

☑ Ageratina *spp. (Crofton weed, mist flower)*

Core data

Common sources: Ageratina adenophora (Crofton weed)
Animals affected: horses
Mode of action: undescribed; toxin unknown
Poisoning circumstances: access to large amount of flowering plant
Main effects: lung fibrosis
Diagnosis: access + pathology
Therapy: nil
Prevention:

- deny access
- pasture improvement

Syndrome names:

Numinbah Horse Sickness
Tallebudgera Horse Disease
Crofton weed poisoning

Chemical structure:

The pneumotoxin responsible is **unknown**. Pyrrolizidine alkaloids are suspected in part because there are some similarities of the lesions to jaagsiekte, the *Crotalaria*-associated lung disease of horses in South Africa caused by dicrotaline (*q.v.*), and lung lesions seen in horses in northern Australia poisoned by fulvine from *Crotalaria crispata* (*q.v.*).

The hepatotoxin responsible for the experimental induction of very persistent necrotic hepatocyte lesions in mouse liver has been isolated and identified as 9-oxo-10,11-dehydroagerophorone (Oelrichs *et al.* 1995, 1998).

Sources:

Family Asteraceae

Ageratina adenophora *(Eupatorium adenophorum*) (Crofton weed) [DM94]

Ageratina riparia (Eupatorium riparium) (mist flower) toxic experimentally, but no field cases recorded.

Ageratina adenophora (Crofton weed) flowering plants (left) and leaves (right) [RAM Photos]

Toxicity:
- **horses** only affected
- south-eastern Queensland - south of Sydney along the coast/hinterland.

Mode of action: undescribed

Conditions of poisoning:
- access to large amounts of plant, readily eaten
- **flowering plant** most toxic
- inhalation of plant material (e.g. pollen) not needed for development of disease
- most cases first affected in summer after access to flowering plant in the previous spring (2 months minimum period after first access)

Clinical signs:
- **coughing** (exacerbated by exercise)
- **↓ exercise tolerance**
- rapid heaving respiration
- double expiratory effort
- rales (↑ after exercise)
- weight loss → emphasises pleuritic ridge
- ± cyanosis
- ± cardiac arrhythmia → sudden death

Pathology:
- Sudden death cases
  - hydrothorax, pulmonary oedema & emphysema
  - hydropericardium, dilation of heart
- `Classical' cases
  - **pulmonary fibrosis** → lungs remain inflated
  - lung cavities containing necrotic debris
  - ± lung septa distended with green oedema fluid (eosinophils)

Diagnosis: access + syndrome

Therapy:

no generally effective treatment
antibiotics & corticosteroids improve some cases

Prevention & control:

confine horses and hand feed if plant infestations heavy
slashing followed by herbicides when regrowth reaches 15-25 cm high, then seeding and fertilising to promote vigorous pasture competition
grazing/browsing with goats?
introduced biological control agents have been ineffective

References:

Se51
Oelrichs PB, Calanasan CA, MacLeod JK, Seawright AA, Ng JC (1995) Isolation of a compound from *Eupatorium adenophorum* (Spreng) and *Ageratina adenophora* (Spreng) [*sic*] causing hepatotoxicity in mice. *Nat. Toxins* **3**:350-354.
Oelrichs PB, Seawright AA, MacLeod JK, Ng JC (1998) The isolation of a hepatotoxic compound from *Eupatorium adenophorum* (Spreng) and some preliminary studies on its mode of action. Chapter 55 in *Toxic Plants and Other Natural Toxicants*. eds Garland T, Barr AC. CAB International, Wallingford. pp. 271-275.
O'Sullivan BM (1979) Crofton weed (*Eupatorium adenophorum*) toxicity in horses. *Aust. Vet. J.* **55**:19-21.
O'Sullivan, BM, Gibson JA, McKenzie RA (1985) Intoxication of horses by *Eupatorium adenophorum* and *E. riparium* in Australia. In Seawright AA, Hegarty MP, James LF, Keeler RF (eds.) *Plant Toxicology*. Queensland Poisonous Plants Committee, Brisbane. pp. 423-426.
Parsons WT, Cuthbertson EG (2001) *Noxious Weeds of Australia.* 2nd edition. CSIRO Publishing, Melbourne, pp.239-245.
Sharma OP, Dawra RK, Kurade NP, Sharma PD (1998) A review of the toxicosis and biological properties of the genus *Eupatorium*. *Nat. Toxins* **6**:1-14.

Lung – Family Rutaceae

Zieria arborescens *(stinkwood)*

Family Rutaceae
Toxin unknown (associated with essential oils and may be related to furans)
Cattle in Northern Tasmania only → `Panting Disease' (Bruce 1912, Philp 1930)
Most cases in winter
Clinical signs

- anorexia, depression
- reluctance to move
- rapid abdominal breathing, grunting
- slight nasal discharge
- illness usually short, but may last up to 20 days

Pathology

- pulmonary oedema & emphysema
- degeneration of pulmonary arteriole walls

References: Se151

Bruce GS (1912) Panting disease. *Agric. Gaz. Tasmania* **20**:135-136. [cited by Hurst 1942]
Philp T (1930) Poisoning by *Zieria smithii*, stinkwood. *Tasmanian J. Agric.* **1**:161. [cited by Hurst 1942]

LIVER

Liver – Family Asteraceae

Argentipallium blandowskianum *[liver-necrosis-inducing phytotoxin]*

Chemical structure: toxin undescribed
Sources:

Family Asteraceae
Argentipallium blandowskianum [= *Helichrysum blandowskianum*] (woolly everlasting daisy) [Se59, Pullar 1937, McLennan 1938, McAuliffe & White 1976]

Toxicity: cattle, sheep
Mode of action: undescribed
Conditions of poisoning:
Clinical signs: As for acute hepatic necrosis syndromes (*q.v.*)
Pathology: As for acute hepatic necrosis syndromes (*q.v.*)
Diagnosis: As for acute hepatic necrosis syndromes (*q.v.*)

Therapy: As for acute hepatic necrosis syndromes (*q.v.*)
Prevention & control: As for acute hepatic necrosis syndromes (*q.v.*)
References:

McAuliffe PR, White WE (1976) "Woolly everlasting daisy" (*Helichrysum blandowskianum*) toxicity in cattle and sheep. *Aust. Vet. J.* **52**:366-368.
McLennan GC (1938) Poisoning of sheep by *Helichrysum blandowskianum* Steetz. *Aust. Vet. J.* **14**:241.
Pullar EM (1937) Poisoning of sheep by *Helichrysum blandowskianum* Steetz (woolly everlasting). *Aust. Vet. J.* **14**:241-244.

Ozothamnus diosmifolius *[liver-necrosis-inducing phytotoxin]*

Chemical structure: undescribed
Sources:

Family Asteraceae
Ozothamnus diosmifolius [= *Helichrysum diosmifolium*] (sagoflower, riceflower) [1998 unpublished report, DPI Poisonous Plants Database]

Toxicity: cattle
Mode of action: undescribed
Conditions of poisoning:

cattle with access to large amounts of plant either in forest pasture or collected for the floristry trade

Clinical signs: As for acute hepatic necrosis syndromes (*q.v.*)
Pathology: As for acute hepatic necrosis syndromes (*q.v.*)
Diagnosis: As for acute hepatic necrosis syndromes (*q.v.*)
Therapy: As for acute hepatic necrosis syndromes (*q.v.*)
Prevention & control: As for acute hepatic necrosis syndromes (*q.v.*)
References:

Liver – Family Poaceae

Cynosurus echinatus [liver-necrosis-inducing phytotoxin]

Chemical structure: undescribed
Sources:

Family Poaceae
Cynosurus echinatus L. (rough dog's-tail grass), native of the Mediterranean region and naturalised in temperate Australia as far north as the Granite belt-southern Darling Downs of Queensland.
Suspected involvement of mycotoxins

Toxicity: cattle, sheep
Mode of action: undescribed
Conditions of poisoning:

cattle, sheep with access to large amounts of plant in pasture; toxicity cases reported from Tasmania (Middleton 1994, Ladds 2001) and Victoria (Blackwood 1993, Morton 1995)

Clinical signs: As for acute hepatic necrosis syndromes (*q.v.*); photosensitisation in survivors (Ladds 2001)
Pathology: As for acute hepatic necrosis syndromes (*q.v.*); massive hepatocyte necrosis (Ladds 2001)
Diagnosis: As for acute hepatic necrosis syndromes (*q.v.*)
Therapy: As for acute hepatic necrosis syndromes (*q.v.*)
Prevention & control: As for acute hepatic necrosis syndromes (*q.v.*)
References:

Blackwood C (1993) Mysterious bovine liver disease. *Post-Grad. Committee in Vet Science, University of Sydney: Control & Therapy Series*. Mailing 177, No.3537. pp.650-652.
Ladds P (2001) Mount Pleasant Laboratory Report. *Vet. Path. Report (ASVP Newsletter)* No.61, p.27-28.
Morton J (1995) Unpublished reports. Agriculture Victoria (Warrnambool).
Middleton M (1994) Unpublished reports. Department of Primary Industry & Fisheries, Tasmania (Animal Industries Division New Town Research Laboratories)

Liver – Family Ulmaceae

☑ Trema tomentosa *[liver-necrosis-inducing phytotoxin]*

Core data

Common sources: Trema tomentosa (poison peach)
Animals affected: ruminants, horses
Mode of action: undescribed
Poisoning circumstances: access to rainforest margins or cleared forest
Main effects: acute periacinar hepatocyte necrosis
Diagnosis: pathology + plant access
Therapy: nil
Prevention: deny access

Chemical structure:

Trematoxin, a glycoside toxic to laboratory mice and guinea pigs, has been isolated from the plant (Oelrichs 1986), but its structure has not been further elucidated.

Sources:

Family Ulmaceae

There are about 15 species of *Trema* in tropical, subtropical and temperate regions of the world and 2 in Australia - *T. tomentosa* and *T. orientalis*, the latter distinguished by its strongly discolorous leaves (Hewson 1989).

Trema tomentosa (Roxb.) Hara [= *Trema aspera* (Brong.) Blume] (poison peach, peach-leaf poison bush, native peach) [DM143] is recognised as toxic, but there appears to be variation in toxicity within the population with some plants apparently non-toxic (Hurst 1942, McCray 1970), suggesting that further investigation may reveal distinct chemotypes or subspecies. Currently, two varieties are recognised based on the degree of hairiness of mature leaves - *tomentosa* and *viridis. T. tomentosa* is distributed in coastal to near-coastal WA, NT, Qld, NSW & Vic and also in tropical Africa, India, SE Asia and tropical Pacific islands to Hawaii. In Australia, its habitat is the margins of coastal rainforest and vine thickets and in open eucalypt woodland. It favours water course margins. Populations occur along water courses in central Australia. (Hewson 1989)

Trema tomentosa flowering & fruiting branch. Note the alternate leaf arrangement and clusters of small white flowers and immature green berries. [RAM Photo]

Trema tomentosa fruiting branch. Note the clusters of mature black berries. [RAM Photo]

Toxicity:

Species affected: **cattle** (Shepherd 1871, Mulhearn 1942, Hall 1964), goats (Shepherd 1871, Jerrett 1988), deer (McKenzie 1985), camels (Trueman & Powell 1991), horses (Hill *et al.* 1985)

Mode of action: undescribed

Conditions of poisoning:

Access to rainforest margins or cleared rainforest. *Trema tomentosa* regrows vigorously after clearing and may be the dominant vegetation under those conditions. Hungry stock introduced to such situations can suffer large mortalities. Cases are reputed to be more frequent when pasture is dry.

Clinical signs: As for acute hepatic necrosis syndromes (*q.v.*)

Pathology: acute periacinar hepatic necrosis

Diagnosis: As for acute hepatic necrosis syndromes (*q.v.*)

Therapy: As for acute hepatic necrosis syndromes (*q.v.*)

Prevention & control: As for acute hepatic necrosis syndromes (*q.v.*)

References:

Se135

Hall WTK (1964) *Aust. Vet. J.* **40**:180.

Hewson HJ (1989) Ulmaceae. *Flora of Australia* **3**:4-13.

Hill BD, Wills LD, Dowling RM (1985) Suspected poisoning of horses by *Trema aspera* (poison peach). *Aust. Vet. J.* **62**:107-108.

Hurst E (1940) *The Poison Plants of New South Wales.* New South Wales poison Plants Committee, Sydney. pp.72-74.

Jerrett I (1988) Poison peach hepatopathy in goats. *Vet. Pathol. Report (ASVP Newsletter)* No. 22, p.19. [confirmatory feeding experiment with sheep]

McKenzie RA (1985) Necropsy findings in red, fallow and rusa deer from south-eastern Queensland. Chapter 4 in McKenzie RA (ed) *Deer Farming Techniques and Diseases of Deer in Queensland.* Department of Primary Industries, Brisbane. pp.27-30.

McCray CWR (1970) Queensland Poisonous Plants Committee Minutes, 10 June 1970, cited by Everist (1981)

McKenzie RA (1992) *Trema tomentosa* poisoning [red deer]. *Vet. Pathol. Report (ASVP Newsletter)* No.34, pp.21-22.

Mulhearn CR (1942) Poison peach (*Trema aspera*): a plant poisonous to stock. *Aust. Vet. J.* **18**:68-72.

Oelrichs PB (1968) Isolation and purification of trematoxin from *Trema aspera*. *Phytochem*. **7**:1691-1693.

Shepherd TW (1871) Stray notes on indigenous and acclimatised medicinal plants in New South Wales. *NSW Med. Gaz.* **2**:71-74. Cited by Hurst (1942)

Trueman KF, Powell MW (1991) Suspected poisoning of camels by *Trema tomentosa* (poison peach). *Aust. Vet. J.* **68**:213-214.

KIDNEY

Kidney – Family Araceae

Philodendron *spp. – cats*

Philodendron spp. and their hybrids are popularly houseplants and potplants from the Family Araceae. Originating in tropical America, 350-400 species are recognised. (Mabberley 1997)

Spoerke & Smolinske (1990) list the following species cultivated in North America:

Philodendron cordatum Kunth
Philodendron scandens Koch & Sello ssp. *oxycardium* (Schott) Bunting [= *P. oxycardium* Schott, *P. cordatum* Hort.] (parlour ivy)
Philodendron bipennifolium Schott [= *P. panduriforme* Hort.] (panda plant)
Philodendron selloum Koch [= *P. johnsii* Hort.]
Philodendron simsii Kunth
Philodendron speciosum Schott

Mabberley (1997) gives the following as the most familiar:

Philodendron bipinnatifidum Endl.
Philodendron scandens K.Koch & Sello

Sellers *et al.* (1978) record 0.7% of the plant as calcium oxalate crystals. Contact dermatitis in humans is thought to be caused by alkyl resorcinol (Reffstrup *et al.* 1982). The nephrotoxin affecting cats is unknown.

Cats are very susceptible to toxicity from chewing the leaves of *Philodendron* spp. used as houseplants. Mortality can be high with 37 of 72 cases reported by Greer (1961) being fatal.

Clinical signs are usually those of renal failure (Greer 1961, Brogger 1970). One case dominated by signs of CNS involvement (excitability, muscle twitching, trembling, teeth grinding) has been reported (Pierce 1970).

References:

Brogger JN (1970) *Mod. Vet. Pract.* **51**:46-.
Greer MJ (1961) Plant poisoning in cats. *Mod. Vet. Pract.* **42** (20):62-.
Pierce JH (1970) Encephalitis signs from *Philodendron* leaf. *Mod. Vet. Pract.* **51**:42-.
Reffstrup T, Hammershoy O, Boll PM, Schmidt H (1982) *Philodendron scandens* Koch et Sello ssp. *oxycardium* (Schott) Bunting, a new source of allergenic alkyl resorcinols. *Acta Chem. Scand.* **36**:291-.
Sellers SJ *et al.* (1978) *Vet. Human Toxicol.* **20**:92-.
Spoerke DG, Smolinske SC (1990) *Toxicity of Houseplants.* CRC Press, Boca Raton, Florida. pp.184-185.

Kidney – Family Caesalpiniaceae

Schotia brachypetala *(drunken parrot tree)*

Nephrotoxin unknown

Schotia brachypetala (drunken parrot tree, tree fuschia, Hottentot bean, Boer bean, weeping Boer bean, huilboerboon (Afrikaans)); Family Caesalpiniaceae; native to southern Africa, cultivated as a street and garden tree in Australia.

Associated with nephrosis in cattle which browsed on flowering branches at Maryborough in September 1998 (RA McKenzie, GJ Storie & IR Fraser, unpublished data, Queensland DPI Poisonous Plant Files, 1998). 80 2-3 year-old Brahman cattle introduced to a paddock with branches of *S. brachypetala* overhanging the fence line; onset of illness 2 weeks after introduction; 6 died; clinical signs – loss of weight, crusty nasal exudate, pale mucous membranes, tarry faeces; clinical pathology (2 steers) – dehydration with either slight leucopaenia or slight absolute neutrophilia + increased concentrations of creatinine, urea & Mg, decreased Ca; necropsy (1 steer) – pale kidneys, haemorrhages throughout the alimentary tract, swollen liver, congested lungs; histopathology – severe subacute diffuse necrosis of epithelium of proximal renal cortical tubules, no oxalate crystals detected.

No literature record of toxicity for domestic animals. Bark, wood and root contain tannins; a decoction of the bark is emetic; wood dust is irritant to the eyes; seeds are edible (Watt & Breyer-Brandwijk 1962). Polyhydroxystilbenes have been isolated from heartwood, but their physiological activity is unknown (Drewes & Fletcher 1974).

References:

Drews SE, Fletcher IP (1974) Polyhydroxystilbenes from the heartwood of *Schotia brachypetala*. *J. Chem. Soc. Perkin Trans.* **1**:961-962 [cited by van Wyk BE, van Oudtshoorn B, Gerike N (1997) *Medicinal Plants of South Africa.* Briza Publications, Pretoria.]

Watt JM, Breyer-Brandwijk MG (1962) *Medicinal and Poisonous Plants of Southern and Eastern Africa.* 2nd. ed. Livingstone, Edinburgh. pp.645-646.

Kidney – Family Lythraceae

Lythrum hyssopifolia *(lesser loosestrife)*

Chemical structure:

Nephrotoxin & hepatotoxin unknown

Source:

- *Lythrum hyssopifolia* L. (lesser loosestrife); Family Lythraceae; a genus of *ca.* 35 species worldwide with 5 in Australia, all probably native; *L. hyssopifolia* distributed in southern Europe and eastern Australia in moist habitats (mostly NSW, Vic, Tas but some in Q, SA, WA) (Hewson 1990)
  - in damp sandy soils or near streams (Lenghaus 1987)
  - germinates autumn-winter, flowers summer (Glastonbury *et al.* 1991)

Toxicity:

- **sheep**
- reported natural intoxication incidents; no reports published in refereed literature except a misattribution of the syndrome to *Anagallis arvensis* (scarlet pimpernel) by Rothwell & Marshall (1986)
  - 1987, Horsham & Stawell (Vic): 4 flocks, 2-8% deaths in adult sheep & weaners (Lenghaus 1987)
  - 1989, Benalla (Vic): (Hindmarsh 1989)
  - 1989/90 & 1990/91, NSW South-western Slopes: 5 flocks,
  - 1991, Wagga Wagga (NSW): morbidity 2.5% (640) and case fatality 62.5%, 9 month-old Merino wethers; mortality 3.0-6.5% (25/750 – 40/620) (Glastonbury 1991)
  - 1993, Benalla (Vic): 2.5% (10/400) deaths (Nimmo-Wilkie & Lancaster 1993)
  - 1993, Wagga Wagga (NSW): mortality data not reported (Glastonbury 1993)
  - 1999, Wagga Wagga (NSW): 2.7% (300 head) deaths (Glastonbury 1999)
  - 1999, Tenterden & Tambellup (WA): 10/300 & 20/390 deaths (Forshaw 1999)
  - 2001, Horsham district (Vic): >50/400 deaths (R Crawford, personal communication 30 April 2001; Jubb 2001)
  - 2002, Wimmera district (Vic): 30/600 deaths in 7-month-old cross-bred lambs (J Samuel, personal communication 21 Jan 2002)
- syndrome reproduced experimentally
  - green succulent *L. hyssopifolia* from 2 different sources fed to 2 sheep for 12 days (no dose rate cited); serum urea, creatinine & liver-associated enzyme concentrations increased from Day 7; necropsy unremarkable; nephrosis + increased sporadic hepatocellular death seen histologically (Lenghaus 1987)

Mode of action: unknown, but observations suggest toxin in sufficient dose is firstly hepatotoxic with its metabolite(s) are then nephrotoxic (Forshaw 1999)

Circumstances of poisoning:

- *L. hyssopifolia* predominates in fallows and cereal stubbles in some years in NSW South-western Slopes (Glastonbury *et al.* 1991) after abnormal summer rains (Seawright 1989)
- sheep with access to pasture dominated by *L. hyssopifolia*, for example:
  - sheep turned onto stubble paddocks with large amount of *L. hyssopifolia* present (Lenghaus 1987)
  - sheep grazing canola, wheat or oat stubbles with abundant *L. hyssopifolia* providing the only green feed (Glastonbury *et al.* 1991)
  - sheep grazing oat stubble severely waterlogged during winter and containing a significant amount of *L. hyssopifolia* (Glastonbury 1991)
  - sheep turned onto dry pasture with *L. hyssopifolia* as the only green plant material available (Nimmo-Wilkie & Lancaster 1993, Wilkie & Lancaster 1993)
  - sheep turned onto pea stubble containing significant amounts of *L. hyssopifolia* (Glastonbury 1999)
  - sheep grazing canola stubble with *L. hyssopifolia* providing the only green feed (Forshaw 1999)

- Merino lambs grazing bean and canola stubble with *L. hyssopifolia* present (R Crawford, personal communication 30 April 2001)
- lambs grazing barley stubble 4 days before deaths began (J Samuel, personal communication 21 Jan 2002)

Clinical signs:

Subacute (Lenghaus 1987, Glastonbury *et al.* 1991))

- time from access to first death 3-14 days (Glastonbury *et al.* 1991)
- found dead within 7-10 days of access to plants (Lenghaus 1987)
- depression, lethargy
- death without struggling

Chronic (Glastonbury 1991)

- weight loss
- weakness
- "tucked-up" appearance

Pathology (Lenghaus 1987, Glastonbury *et al.* 1991, Nimmo-Wilkie & Lancaster 1993):

Clinical pathology

- ↑ serum creatinine & urea ± ↑ serum bilirubin & liver-associated enzymes (including GLDH)

Necropsy

- scattered petechial haemorrhages (subcutis, kidneys, diaphragm, thoracic wall, omentum, mediastinum, heart)
- perirenal oedema, mild ascites
- pale swollen kidneys
- ± jaundice
- ± tan or orange-coloured swollen liver

Histopathology

- **renal tubular necrosis**: coagulation necrosis of tubular epithelium, hyaline casts
  - interstitial fibrosis & loss of tubules (cases with access > 5 days)
  - dilated ascending loops of Henle & distal convoluted tubules + some tubular regeneration (cases with access > 10 days)
  - ± slight to moderate haemoglobinuric nephrosis and/or renal haemosiderosis
- ± hepatocellular damage
  - ± periacinar or mid-zonal hepatocyte necrosis + biliary hyperplasia
  - ± hepatocyte fatty change
  - ± individual hepatocyte necrosis

Diagnosis: access + pathology

Therapy:

- no specific therapy
- deaths cease quickly after removal from access (Lenghaus 1987); but not in all cases (Forshaw 1999)

Prevention and control: prevent access

References:

Forshaw D (1999) Lesser loosestrife (*Lythrum hyssopifolia*) toxicity in sheep. *Vet. Path. Report (Newsletter Aust. Soc. Vet. Path.)*, No.53 (September), p.26.

Glastonbury JRW (1991) "Lesser loosestrife", *Lythrum hyssopifolia* poisoning. *Vet. Path. Report (Newsletter Aust. Soc. Vet. Path.)*, No.30 (March), p.20.

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Hindmarsh M (1989) *North East Animal Health Newsletter (Vic. Dept. Agric.)*, December, p.5.

Jubb T (2001) *Animal Health Australia Animal Health Surveillance Quarterly Report 1 January - 31 March 2001.* **6** (1):16-17.

Lenghaus C (1987) Lesser loosestrife. *Vet. Path. Report (Newsletter Aust. Soc. Vet. Path.)*, No 16 (April), p.14.

Nimmo-Wilkie JS, Lancaster M (1993) Lesser loosestrife poisoning in sheep. *Vet. Path. Report (Newsletter Aust. Soc. Vet. Path.)*, No.36 (February), p.23.

Rothwell JT, Marshall DJ (1986) Suspected poisoning of sheep by *Anagallis arvensis* (scarlet pimpernel). *Aust. Vet. J.* **63**:316.

Seawright AA (1989) *Animal Health in Australia. Volume 2 (Second Edition). Chemical and Plant Poisons.* Australian Government Publishing Service, Canberra. p.93.

Wilkie J, Lancaster M (1993) Sun, rain, weeds. *North East Animal Health Newsletter (Vic. Dept. Agric.)*, March, p.3.

Kidney – Family Poaceae

☑ Pennisetum clandestinum *(kikuyu grass) (*q.v.*)*

Kidney – Family Vitaceae

Vitis vinifera *(grapes or raisins) – dogs*

Chemical structure: Toxin(s) unknown
Sources:
grapes (red seedless variety in 3 of 5 cases)
raisins
Organ systems affected: kidney
Toxicity:
dogs
doses ingested ranged between 1.2 & 4.4 kg (56 – 150 g/kg)
Mode of action: undescribed
Conditions of poisoning:
dogs ingesting large amounts of grapes or raisins
– fresh table grapes or from vines or crushings from wineries
- raisins from various commercial sources
Clinical signs:
- vomiting within hours of ingestion in all cases: vomitus contained partly-digested grape/raisin
- anorexia (60% of cases)
- diarrhoea (50%)
- oliguria or anuria with or without isosthenuria (50%)
- lethargy (40%)
- signs of abdominal pain (30%)

signs continued for several days to 3 weeks after ingestion
Pathology:
Clinical biochemistry abnormalities developed 24 hr to several days after ingestion
- azotaemia (90%): 23-209 mg BUN/dl
- increased serum creatinine concentrations (90%): 4.3-18 mg/dl
- hypercalcaemia (70% of cases): 12.3-26.0 mg Ca/dl
- hyperphosphataemia (70%): 6.4-22.0 mg PO_4/dl
- increased Ca x PO_4 product (70%): 81-390 mg/dl

Necropsy & histopathology (1 dog only):
- mild renal tubular damage
- metastatic mineralisation of numerous tissues

Diagnosis:
history of ingestion + syndrome
Therapy:
Prognosis is poor if oliguria or anuria develops (half the observed 10 cases died or were killed)
Recommended therapeutic regimen:
- decontaminate (emesis, lavage, activated charcoal)
- IV fluids for a minimum of 48 hrs
- monitor kidney function (serum biochemistry) for 72 hrs

Recovery in observed cases followed vigorous treatment including:
- IV fluids
- diuretics (mannitol, furosemide)
- dopamine
- peritoneal dialysis

Prevention & control:
prevent large intakes of grapes (fresh or dried)
References:

Gwaltney-Brant S, Holding JK, Donaldson CW, Eubig PA, Khan SA (2001) Renal failure associated with ingestion of grapes or raisins in dogs. (Letter to the editor) *J. Am. Vet. Med. Assoc.* **218**:1555-1556.

HAEMOLYSIS

Haemolysis – Family Aceraceae

Acer *spp. (maples)*

Chemical structure:

Gallic acid + another uncharacterised oxidant have been found in *Acer* spp. leaves and shown to be capable of causing haemolysis and methaemoglobin formation with equine erythrocytes *in vitro* (Boyer *et al.* 2002)

2,3-dihydro-3,5-dihydroxy-6-methoxy-4H-pyran-4-one, a potential co-oxidant, was found in aqueous extract of *A. rubrum* and may contribute to methaemoglobin formation (Boyer *et al.* 2002)

Sources:

***Acer* spp. (maple trees)** native to North America; cultivated elsewhere

Acer rubrum (red maple, Canadian maple, swamp maple, soft maple)

Acer saccharinum (silver maple) (Murphy M, personal communication VETTOX 1997, Boyer *et al.* 2002)

Acer saccharum (sugar, striped, fiddle-back or bird's eye maple) (DJ Blodgett, personal communication VETTOX 1997, Boyer *et al.* 2002)

Toxicity

horses in North America

Wilted leaves are toxic. The toxicity is not destroyed by prolonged storage or freezing for short periods (George *et al.* 1982).

As little as 1.5 g/kg *A. rubrum* leaves have been fatal (George *et al.* 1982)

Equids have a decreased capacity to reduce methaemoglobin compared with other mammals (Robin & Harley 1967) and equine erythrocytes appear to be deficient in the system that links glutathione reduction to the pentose phosphate shunt, impairing capacity to deal efficiently with oxidative damage (Boyer *et al.* 2002).

Clinical signs

sudden death
depression, lethargy
weakness
polypnea
tachycardia
pale mucous membranes
jaundice
cyanosis
haemoglobinuria
± abortion (Stair *et al.* 1993)

Two clinical patterns have been described (George *et al.* 1982): horses dying of methaemoglobinaemia within 18 hr and others dying of haemolytic anaemia in 3-5 days

Pathology

methaemoglobinaemia
Heinz body haemolytic anaemia
jaundice
splenomegaly
swollen, black kidneys (nephrosis with haemoglobin casts)
periacinar hepatocyte degeneration (hypoxia)

Therapy

ascorbic acid (vitamin C) for its antioxidant effect (McConnico & Brownie 1992)
@ 125 mg/kg orally, then 50 mg/kg SC, twice daily
or @ 30 mg/kg/12 hours by IV infusion replaced by oral doses after several days

blood transfusion, IV fluid therapy

Prevention

Deny equids access to shed leaves of *Acer* spp.

References:

Boyer JD, Breeden DC, Brown DL (2002) Isolation, identification, and characterization of compounds from *Acer rubrum* capable of oxidising equine erythrocytes. *Am. J. Vet. Res.* **63**"604-610.

Divers TJ, George LW, George JW (1982) Hemolytic anemia in horses after ingestion of red maple leaves. *J. Am. Vet. Med. Assoc.* **180**:300-302.

George LW, Divers TJ, Mahaffey EA, Suarez MJH (1982) Heinz body anemia and methemoglobinemia in ponies given red maple (*Acer rubrum* L.) leaves. *Vet. Pathol.* **19**:521-533.

Long PH, Payne JW (1984) Red maple-associated pulmonary thrombosis in a horse. *J. Am. Vet. Med. Assoc.* **184**:977-978.

McConnico RS, Brownie CF (1992) The use of ascorbic acid in the treatment of 2 cases of red maple (*Acer rubrum*)-poisoned horses. *Cornell Vet.* **82**:293-300.

Plumlee KH (1991) Red maple toxicity in a horse. *Vet. Human Toxicol.* **33**:66-67.

Robin H, Harley J (1967) Regulation of methaemoglobinaemia in horse and human erythrocytes. *Aust. J. Exp. Biol. Med. Sci.* **45**:77-88.

Stair EL, Edwards WC, Burrows GE, Torbeck K (1993) Suspected red maple (*Acer rubrum*) toxicosis with abortion in two Percheron mares. *Vet. Human Toxicol.* **35**:229-230.

Tennant B, Gill SG, Glickman LT, Mirro EJ, King JM, Polak DM, Smith MC, Kradel DC (1981) Acute hemolytic anemia, methemoglobinemia, and Heinz body formation associated with ingestion of red maple leaves by horses. *J. Am. Vet. Med. Assoc.* **179**:143-150.

Weber M, Miller E (1997) Presumptive red maple (*Acer rubrum*) toxicosis in Grevy's zebra (*Equus grevy*). *J. Zoo Wildl. Med.* **28**:105-108.

RED URINE (NON-HAEM PIGMENTS)

Red urine (non-haem pigments) – Family Fabaceae

Trifolium pratense *(red clover) – red urine*

Deer in New Zealand

Unidentified pigment

Urine is colourless when voided

→ scarlet-red about 1 hour after exposure to air

→ red-stained hocks, tail, perineum

No ill health in affected deer

Reference:

Niezen JH, Barry TN, Wilson PR, Lane G (1992) Red urine from red deer grazed on pure red clover swards. *N. Z. Vet. J.* **40**:164-167.

Red urine (non-haem pigments) – Family Xanthorrhaceae

Xanthorrhoea minor *ssp.* lutea *(grasstree) – urine red pigmentation*

Cattle in Victoria

Pigment probably a phenol

No ill-health in affected cattle

Reference: Se149

Harrison MA, Beilby CA, Friend SCE, Caple IW (1978) Redwater in cattle associated with ingestion of *Xanthorrhoea minor*. *Aust. Vet. J.* **54**:40.

NERVOUS – CONVULSIONS

Nervous – Convulsions – Family Combretaceae

Terminalia oblongata *(yellow-wood) - sheep*

See notes on yellow-wood under tannins.

Convulsive syndrome

Sheep

Toxin unknown.

Clinical signs

- sudden noise or disturbance → transient convulsions

References: DM155

Nervous – Convulsions – Family Mussaceae

Musa *sp. (bananas)*

Most cultivated bananas are hybrids of *Musa acuminata* and *Musa balbisiana* and are sometimes known as *Musa* x *paradisica* but their taxonomy is confused (Jackson & Jacobs 1985).

Dogs:

There has been a report from a veterinarian through the Queensland Poisons Information Centre of two incidents of strychnine-like tetanic seizures in puppies that had access to banana flowers (Unpublished data, Queensland Poisonous Plants Database, 27 Sep 1990).

Cattle:

Cattle fed bananas in north-eastern New South Wales developed neurological signs (ARB Jackson, personal communication 17 April 2001).

Green bananas are reported to have caused sickness in cattle [no further details provided] (Hungerford 1990).

References:

Hungerford TG (1990) *Diseases of Livestock.* 9th edition. McGraw-Hill Book Co., Sydney. p.1648.

Jackson DL, Jacobs SWL (1985) *Australian Agricultural Botany.* Sydney University Press, Sydney. p.333.

McKinney B, Crawford MA (1965) Fibrosis in guinea-pig heart produced by plantain diet. *Lancet* **2**:880-882.

Nervous – Convulsions – Family Solanaceae

Brunfelsia *spp. (francisia, yesterday-today-&-tomorrow)*

Core data

Common sources: Brunfelsia australis (francisia, yesterday-today-and-tomorrow)
Animals affected: dogs
Mode of action: undescribed
Poisoning circumstances: ripe fruits consumed by young dogs
Main effects: convulsions, gastroenteritis
Diagnosis: syndrome + fruit access
Therapy: decontamination + anticonvulsants
Prevention: deny access to fruits

Chemical structure:

Neurotoxin unknown

Sources:

Brunfelsia spp. (about 40 species; South American origin; Family Solanaceae)
known toxic species

Brunfelsia australis [= *Brunfelsia bonodora*] (francisia, yesterday-today-and-tomorrow) in Australia (McBarron & de Sarem 1975; Neilson & Burren 1983)

Brunfelsia calycina in North America (Spainhour *et al.* 1990)

Toxicity: **dogs**, cattle, laboratory rodents

Mode of action: undescribed

Conditions of poisoning: Fruits toxic. Dogs attracted to ripe fruit will eat large amounts

Clinical signs:

- vomiting
- diarrhoea
- urination
- ataxia
- muscle tremor
- convulsions: extensor rigidity, opisthotonus
- ± excitement, nystagmus, stomatitis, haematuria

Pathology: gastroenteritis
Diagnosis: differential diagnoses include strychnine, *Bufo marinus*, metaldehyde, organophosphorus compounds, *Macadamia* nut poisonings, canine distemper
Therapy:
- relapses occur
- emetic followed by activated charcoal @ 1-3 g/kg plus a saline cathartic (e.g. $MgSO_4$ @ 1g/kg)
- anticonvulsant therapy (diazepam IV) / anaesthesia (pentobarbitone sodium IV) used successfully to treat seizures (Neilson & Burren 1983)

Prevention & control: deny access
References:

Se314
McBarron EJ (1975) *Brunfelsia* fruits poisonous to dogs. *Agric. Gazette NSW* **86**:36-37.
McBarron EJ, de Sarem W (1975) Poisoning of dogs by the fruits of the garden shrub *Brunfelsia bonodora*. *Aust. Vet. J.* **51**:280.
Neilson J, Burren V. (1983) Intoxication of 2 dogs by fruit of *Brunfelsia australis*. *Aust. Vet. J.*. **60**:379-380.
Spainhour CB, Fiske RA, Flory W, Reagor JC (1990) A toxicological investigation of the garden shrub *Brunfelsia calycina* var. *floribunda* (yesterday-today-and-tomorrow) in three species. *J. Vet. Diagn. Invest.* **2**:3-8.

NERVOUS – MANIA

Nervous – Mania – Family Fabaceae

Pisum sativum *var.* arvense *(field pea)*

Incidents of manic behaviour in cattle and sheep grazing pea crops or silage made from them have been recorded from Victoria (Lenghaus 1987) and one such case has occurred in cattle in southern Queensland (Reardon & McKenzie 2002).

One particular case involved a mob of 23 2-year-old Simmental or Simmental x Hereford bulls moved onto a sparse drying pea crop (specific identity not stated; presumed to be *Pisum arvense* – field pea – based on data in Jackson & Jacobs 1985) in a perennial ryegrass pasture in the Hamilton region of western Victoria (Lenghaus 1987). One week later on being driven for yarding, one bull became intractable and jumped over or through 5 fences before collapsing. Subsequently it had intermittent convulsions, became comatose and died in 24 hr. About one third of the mob had signs of hyperexcitability or frenzy, incoordination, and inability to charge, excessive salivation and retention of food in the mouth. Affected bulls were easily stimulated into extreme mania leading to convulsions. If left alone, they quickly quietened. They did not appear blind. The mob was treated with injected steroids, diuretics and thiamine and thiamine powder was added to hay for 3 days to address possible cerebral oedema or polioencephalomalacia. All but one recovered during the following week. One bull developed a dummy state. Histopathology of tissues from the first dead bull revealed perivascular oedema around medium-sized blood vessels and sporadic neuronal necrosis in the cerebrum and aspiration pneumonia. Mature cows subsequently grazing the crop were unaffected. Numerous seedheads of ryegrass were parasitised by *Claviceps purpurea*, but Lenghaus (1987) notes anecdotal reports other cases of mania in cattle grazing pea crop stubbles where ergotism was not a differential diagnosis and in lambs fed pea crop silage.

About 40 1-2 year-old home-bred Hereford heifers and cows near Warwick, Queensland in early September 2001 grazed a pea crop (*Pisum sativum* var. *arvense*) at the pre-flowering stage of growth for about 14 days (Reardon & McKenzie 2002). On being yarded, they generally appeared aggitated and 80-90% had bilateral serous nasal discharge. Five % had noticable hyperexcitability progressing to mania if stimulated. Mania was manifest by such behaviour as leaping into troughs and then charging back into the mob or breaking through fences while being moved to the yards (2 animals). Noise (for example a motorcycle engine) particularly triggered manic behaviour. Affected animals were hypersensitive to external stimuli, bellowed repeatedly, urinated frequently. Heart rates were up to 100 beats/min. Rectal temperatures were normal. No abnormal odour was detected in the breath suggestive of ketosis. Plasma Ca and Mg were within normal ranges. A sample of the crop examined for fungal pathogens and endophytes yielded no pathogens. A previous mob of cattle on the crop contained 2 animals with broken hind limbs and one animal reported to be circling. The pea crop planted on 30 April 01 was on clay-loam soil. The previous crop had been soyabeans (*Glycine max*) planted on 4 February 00. The paddock had received 200 kg superphosphate/ha on 4 February 00, 3 tonne agricultural lime/ha in February 2001 and

160 kg superphosphate/ha on 30 May 01. The pea crop had been under some water stress until a recent 25 mm fall of rain.

References:

Jackson DL, Jacobs SWL (1985) *Australian Agricultural Botany.* Sydney University Press, Sydney. p.162.
Lenghaus C (1987) Peas. *Vet. Path. Report (ASVP Newsletter)* No.16, p.13.
Reardon CR, McKenzie RA (2002) Pea mania: deranged behaviour in cattle grazing a pea crop. *Aust. Vet. J.* **80**:16-19.

NERVOUS – ATAXIA

Nervous – Ataxia – Families Cycadaceae & Zamiaceae

☑ *Cycads*

Zamias in May
Lift up bulging sporophylls.
Hush *Meliphaga*!
Haiku (2 May 1984). R.A. McKenzie

Core data

Syndrome name: zamia staggers
Common sources: Cycas spp., *Macrozamia* spp.
Animals affected: cattle
Mode of action:

- toxin unknown
- MAM derivative suspected

Poisoning circumstances: young leaves or seeds ingested
Main effects:

- irreversible posterior ataxia
- spinal cord white matter degeneration

Diagnosis: access + pathology
Therapy: nil
Prevention: deny access

Cycads (zamias) have grown on earth for at least 250 million years. This plant group is older than the dinosaurs and, unlike them, still survives in the Americas, Africa, Asia and the Pacific islands as well as in Australia. These plants form an important part of the botanical heritage of Australia and the world. As a community, we have a responsibility to preserve viable populations of cycads in their natural habitats. One challenge is to develop management systems that minimise the damage that cycad poisoning can do to livestock production enterprises while preserving the plants for future generations of humans to appreciate. Cycads are stately and beautiful plants and deserve respect and preservation.

Syndrome names:

- **zamia staggers** (Australia)
- rickets [*sic*] (Australia)
- wobbles (Australia)
- derriengue (Caribbean)

Chemical structure:

The neurotoxin responsible for this syndrome is **unidentified**. A derivative of methylazoxymethanol (MAM) (*q.v.*) produced by metabolism by ruminal microbes is suspected (Shimizu *et al.* 1986, Seawright *et al.* 1998). Shimizu *et al.* (1986) produced spinal cord lesions in goats dosed orally with cycasin (4-6 mg/kg for 88-124 days).

A crystalline toxin, later recognised from *Macrozamia riedlei* material as a glycoside of MAM (Lythgoe & Riggs 1949, Langley *et al.* 1951), was first characterised by Cooper (1940) from the Australian cycad *Macrozamia spiralis*. Neocycasins were described from *Cycas revoluta* and *C. circinalis* by Nagahara (1964). The crystal structure of macrozamin was described by Cannon *et al.* (1980).

MAM glycosides (cycasin, neocycasins and macrozamin) are intrinsically non-toxic (parenteral administration causes no toxicity)

MAM glycosides have been demonstrated in all genera of Australian cycads; concentrations are often low and variable (Moretti *et al.* 1983, Seawright *et al.* 1998a). Osborne & Nair (1993) noted overestimates from using certain assay methods (chromotropic acid, HPLC). de Luca *et al.* (1980) and Moretti *et al.* (1981, 1983) used GLC method.

MAM glycoside concentration in seeds >> leaves.

Macrozamin concentration in *M. riedlei* sarcotesta (red fleshy outer covering of seeds) greatly exceed that in the kernel of seeds.

Cycad species*	Origin	Plant part	Cycasin [% fresh weight]	Macrozamin [% fresh weight]	References
Bowenia spectabilis	Australia	seeds	0.42		de Luca *et al.* 1980
"	Australia	seeds	0.42	5.04	Moretti *et al.* 1981, 1983
Bowenia serrulata	Australia	seeds	0.26	4.33	Moretti *et al.* 1981, 1983
Cycas basaltica	Australia	seeds	0.12	0.29	Moretti *et al.* 1981, 1983
Cycas cairnsiana	Australia	seeds	0.10	0.20	Moretti *et al.* 1981, 1983
Cycas lane-poolei	Australia	seeds	0.72		de Luca *et al.* 1980
"	Australia	seeds	0.72	0.45	Moretti *et al.* 1981, 1983
Cycas pruinosa	Australia	seeds	0.10	0.33	Moretti *et al.* 1981, 1983
Lepidozamia peroffskyana	Australia	seeds	0.21		de Luca *et al.* 1980
"	Australia	seeds	0.21	1.11	Moretti *et al.* 1981, 1983
Macrozamia diplomera	Australia	seeds	0.16		de Luca *et al.* 1980
"	Australia	seeds	0.16	2.41	Moretti *et al.* 1981, 1983
Macrozamia fawcettii	Australia	seeds	0.06	2.49	Moretti *et al.* 1983
Macrozamia heteromera	Australia	seeds	0.08		de Luca *et al.* 1980
Macrozamia miquelii	Australia	seeds	0.09	3.88	Moretti *et al.* 1981, 1983
Macrozamia moorei	Australia	seeds	0.08		de Luca *et al.* 1980
"	Australia	seeds	0.08	3.72	Moretti *et al.* 1981, 1983
Macrozamia riedlei	Australia	seeds		0.4	Cannon *et al.* 1980
"	Australia	sarcotesta		3.88	Ladd *et al.* 1993
Ceratozamia matudai	Mexico	seeds	0.08	1.05	Moretti *et al.* 1983
Ceratozamia mexicana	Mexico	ovules	0.01		de Luca *et al.* 1980
"		seeds	0.02	1.01	Moretti *et al.* 1983
Ceratozamia sp. Hidalgo	Mexico	seeds	0.01	1.06	Moretti *et al.* 1983
Ceratozamia sp. Oaxaca	Mexico	seeds	0.03	1.02	Moretti *et al.* 1983
Cycas circinnalis	Pacific	seeds	0.10	0.42	Moretti *et al.* 1983
Cycas revoluta	Japan	seeds	0.28		de Luca *et al.* 1980
"		seeds	0.28	0.26	Moretti *et al.* 1981, 1983
Cycas thouvarsii	E.Afr	seeds	0.06	0.31	Moretti *et al.* 1983
Dioon califanoi	S.Am	seeds	0.04		de Luca *et al.* 1980
Dioon edule	S.Am	seeds	0.02		de Luca *et al.* 1980
Dioon edule var. *angustifolium*	S.Am	seeds	0.12	0.64	Moretti *et al.* 1983
Dioon purpusii	S.Am	seeds	0.02		de Luca *et al.* 1980
Dioon sp. Guerrero	Mexico	seeds	0.13	0.62	Moretti *et al.* 1983
Dioon sp. Nayarit	Mexico	seeds	0.01	0.65	Moretti *et al.* 1983
Dioon sp. Sonora	Mexico	seeds	0.01	0.68	Moretti *et al.* 1983
Dioon sp. Puerto Escondido, Oaxaca	Mexico	seeds	0.03		de Luca *et al.* 1980
Encephalartos altensteinii	S.Afr	seeds	0.06		de Luca *et al.* 1980
"		seeds	0.07	2.11	Moretti *et al.* 1983
Encephalartos ferox	S.Afr	seeds	0.05	2.10	Moretti *et al.* 1983
Encephalartos lebomboensis	S.Afr	seeds	0.05	2.16	Moretti *et al.* 1983
Encephalartos umbeluziensis	S.Afr	seeds	0.09		de Luca *et al.* 1980
"		seeds	0.08	2.86	Moretti *et al.* 1983
Encephalartos villosus	S.Afr	seeds	0.08	2.09	Moretti *et al.* 1983
Microcycas calocoma	Caribbean	fronds	0.05		de Luca *et al.* 1980
"		seeds	0.07	0.13	Moretti *et al.* 1983
Stangeria eriopus	S.Afr	seeds	0.03		de Luca *et al.* 1980
"		seeds	0.02	4.70	Moretti *et al.* 1983
Zamia integrifolia	Florida	seeds	0.38		de Luca *et al.* 1980
Zamia latifoliolata	Caribbean	seeds	0.02	1.02	Moretti *et al.* 1983
Zamia sp. Nayarit	Mexico	seeds	0.01	1.25	Moretti *et al.* 1983
Zamia sp. Oaxaca	Mexico	seeds	0.16	1.16	Moretti *et al.* 1983
Zamia sp. Oaxaca	Mexico	seeds	0.15	1.23	Moretti *et al.* 1983
Zamia sp. Vera Cruz	Mexico	seeds	0.28	1.01	Moretti *et al.* 1983

*** exotic species may be cultivated in Australia**

MAM glycosides hydrolysed in rumen → MAM (*q.v.*) which yields a hepatotoxic metabolite (highly reactive methylating agent) in the liver.

cycads also contain another neurotoxin, the non-protein amino acid L-β-methylaminoalanine (BMAA), but this is not considered to be involved in the pathogenesis of the posterior ataxia syndrome (Duncan *et al.* 1992) [See also the section on neurolathyrism; *Lathyrus* spp.]

Sources:

All cycads are listed in CITES (the Convention on International Trade of Endangered Species) (Norstog & Nicholls 1997). Many species in Australia are rare and endangered (Jones 1993; Hill & Osborne 2001, Jones 2002). A world list of cycads is given by Stevenson *et al.* (1995).

All cycad species can reasonably be suspected of being potentially toxic to domestic animals and humans. See also MAM for hepatotoxicity.

Australia

The cycad genera in Australia are *Bowenia, Cycas, Macrozamia* and *Lepidozamia* (zamia, zamia palm [*sic*], burrawang) [DM41; Hill 1995,1998; Jones 1993, 2002; Hill & Osborne 2001].

Family Cycadaceae

***Cycas* spp.** (tropics - Q, NT, WA) [DM43]

27 *Cycas* spp. in Australia, *ca.* 50 worldwide (Hill 1995, 1998; Jones 1993; Hill & Osborne 2001)

Poisonings causing neurotoxicity in cattle have involved:

Northern Territory (Top End)

Cycas armstrongii Miq. (zamia, tree zamia, zamia palm, cycad palm) – distributed on Melville Island and in the Top End, NT, from the Adelaide River west to the Finniss River and south to the township of Adelaide River with sporadic occurrences further south towards Hayes Creek - posterior ataxia was induced in cattle fed 45 kg fresh leaves at 0.5-1.5 kg/head/day; other cattle were unaffected after eating 80 kg (Wesley-Smith 1973); signs were induced in 3 steers each fed 27 kg wet weight (4 kg dry weight) of fresh leaf over 50 days and spinal cords had white matter degeneration (Hooper *et al.* 1974).

Cycas angulata R.Br. (zamia, zamia palm) – distributed along the lower reaches of the Wearyan, Foelsche and Robinson Rivers, near Borroloola, NT, and on the Bountiful Island Group in the Gulf of Carpentaria, Qld. - probably responsible for poisoning of cattle in eastern Arnhem Land in 1977 (Hooper 1978; John Maconochie in a personal communication to S.L. Everist 1978)

Queensland (Tropical coast and hinterland)

Cycas media R.Br. [= *C. kennedyana* F.Muell. , *C. normanbyana* F.Muell.] (zamia, zamia palm, tree zamia) - distributed from south of Mackay to coastal Cape York Peninsula

Note on toxicity of *C. media*: The plants associated with field cases in cattle in Queensland and used for feeding experiments there have all been referred in the literature to *C. media*, but reclassification of the plants in this taxon by Hill (1996) means that plants in the newly-recognised species *C. megacarpa* and *C. ophiolitica* as well as *C. media* in the currently-accepted sense and hybrids between these taxa have probably all been involved.

Cycas megacarpa K.D.Hill † - distributed from near Mt.Morgan south to near Goomeri, Qld; has been cultivated under the name *C. kennedyana* [*sic*] which name more correctly refers to *C. media.*

Cycas ophiolitica K.D.Hill † - distributed between Marlborough and Rockhampton, Qld; cultivated as *Cycas* 'Marlborough Blue' and as *C. normanbyana* [*sic*] which name more correctly refers to *C. media.*

Western Australia (Kimberley region)

Cycas basaltica C.A.Gardner - distributed widely in the northern Kimberley Region, WA, from offshore islands west of Port Warrender to near Kalumburu

Cycas furfuracea W.Fitzg. - distributed in the Kimberley Region, WA, in the King Leopold Ranges, at Kimbolton and on islands to the north including Heywood Island

Cycas lane-poolei C.A.Gardner - distributed in scattered populations in the north-western Kimberley region

Cycas pruinosa Maconochie - distributed widely in the eastern and southern Kimberley Region, WA, and in the Spirit Hills on "Bullo River" Station, NT

Note on zamia staggers in the Kimberley region of WA: Gardner & Bennetts (1956) record the occurrence of the disease in the region, attributing it to *C. media* or *C. angulata* and suggesting that *C. lane-poolei* was probably also toxic. Currently, the 4 species listed above are recorded from the region (Hill 1998). Everist (1981) cited a personal communication from John Maconochie (1978) linking the *C. media* referred to by Gardner & Bennetts (1956) to *C. basaltica.*

Other species occurring naturally in Australia (from Hill 1998), but for which no published toxicity records are available, are listed below. Some species have a restricted distribution, are regarded as rare and endangered, or both and are indicated by the symbol †:

Cycas arenicola K.D.Hill - distributed along the upper reaches of the East Alligator and Liverpool Rivers, Top End, NT

Cycas arnhemica K.D.Hill - occurs in three populations in the Top End, NT, on Groote Eylandt (ssp. *muninga*) and in Arnhem Land on the lower Blyth River (ssp. *natja*) and on the upper Goyder River (ssp. *arnhemica*)

Cycas badensis K.D.Hill † - occurs only on Moa and Badu Islands, Torres Strait

Cycas brunnea K.D.Hill - occurs at he headwaters of Lawn Hill Creek, north-western Qld and on "Wollogorang" Station, north-eastern NT

Cycas cairnsiana F.Muell. † - distributed in the Newcastle Range region of north-eastern Qld; cultivated as *Cycas* 'Mount Surprise' or *Cycas* 'Champion's Blue Surprise'

Cycas calcicola Maconochie - distributed in the Daly River basin, Top End, NT, and north along the Finniss Range from Katherine in the south-east to north of Litchfield Park and south towards the Daly River Police Station

Cycas canalis K.D.Hill - distributed in the Top End, NT, with populations in near-coastal sites at Channel Point, north of the mouth of the Daly River, and inland from west of Dorisvale to around the Douglas Daly Reserve.

Cycas conferta Chirgwin - distributed in the Top End, NT, from Kakadu National Park (Twin Falls and Goodparla Homestead) to west of Pine Creek

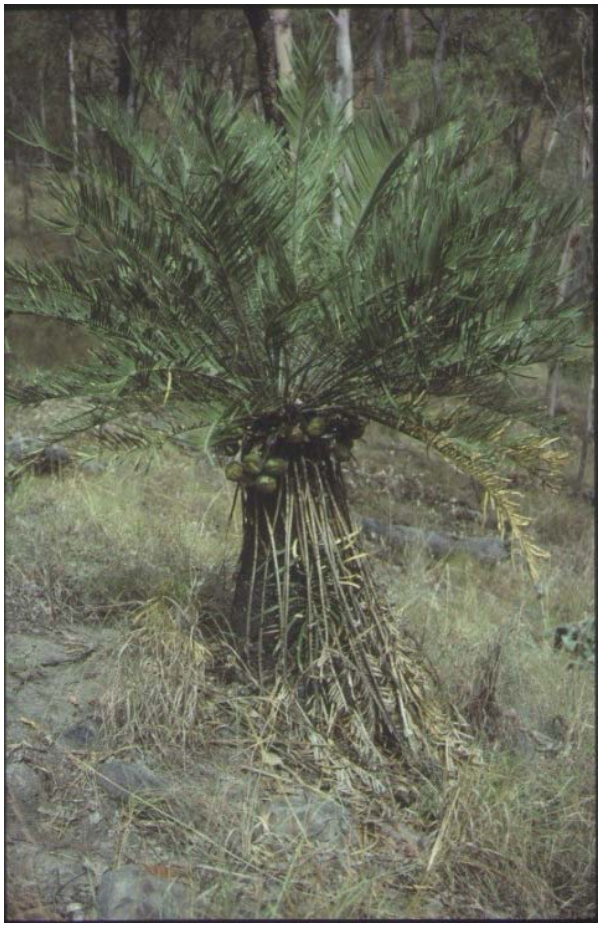
Cycas couttsiana K.D.Hill - distributed in the southern Gregory Range, northern Qld; cultivated as *Cycas* 'Glen Idle Blue'

Cycas cupida P.I.Forster † - restricted to one population in an area of 60 km^2 in the Terrace Range, south of Charters Towers, Qld; a blue-leaved species (Forster 2001)

Cycas desolata P.I.Forster † - restricted to two populations near Charters Towers, Qld
Cycas maconochiei Chirgwin & K.D.Hill - distributed in the Top End, NT, with populations in the north-west, on the Cox Peninsula (west of Darwin) , and towards the coast south of the Daly River
Cycas orientis K.D.Hill - distributed in eastern Arnhem Land, NT
Cycas platyphylla K.D.Hill - distributed in the Petford district, north-western Atherton Tableland and 250km south on "Wandovale" Station
Cycas semota K.D.Hill † - restricted to the Bamaga district, far northern Cape York Peninsula, Qld
Cycas silvestris K.D.Hill † - northern Cape York Peninsula, Qld
Cycas tuckeri K.D.Hill † - central Cape York Peninsula, Qld
Cycas xipholepis K.D.Hill - distributed widely in central Cape York Peninsula from south of Coen to Batavia Downs, west to Merapah, Qld
Cycas yorkiana K.D.Hill - northern Cape York Peninsula, Qld

Hybrid or intergrade populations of *Cycas* occur (Hill & Osborne 2001):
Cycas armstrongii x *C. conferta* - Top End, NT
Cycas armstrongii x *C. maconochiei* - Top End, NT
Cycas calcicola x *C. conferta* - Top End, NT
Cycas arnhemica x *C. orientis* - Top End, NT
Cycas basaltica x *C. lane-poolei* - southern Mitchell Plateau, Kimberley Region, WA
Cycas media x *C. platyphylla* - in the Irvinebank-Ravenshoe-Mt.Garnet district on the western Atherton Tableland, Qld
Cycas media x *C. ophiolitica* - central coastal Qld
Cycas megacarpa x *C. ophiolitica* - central coastal Qld

Other *Cycas* spp. cultivated in gardens in Australia include:
**Cycas revoluta* Thunb. (sago "palm") from the southern Japanese islands is commonly cultivated in gardens and as tub specimens
**Cycas circinalis* L. from the Pacific Islands and southern Asia
**Cycas rumphii* Miq. from the Pacific Islands
**Cycas thouarsii* R.Br. ex Gaudich. from eastern Africa



Cycas megacarpa female plant. Note hanging sporophylls with seeds [RAM Photo]

Leaf of *Cycas* sp. Note mid-ribs in pinnae (leaflets). [RAM Photo]

Fruiting female plant of *Cycas angulata* [RAM Photo]

Family Zamiaceae

***Macrozamia* spp.** (subtropics/temperate - Q, NSW, WA) [DM44-47]

38 *Macrozamia* spp., all in Australia (Hill 1995, 1998; Jones 1993, 2002; Hill & Osborne 2001)

Species recorded as toxic under natural conditions or in feeding trials include:

Macrozamia communis L.A.S. Johnson (burrawang, burrawan, zamia, wild pineapple) - distributed in coastal and sub-coastal NSW from Taree to Bega

Macrozamia heteromera C.Moore (burrawang, zamia, wild pineapple) - occurs in the Warrumbungle Range and areas to its south-west, NSW *Macrozamia heteromera* (seeds): 2200 of 6000 sheep died after exposure to cycads with disintegrating female cones in the Pilliga Scrub in May 1929. These sheep were accustomed to eating a compressed feed supplement ("sheep nuts") and were thus believed conditioned to eat cycad seeds. Lesions: acute periacinar hepatocyte necrosis. (Seddon *et al.* 1931) This incident could have been associated with other species of similar form recorded from the Pilliga Scrub including *M. polymorpha* and *M. glaucophylla*.

Macrozamia lucida L.A.S.Johnson (zamia, wild pineapple) - distributed in southern Qld from Brisbane to Maleny with a disjunct population in far northern NSW; cultivated – leaves toxic in experiments by Hall (1957) and Hall & McGavin (1968)

Macrozamia miquelii (F.Muell.) A.DC. (zamia, zamia palm [*sic*], wild pineapple) - disjunct populations occur in southern Qld from near Marlborough south to near Brisbane - leaves toxic in experiments by Hall (1957)

Macrozamia moorei F.Muell. (zamia, zamia palm [*sic*]) - distributed in central Qld from north of Springsure to the Carnarvon Range; cultivated

Macrozamia pauli-guilielmi W.Hill & F.Muell. (zamia, zamia palm [*sic*], wild pineapple, pineapple zamia) - occurs in the Wide Bay district of southern Qld from eat of Gympie to south of Childers – both posterior ataxia and liver damage produced experimentally with leaves by Hall (1957)

Macrozamia riedlei (Fisch. Ex Gaudich) C.A.Gardner (zamia, zamia palm [*sic*]) - distributed from Perth to Albany and west to the coast (WA).

Gardner & Bennetts (1956) cite Edwards HH (1894) *J. Bureau Agric. West. Aust.* **1**:225 as reporting reproduction of the ataxic syndrome in calves fed young leaves of *M. riedlei* at 1.8 kg (4 lb) daily for 11 days or 2.7 kg (6 lb) daily for 7 days – a very rapid onset suggesting that the result should be accepted cautiously. In contrast, Bennetts & Filmer in 1926 (Bennetts HW 1934-35 *J. Roy. Soc. West. Aust.* **21**:xxi) failed to induce toxicity by feeding an 8 month-old steer with young leaf of *M. riedlei* at 0.9 kg (2 lb) daily for 28 days followed immediately by 1.8 Kg (4 lb) daily for 28 days, but poisoned another steer with seeds (dose not cited by Gardner & Bennetts 1956). Gardner & Bennetts (1956) cite unpublished departmental records attributed to JM Armstrong (1955) as reporting reproduction of an ataxia in a sheep fed 21.8 kg (48 lb) of young *M. riedlei* leaves in 13 weeks; posterior ataxia was first noted at 6 weeks, progressing to recumbency with posterior paralysis at 10 weeks. Histological examination of spinal cord revealed lesions described as demyelination.

Other species occurring naturally in Australia (from Hill 1998), but for which no published toxicity records are available are listed below. Some species have a restricted distribution, are regarded as rare and endangered, or both and are indicated by the symbol †:

Macrozamia cardiacensis P.I.Forst. & D.L.Jones † - occurs on very steep slopes (including "Cardiac Hill") in Mt.Walsh National Park, south-eastern Qld

Macrozamia concinna D.L.Jones - distributed from the upper Hunter Valley north to the hills and ranges around the Liverpool Plains, NSW

Macrozamia conferta D.L.Jones & P.I.Forst. † - occurs in the Herries and other ranges to the south-east of Warwick in southern Qld.

Macrozamia cranei D.L.Jones & P.I.Forst. † - occurs in the Texas area, southern Qld

Macrozamia crassifolia P.I.Forst. & D.L.Jones † - occurs near Eidsvold and Mundubbera in the Burnett district, southern Qld

Macrozamia diplomera (F.Muell.) L.A.S.Johnson (burrawang, zamia, wild pineapple) - distributed on southern parts of the north-west slopes from the eastern Warrumbungle Range east to the Mooki River

Macrozamia douglasii W.Hill ex F.M.Bailey - occurs in the Cooloola section and on Fraser Island in the Great Sandy National Park, Qld

Macrozamia dyeri (F.Muell.) C.A.Gardner - distributed in coastal shrublands around Esperence in southern WA from Munglinup River eastwards to Israelite Bay. This species is included within *M. riedlei* by Gardner & Bennetts (1956), making positive identification of this species as toxic uncertain

Macrozamia elegans K.D.Hill & D.L.Jones † - occurs north and west of Richmond in the lower Blue Mountains, NSW

Macrozamia fawcettii C.Moore - distributed along the far north coast and ranges, NSW, from Richmond River to Coffs Harbour

Macrozamia fearnsidei D.L.Jones † - occurs in the Expedition mountain ranges north and east of Injune, central Qld

Macrozamia flexuosa C.Moore [=*M. pauli-guilielmi* ssp. *flexuosa* (C.Moore) L.A.S.Johnson] - occurs from Buladelah to Lake Macquarie (southern North Coast to extreme northern Central Coast), NSW

Macrozamia fraseri Miq. - distributed from Eneabba south to the Swan River, WA. This species is included within *M. riedlei* by Gardner & Bennetts (1956), making positive identification of this species as toxic uncertain. Hybrids (intergrades) with *M. riedlei* occur.

Macrozamia glaucophylla D.L.Jones [= *Macrozamia* 'Northern Pilliga'] - distributed from the northern Piliga Scrub to around Narrabri, NSW

Macrozamia humilis D.L.Jones † - occurs in a single population near Inverell, NSW

Macrozamia johnsonii D.L.Jones & K.D.Hill † - occurs near Dalmorton in north-eastern NSW, usually on steep slopes in forest

Macrozamia lomandroides D.L.Jones † - occurs in a restricted area south of Bundaberg and east of Childers, Qld

Macrozamia longispina P.I.Forst. & D.L.Jones † - distributed in south-eastern Qld on the coastal range west of Gympie from Widgee Mountain south through Manumbar

Macrozamia macdonnellii (F.Muell. ex Miq.) A.DC. † - occurs in the MacDonnell Ranges of central Australia (NT)

Macrozamia montana K.D.Hill - occurs on very steep ridges in ranges or south-eastern escarpment of the northern tablelands of NSW between the catchments of the Macleay and Manning Rivers

Macrozamia mountperriensis F.M.Bailey † - distributed in south-eastern Qld from Gin Gin to Mount Perry and to south of Brooweena

Macrozamia occidua D.L.Jones & P.I.Forst. † - occurs in Sundown National Park, southern Qld

Macrozamia parcifolia P.I.Forst. & D.L.Jones † - occurs near Biggenden in the Wide Bay district, southern Qld

Macrozamia platyrachis F.M.Bailey † - occurs on Blackdown Tableland and the Expedition Range, central Qld

Macrozamia pleurinervia (L.A.S.Johnson) D.L.Jones † [=*M. pauli-guilielmi* ssp. *pleurinervia* L.A.S.Johnson; = *M. machinii*] - distributed in northern NSW between Inverell and Tenterfield

Macrozamia polymorpha D.L.Jones † [= *Macrozamia* 'Southern Pilliga'] - distributed from Coonabarabran north to the southern Pilliga Scrub, NSW

Macrozamia reducta K.D.Hill & D.L.Jones - widespread in central eastern NSW from western suburbs of Newcastle west towards Mudgee and south to Glen Davis; cultivated as "dwarf communis"

Macrozamia secunda (Benth.) C.Moore - distributed from Gilgandra to Grenfell and east to Mudgee and Capertee on the central western slopes to the lower central tablelands, NSW

Macrozamia spiralis (Salisb.) Miq. (burrawang, zamia, wild pineapple) - distributed in central coastal NSW from the Goulburn River Valley to the lower eastern Blue Mountains and the Sydney-Campbelltown districts

Macrozamia stenomera L.A.S.Johnson - occurs in and around the Nandewar and Moonbi Ranges, north-western slopes and western northern tablelands, NSW

Macrozamia viridis D.L.Jones & P.I.Forst. † - restricted to the Wyberba and Girraween districts in southern Qld

Hybrids or intergrades between *Macrozamia* species occur (Hill & Osborne 2001):

Macrozamia diplomera x *M. polymorpha* - near Coonabarabran, NSW

Macrozamia fearnsidei x *M. moorei* - central Qld

Macrozamia flexuosa x *M. reducta* - near Cessnock, NSW

Macrozamia fraseri x *M. riedlei* - south-western WA

Macrozamia lomandroides x *M. pauli-guilielmi* - Wide Bay district, Qld

Macrozamia lucida x *M. miquelii* - Brisbane area, Qld

Macrozamia reducta x *M. secunda* - near Mudgee, NSW