SIMPLE ORGANIC TOXINS

Fluoroacetate

Core data

Common sources:
- Compound 1080 vertebrate pesticide
- *Gastrolobium* spp. (poison bushes)
- *Acacia georginae* (Georgina gidyea)

Animals affected: dogs, ruminants, horses, pigs
Poisoning circumstances:
- 1080 vertebrate pesticide
- plants highly palatable & readily eaten
- forced exercise precipitates deaths in ruminants
- secondary poisoning of dogs scavenging carcases

Main effects:
- blocks citric acid cycle
- carnivores, pigs:
  - CNS + alimentary tract hyperactivity predominate
- herbivores:
  - heart failure predominates
  - myocardial scarring in survivors

Diagnosis:
- assay stomach contents
- myocardial histopathology

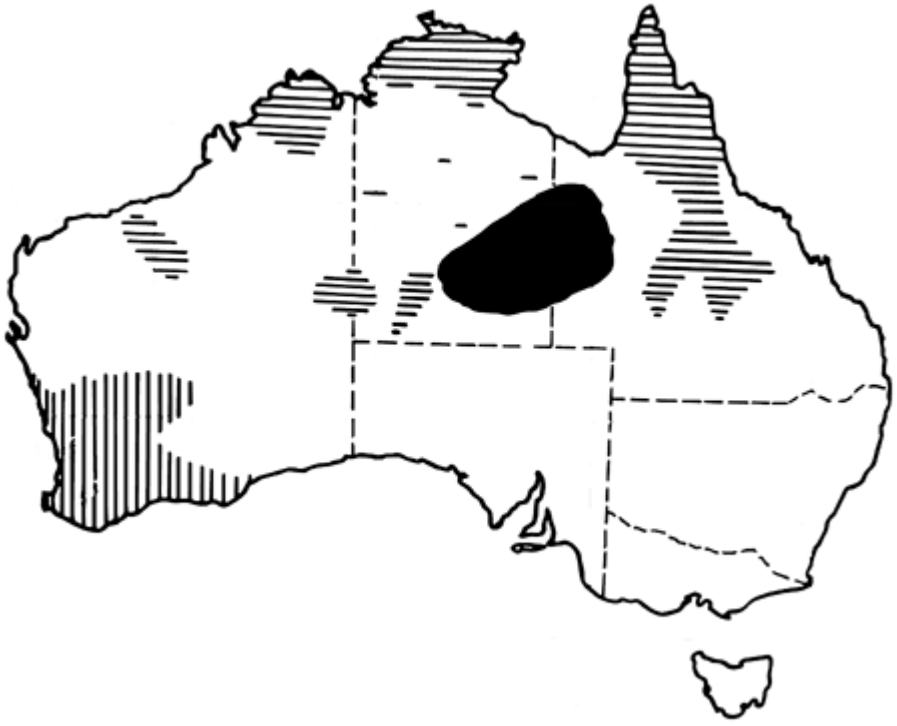
Therapy: dogs:
- prognosis grave
- anaesthesia + acetate ions

Prevention:
- genetically-engineered rumen bacterium detoxification (under development)
- muzzle dogs potentially exposed to 1080 baits

Chemical structure:

CFH_2COO^-
Fluoroacetate ion

Sources:
- **vertebrate pesticides**
  - **sodium monofluoroacetate** = compound **1080** (used, for example, against dingos, feral pigs, rabbits in Australia; brush-tailed possums in New Zealand). For a discussion of baits for rabbits, see Allen & Fisher (1999)
  - fluoroacetamide = compound 1081 = FCH_2CONH_2
  - related vertebrate pesticide gliftor = 1,3-difluoro-2-propanol (DFP)
- **plant sources** are all shrubs or trees (Hall 1972, McEwan 1964a,b, 1978)



Fluoroacetate-containing plants' distribution in Australia. Solid area: *Acacia georginae.* Vertical bars: 34 *Gastrolobium* spp. + *Nemcia spathulata*. Horizontal bars: *Gastrolobium grandiflorum* [modified after Twigg & King 1991]

Features present in toxic *Gastrolobium* species and which may be useful for differentiation of these from non-toxic species (Gardner & Bennetts 1956) include

- flowers in many-flowered terminal racemes, sometimes with axillary racemes (raceme = inflorescence with a single unbranched elongated stem carrying flowers on individual stalks of equal length; terminal = at the end of branches; axil = angle between stem and leaf)
- flower buds subtended by a boat-shaped bract which falls when the flower opens
- leaves in opposite pairs or in whorls of 3, 4 or 5
- stipules present at the leaf base or leaf stalk in young leaves, persisting in some species

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- Western Australia (most in the south west)

Family Fabaceae

***Gastrolobium* spp.** (some species were formerly classified as *Oxylobium* spp.)

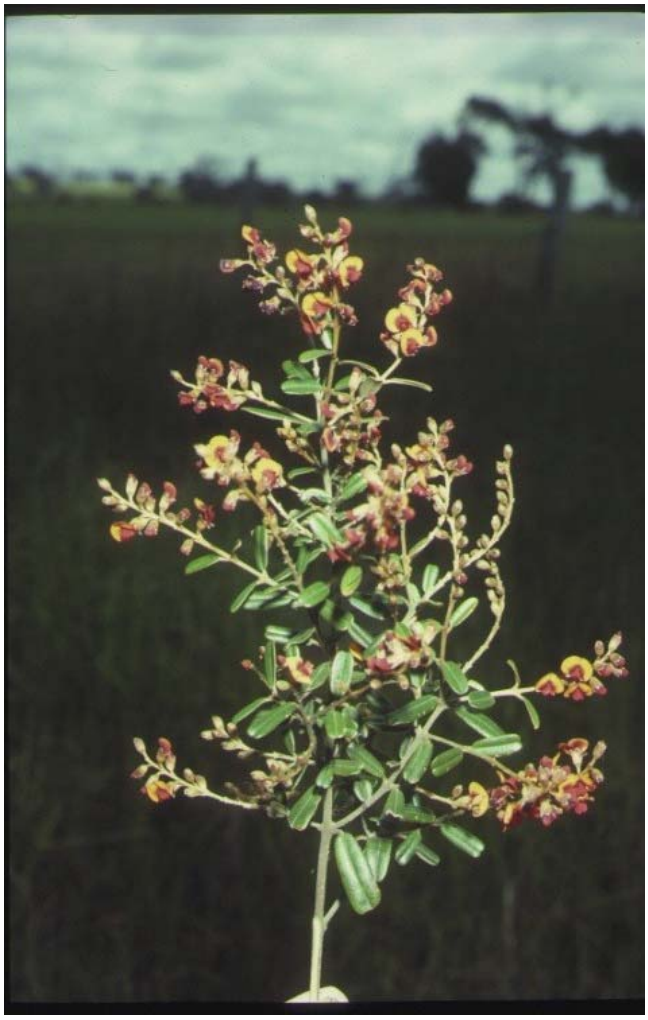
There are 50 *Gastrolobium* species in WA, all endemic to Australia (Paczkowska & Chapman 2000); 34 species are toxic or suspected to be so [Aplin 1971] and 14 of these are endangered species (†) (Sampson & Hopper 1989)

Gastrolobium appressum (scale-leaf poison) †
Gastrolobium bennettsianum (cluster poison)
Gastrolobium bilobum (heart-leaf poison)
Gastrolobium callistachys (rock poison) †
Gastrolobium calycinum (York Road poison)
Gastrolobium crassifolium (thick-leaf poison)
Gastrolobium densifolium (mallet poison) †
Gastrolobium floribundum (wodjil poison)
Gastrolobium forrestii (river poison)
Gastrolobium glaucum (spike poison) †

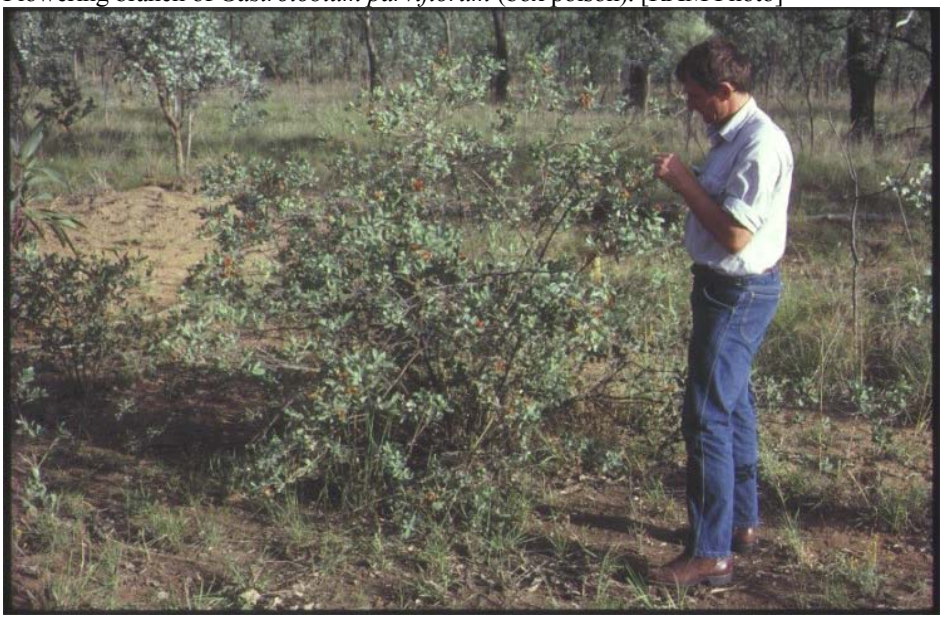
Gastrolobium grandiflorum (wall-flower poison)
Gastrolobium graniticum (formerly *Oxylobium graniticum*) (granite poison) †
Gastrolobium hamulosum (hook-point poison) †
Gastrolobium heterophyllum (formerly *Oxylobium heterophyllum*) (slender poison) †
Gastrolobium laytonii (kite-leaf poison, breelya)
Gastrolobium microcarpum (sandplain poison)
Gastrolobium ovalifolium (runner poison) †
Gastrolobium oxyloboides (Champion Bay poison)
Gastrolobium parviflorum (formerly *Oxylobium parviflorum*) (box poison)
Gastrolobium parvifolium (berry poison)
Gastrolobium polystachyum (horned poison, Hill River poison)
Gastrolobium propinquum (Hutt River poison) †
Gastrolobium pyncnostachyum (round-leaf poison)
Gastrolobium racemosum (formerly *Oxylobium racemosum*) (net-leaf poison) †
Gastrolobium rigidum (formerly *Oxylobium rigidum*) (rigid-leaf poison) †
Gastrolobium rotundifolium (Gilbernine poison) †
Gastrolobium spectabile (formerly *Oxylobium spectabile*) (Roe's poison)
Gastrolobium spinosum (prickly poison)
Gastrolobium stenophyllum (narrow-leaf poison) †
Gastrolobium tetragonophyllum (formerly *Oxylobium tetragonophyllum*) (brother-brother)
Gastrolobium tomentosum (woolly poison) †
Gastrolobium trilobum (bullock poison)
Gastrolobium velutinum (Stirling Range poison)
Gastrolobium villosum (crinkle-leaf poison)

- *Nemcia spathulata* (formerly *Gastrolobium spathulatum*) (Family Fabaceae) [Twigg *et al.* 1996a,b]

Flowering shrubs of *Gastrolobium parviflorum* (box poison) in natural habitat. [RAM Photo]



Flowering branch of *Gastrolobium parviflorum* (box poison). [RAM Photo]



Whole plant of *Gastrolobium grandiflorum* (heart-leaf poison bush) in natural habitat. [RAM Photo]

Flowering branch of *Gastrolobium grandiflorum* (heart-leaf poison bush). [RAM Photo]

- Western Australia & Northern Territory
  - *Gastrolobium brevipes* (Twigg *et al.* 1999)
- Queensland & Northern Territory
  - ***Gastrolobium grandiflorum*** (desert poison bush, heart-leaf poison bush) [DM123]
  - ***Acacia georginae*** (Georgina gidyea) (Family Mimosaceae) [DM146]

- Africa
  - *Dichapetalum* spp. (gifblaar [Afrikaans, =poison leaf]) – Family Dichapetalaceae; 124 species total (Mabberley 1997); 12 species toxic (Vickery & Vickery 1973); fluoroacetate identified as toxic principle by Marais (1944)

Dichapetalum braunii	Tanzania
Dichapetalum cymosum [=*Dichapetalum venenatum*]	South Africa, Namibia, Zimbabwe, Botswana, Angola
Dichapetalum deflexum	Mozambique, Tanzania
Dichapetalum guineense	Sierra Leone, Ivory Coast, Ghana, Togo, Dahomey, Nigeria
Dichapetalum heudelotti	Guinea, Sierra Leone
Dichapetalum macrocarpum	Tanzania, Mozambique
Dichapetalum michelsonii	Rwanda, Burundi, Zaire
Dichapetalum mossambicense	Tanzania, Mozambique
Dichapetalum ruhlandii	Kenya, Tanzania
Dichapetalum stuhlmanii	Tanzania
Dichapetalum tomentosum	Nigeria, Cameroons, Zaire, Congo
Dichapetalum toxicarium	Guinea, Sierra Leone, Liberia, Ivory Coast, Ghana

  2 species in Australia are believed to be non-toxic: *Dichapetalum papuanum* occurs in northern Queensland, Papua-New Guinea, Solomon Islands – negative for fluoroacetate (Queensland Herbarium voucher AQ180162); *Dichapetalum timorense* occurs in Arnhem Land, Papua-New Guinea, Irian Jaya, Sumatra – there are no records of toxicity
  - *Spondianthus preussi* (Kamgue *et al.* 1979) (Family Euphorbiaceae; monotypic genus)
- South America
  - *Palicourea* spp. (Family Rubiaceae) – 200+ species (Mabberley 1997); 3 species toxic (Tokarnia *et al.* 1979, De Oliveira 1963)

Palicourea grandiflora

Palicourea juruana
Palicourea marcgravii

Toxicologically insignificant amounts of fluoroacetate have been detected in tea (*Camelia sinensis*) and guar gum (*Cyamopsis tetragonolobus*) (Vartianen & Kauranen 1984, Twigg *et al.* 1996b) and in other plants exposed to artificially high soil fluoride concentrations (Lovelace *et al.* 1968) suggesting that the capacity to synthesise fluoroacetate is widespread in plants.

Toxicity:
- *Gastrolobium* spp.
  – leaves, flowers & pods toxic
  - variation in fluoroacetate concentrations occurs with species, plant part (young leaves, flowers, fruit > mature leaves, stems) and season of the year (Twigg *et al.* 1996b)
  - fluoroacetate concentration in 38% of 34 WA species tested was > 400 mg/kg; 68% of species had concentration > 100 mg/kg (Aplin 1971)
  - fluoroacetate concentrations in *G. spinosum, G. villosum, G. bilobum* and *G. calycinum* ranged from 0.7 to 3875 mg/kg; young leaves & flowers 73-3875 mg/kg (Twigg *et al.* 1996b); seeds can contain >6000 mg/kg (Twigg 1994)
  - *G. bilobum* (heart-leaf poison [WA]) and *G. parviflorum* (box poison) leaf can contain up to 2600 mg fluoroacetate/kg; seed >6500 mg/kg
  - *G. grandiflorum* leaf can contain up to 185 mg/kg (Aplin 1971)
  - *G. brevipes* from Northern Territory: young leaf 17-99 mg/kg, young pods 56-301 mg/kg (Twigg *et al.* 1999)
- *Nemcia spathulata* – 2 leaf samples contained 40 & 80 mg/kg (Twigg *et al.* 1996a)
- *Acacia georginae* (Bailey 1896, Bell *et al.* 1955, Barnes 1958, Oelrichs & McEwan 1961, Murray *et al.* 1961, Whittem & Murray 1963)
  - pods & young leaves toxic (variation between plants); leaf can contain up to 25 mg/kg (Oelrichs & McEwan 1961)
  - dry season (Jul-Nov)/drought → mortalities
- *Dichapetalum* spp.
  - *Dichapetalum braunii* can contain up to 8000 mg/kg (Emsley 1992)
  - some antelopes (eland, kudu) appear to be more resistant than goats to *Dichapetalum cymosum*: goat, springbok, gemsbok LD_{100} = 1.01-1.60 mg fluoroacetate/kg; eland, kudu LD_{100} = 6-8 mg/kg (Basson *et al.* 1982)
- animal susceptibility (LD_{50} mg/kg)
  - dogs 0.05 [lethal dose 0.05-1.0 mg/kg]
  - cattle, sheep, horses, pigs 0.2-4.0
  - poultry 10-30

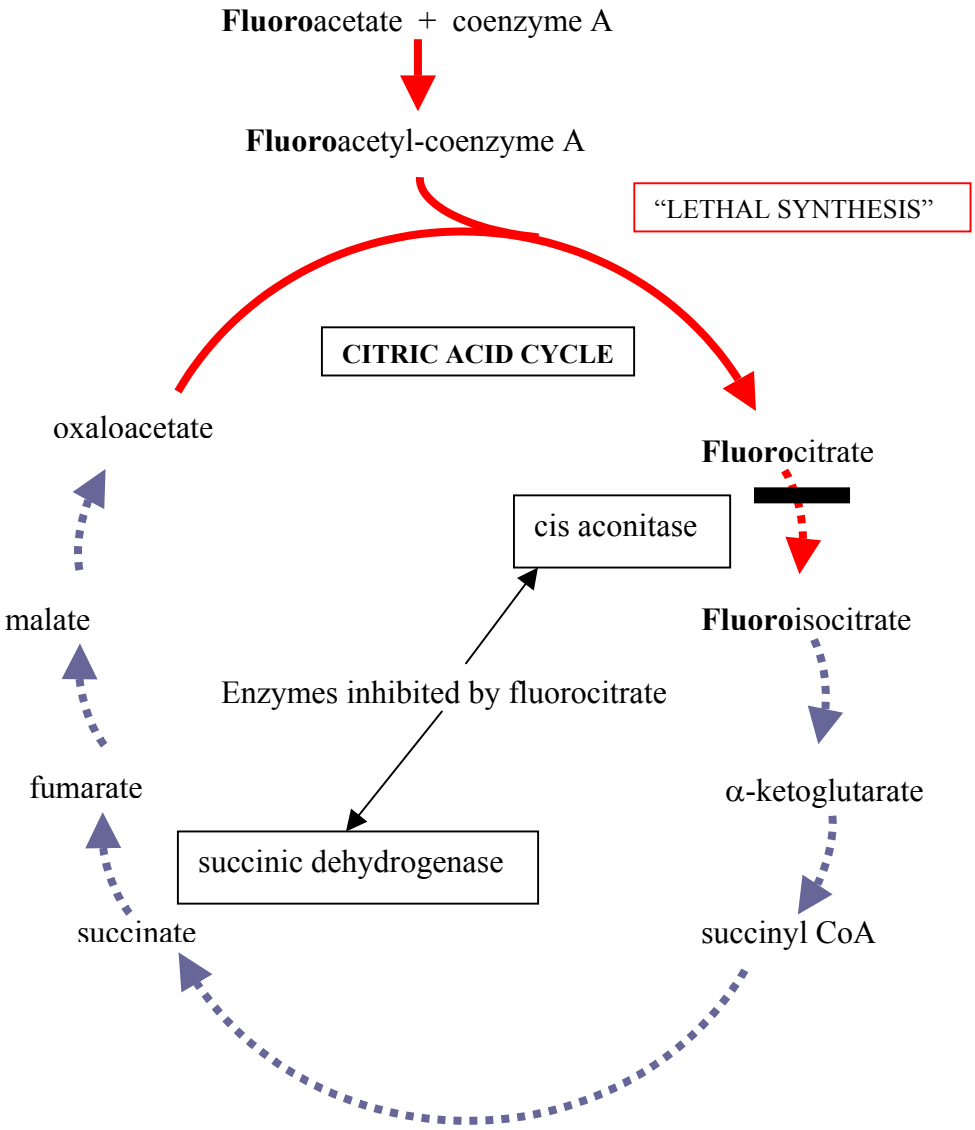
Data on susceptibility of Australian animals (mammals, birds, reptiles, amphibians) to 1080 are published in a series of papers from CSIRO Division of Wildlife and Rangelands Research (McIlroy 1981a,b, 1982a,b, 1983a,b, 1984, McIlroy *et al.* 1985, McIlroy 1986, McIlroy *et al.* 1986)

- evidence of **adaptation in Australian native animals** to fluoroacetate (hepatic detoxication) (Twigg & King 1991)-

Species	LD_{50} mg/kg Western Australia	LD_{50} mg/kg Eastern Australia
Antechinus flavipes (a small marsupial carnivore)	12.5	3.5
Rattus sordidus (a native rat)	20	0.3
Rattus fuscipes (a native rat)	20-80	1.1
Macropus eugenii	20	0.3
Macropus fuliginosus (western grey kangaroo)	40	Does not occur
Trichosurus vulpecula (brush-tailed possum)	125	0.75

Mode of action:

- fluoroacetate is converted to fluoroacetyl-CoA → fluorocitrate → **irreversible blockage of the citric acid cycle enzymes** cis-aconitase and succinic dehydrogenase → accumulation of citrate in cells, inhibition of glucose metabolism, failure of cellular respiration.



Conditions of poisoning:

- plants containing toxic amounts of fluoroacetate are **palatable** as well as being highly toxic, making them very hazardous for domestic herbivores
- *Gastrolobium* spp. are most toxic when carrying flowers or pods (seeds) or young leaf growth
- *Acacia georginae* is most toxic when carrying pods (seeds) or young leaf growth
- forced exercise and drinking precipitate deaths of cattle or sheep from *Acacia georginae*
- dogs and cats eating native marsupial and bird species that have fed on *Gastrolobium* spp. plants without ill effect including ingestion of viscera containing leaves and seeds (Gardner & Bennetts 1956) and bones of bronze-wing pigeons previously cooked as food for humans (Drummond 1842 cited by Marchant 1994).
- poisoned animals may be a source of **secondary poisoning of dogs** scavenging pig, rabbit, possum or other carcases poisoned by 1080 (Meenken & Booth 1997) or sheep poisoned by *Gastrolobium* spp. plants (Drummond 1842 cited by Marchant 1994)

- baits laid for feral vertebrate control (e.g. rabbits) may be eaten by herbivores if allowed access (Allen & Fisher 1999)
- concentrations of fluoroacetate from 1080 baits toxic to scavengers (e.g. foxes, dogs) may persist in target pest carcases for >75 days (Eason *et al.* 1998) and significantly affect scavenger populations (McIlroy & Gifford 1991)
- fluoroacetate is leached from most poisoned baits (pellets, carrot, but not oats) significantly by rainfall >5mm, reducing their toxicity (Allen & Fisher 1999); plants that take up fluoroacetate from leached baits do not contain sufficient toxin to be hazardous to grazing or browsing herbivores (Ogilvie *et al.* 1998); fluoroacetate leached into soils is degraded rapidly by soil microbes (King *et al.* 1994); microbes in bait material itself may contribute significantly to degradation of fluoroacetate by defluorination (Wong *et al.* 1991); Under New Zealand conditions, livestock and dogs are recommended to be excluded from baited areas for 8 weeks or until 100 mm of rain has fallen, but deaths have been recorded in both livestock and dogs well beyond this time, up to 20 weeks for sheep and 10 weeks for dogs (Orr & Bentley 1994).

Clinical signs:

- **carnivores, pigs → CNS** disturbance, **GI hyperactivity** dominate the toxicity syndrome
  - hyperactive, vomit, froth at the mouth, urinate, defaecate repeatedly
  - dyspnoea + frothing at mouth & nostrils
  - dogs: periodical wild running, hysteria, barking
  - intermittent tonic/clonic convulsions, opisthotonus, paddling
- **herbivores → heart failure** dominates the toxicity syndrome
  - tachycardia, arrhythmia, polypnoea
  - rapid collapse, terminal convulsions, death
  - hypersensitivity, hyperactive
  - depression, muscle weakness

Pathology:

- hyperglycaemia, acidosis
- increased citrate concentrations in blood, kidney
- multifocal myocardial degeneration and necrosis in sheep (Schultz *et al.* 1982)
- myocardial scarring in survivors of *Acacia georginae* poisoning (Whittem & Murray 1963)

Diagnosis:

- **assay** stomach/rumen contents, liver, kidney, plants, suspected baits for fluoroacetate
  - modified HPLC method developed in South Africa (Minnaar *et al.* 2000a,b)
  - recommended sampling protocol from necropsies (Minnaar *et al.* 2000): collect 100g each of liver & kidney & stomach/rumen contents into separate scrupulously-clean glass or plastic containers as soon after death as possible and freeze immediately. Submit to the laboratory frozen (to be assayed within 14 days of collection). If samples are not frozen, assay within 7 days of collection.
  - in rumen samples spiked with fluoroacetate and held at room temperature (10-27^{o}C), the concentration after 83 days was 85% of the starting concentration; spiked liver sample concentration decreased to 52% after 104 days. Further time lapse revealed no further decreases (Minnaar *et al.* 2000).
  - fluoroacetate assay available from Queensland Department of Natural Resources, Alan Fletcher Research Station, 27 Magazine Street (PO Box 36), Sherwood 4075; Phone 07 3375 0700; samples are tested in batches, usually monthly; cost about $70 per sample (2001)
- **botanical examination of rumen contents** for the presence of source plants
- **histopathology** of the myocardium (in ruminants). Lesions may be present if the animal survives more than 24 hrs.

The difficulty of field diagnosis of fluoroacetate poisoning due to the lack of unequivocal clinical signs and lesions may lead to over-diagnosis (misdiagnosis) of this intoxication in endemic areas. In the endemic area for *Gastrolobium granidflorum* toxicity in Queensland, introduction of vaccination of cattle against botulism virtually halved the number of deaths in cattle in vaccinated herds. All these deaths had been previously ascribed by owners to fluoroacetate poisoning (John Cuskelly, personal communication, 11 July 2002)

Therapy:

- prognosis usually grave

- principal of theoretical therapy: **provide acetate ions** to prevent conversion of fluoroacetate to fluorocitrate; obviously needs to be early to be effective
- livestock
  - no effective practical therapy has been demonstrated to date
  - acetamide (2.0-5.0 g/kg) can prevent experimental poisoning if given before or simultaneously with a dose of fluoroacetate-containing plant (South African work with *Dichapetalum cymosum* in sheep) (Egyed & Schultz 1986)
- dogs
  - **prognosis is always grave**
  - emesis or gastric lavage only if patient has not already vomited, no clinical signs have occurred and constant monitoring for seizures is maintained
  - activated charcoal PO – helpful if given very early in clinical course
  - **barbiturates** to effect to control seizures (Tourtellotte & Coon 1950); a 50:50 tiletamine-zolazepam combination (Zoletil®; zolazepam is a benzodiazepine, tiletamine is in the phencyclidine family) has been used successfully (P.A.Darvall, personal communication 13 June 2001)
  - oxygen, artificial respiration or both as indicated
  - monitor and treat for cardiac arrhythmias
  - administer various **"antidotes" to supply acetate ions** – efficacy doubtful (see box below)
  - 4-methylpyrazole (*q.v.*) may be useful, particularly against gliftor (DFP) (Feldwick *et al.* 1994)

Fluoroacetate (1080) Suggested Therapeutic Protocols, Dogs - ***prognosis is always grave***

Barbiturate anaesthesia (prolonged; 12-18 hrs commonly) plus **one** of the following 2 acetate ion donors (these "antidotes" are probably only effective if therapy commences very early in the clinical course)

- Monoacetin (glyceryl monoacetate) (Sigma Chemical Co.) - IM 0.1-0.5 mg/kg hourly for several hours (total 2-4 mg/kg or IV diluted (0.5-1.0%) [N.B. IV → danger of haemolysis] [Os293 says 0.55 g/kg IM hourly until total dose of 2-4 g/kg is reached. Probably a typographical error – g should read mg.] Has a short shelf life, not very effective against high doses of fluoroacetate [Beasley 101]
- Acetate ions in electrolyte solution [P54 electrolyte solution (Donawick formula)] - IV infusion @ 50-150 ml/kg)

Ethanol (50%) and acetic acid (5%), 8 ml/kg of each PO (supply acetate ions) [Os293] has been suggested, but is reported as ineffective in most cases and contraindicated with barbiturates as the combination produces prolonged anaesthesia (days) and a high incidence of pneumonia [Beasley 101]

Calcium gluconate (5% solution slowly IV in fluids @ 0.2-0.5 ml/kg) recommended to control tetany [Beasley 101] – probably less effective than barbiturates

Sodium bicarbonate (8.4% w/v Astra) - IV infusion @ 300 mg/kg over 15-30 minutes (clinically-developed protocol, limited data available, not experimentally confirmed)

- recent experimental work with rats indicates that the use of ion exchange resins PO, in particular colestipol or combinations of such materials, may reward further investigation (Norris *et al.* 2000)

Prevention & control:
- **muzzle dogs** potentially exposed to 1080 baits (cheap and effective)
- introduction of virally-vectored fertility control (immunocontraception) of vertebrate pests (e.g. rabbits) in Australia and elsewhere will, if successful, render the use of 1080 poisoned baits obsolete (Hinds *et al.* 1998)
- dyeing fluoroacetate-impregnated baits green or blue reduces their attractiveness to birds (and humans) (Allen & Fisher 1999)
- introduction of **genetically-engineered rumen bacteria** (*Butyrivibrio fibrisolvens*) with fluoroacetate-catabolising capacity into the ruminal flora is a possible future measure under development

(Gregg *et al.* 1994, 1998). These bacteria express fluoroacetate dehalogenase activity. If introduced, care will be required with environmental implications: the temptation to overstock land carrying fluoroacetate-containing plants must be avoided to ensure sustainable production. Currently, introduction is suspended/rejected because of concern that feral vertebrate targets (rabbits, goats) of 1080 may acquire resistance if the bacterium establishes in their alimentary tracts. Work is on-going.

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Se54, VM8/1519, Os289

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💣☑ *Oxalates (acute poisoning)*

Core data

Common source:

- *Oxalis pes-caprae* (soursob)
- *Portulaca* spp. (pigweed)
- *Setaria sphacelata* (setaria)
- + others

Animals affected: sheep, cattle
Poisoning circumstances:

- greatest soluble oxalate concentrations in lush growing leaf
- hungry animals
- rumen flora unadapted to oxalate intake

Main effects:

- hypocalcaemia
- nephrosis with calcium oxalate crystals in tubules

Diagnosis:

- clinical pathology
- kidney histopathology
- soluble oxalate assay on plants

Therapy: calcium borogluconate IV, SC (prognosis guarded)

Chemical structures:

- Oxalic acid (dicarboxylic acid)
- Water-Soluble oxalate ion or acid oxalate ion (usually with Na^+, K^+ or NH_4^+)
- Water-Insoluble calcium oxalate (crystals – raphides, prisms, druses)

Sources:

- **plants** containing hazardous amounts of **soluble oxalates** (> 2% in dry matter) are **numerous** [Everist (1981) lists species for Australia as indicated], important examples are given in bold

USA:

Family Chenopodiaceae

Halogeton glomeratus (halogeton, barilla) – the classic cause of acute oxalate poisoning in North America

Australia:

Family Aizoaceae (Prescott & Venning 1984)

Mesembryanthemum spp. (ice plants) - 74 species from North America, southern Europe, the Middle East & Africa; 3 species naturalised in Australia (Prescott & Venning 1984)

Mesembryanthemum nodiflorum L. (slender ice plant) - Jacob & Peet (1989) recorded cases in sheep in WA with up to 18% soluble oxalate

Mesembryanthemum crystallinum L. (ice plant) - sheep poisoned in WA; 7.6% soluble oxalate (JG Allen, personal communication 21 March 1997)

Tetragonia tetragonioides (Pallas) Kuntze (New Zealand spinach) (Ev918)

Trianthema spp. (Ev918); about 20 species in tropics and subtropics with 12 species (10 endemic, 1 pantropical, 1 naturalised) in northern and arid Australia (Prescott & Venning 1984)

Trianthema portulacastrum L. (black pigweed) (?) (Ev918)

Trianthema triquetra Willd. (Ev918)

Zaleya galericulata (Melville) H.Eichler [= *Trianthema galericulata* Melville] (?) (Ev918)

Family Amaranthaceae

Amaranthus mitchellii (Ev889)

Amaranthus retroflexus (Ev889)

Family Chenopodiaceae (Wilson 1984)

Atriplex spp. (saltbushes) (Ev890); 61 species in Australia (>250 worldwide) (Wilson 1984)

Atriplex muelleri Benth. (annual saltbush, boggabri, Mueller's saltbush) (Ev890)

Atriplex semibaccata R.Br. (creeping saltbush, berry saltbush) (Ev890)

Beta vulgaris L. (beet) (Ev890); cultivated

Chenopodium spp. (Ev893)

Chenopodium album L. (fat hen, white goosefoot)

Chenopodium auricomum Lindley (Queensland bluebush)

Chenopodium hubbardii Aellen

Chenopodium pumilio R.Br. (small crumbweed, clammy goosefoot)

Gardner & Bennetts (1956) reported an association between this species and deaths in sheep in WA in a year of very high rainfall. No clinical or pathological data were recorded. An incident in south-western Australia in 2002 killed 40 of 300 hungry ewes given access to a *C. pumilio*-dominated pasture causing marked hypocalcaemia, pulmonary oedema and renal oxalosis (Allen 2002).

Einadia trigonos (Schultes) Paul G. Wilson [=*Chenopodium trigonon* Schultes]; there are 3 subspecies - *trigonos, stellulata, leiocarpa* (Wilson 1984)

Enchylaena tomentosa R.Br. (ruby saltbush) (Ev898); there are 2 subspieces - *tomentosa, glabra* (Wilson 1984)

Kochia scoparia (Ev903) (not listed as naturalised in *Flora Aust.* **4** (1984)) - used in Western Australia as a salt-tolerant species, but now under eradication as a weed

Neobassia proceriflora (F.Muell.) A.J.Scott [= *Threlkeldia proceriflora* F.Muell.] (soda bush) [DM86]

Salsola kali L. (soft roly-poly) [DM107] (Ev913)

Scleroblitum atriplicinum (F.Muell.) Ulbr. [= *Chenopodium atriplicinum* (F.Muell.) F.Muell. (?) (Ev893)

Sclerolaena spp.

Sclerolaena anisacanthoides (F.Muell.) Domin [= *Bassia anisacanthoides* F.Muell.] (Ev890)

Sclerolaena calcarata (Ising) A.J.Scott [= *Bassic calcarata* Ising] (Ev890)

Sclerolaena muricata (Moq.) Domin [= *Bassia quinquecuspis* (F.Muell.) F.Muell.] (five-spined saltbush or bassia; black, prickly or spiny roly-poly; electric burr,) (Ev890) ; there are 3 subspecies - *muricata, semiglabra, villosa* (Wilson 1984)

Family Oxalidaceae

Oxalis spp. (sorrels)

Oxalis acetosella (Ev909)

Oxalis corniculata (yellow wood-sorrel) (Ev909)

***Oxalis pes-caprae* L.** (soursob, Bermuda buttercup [NZ, USA], buttercup oxalis [UK, USA], cape cowslip [UK], geelsuring [S.Afr.], yellow sorrel [S.Afr.], sourgrass) (Ev909)

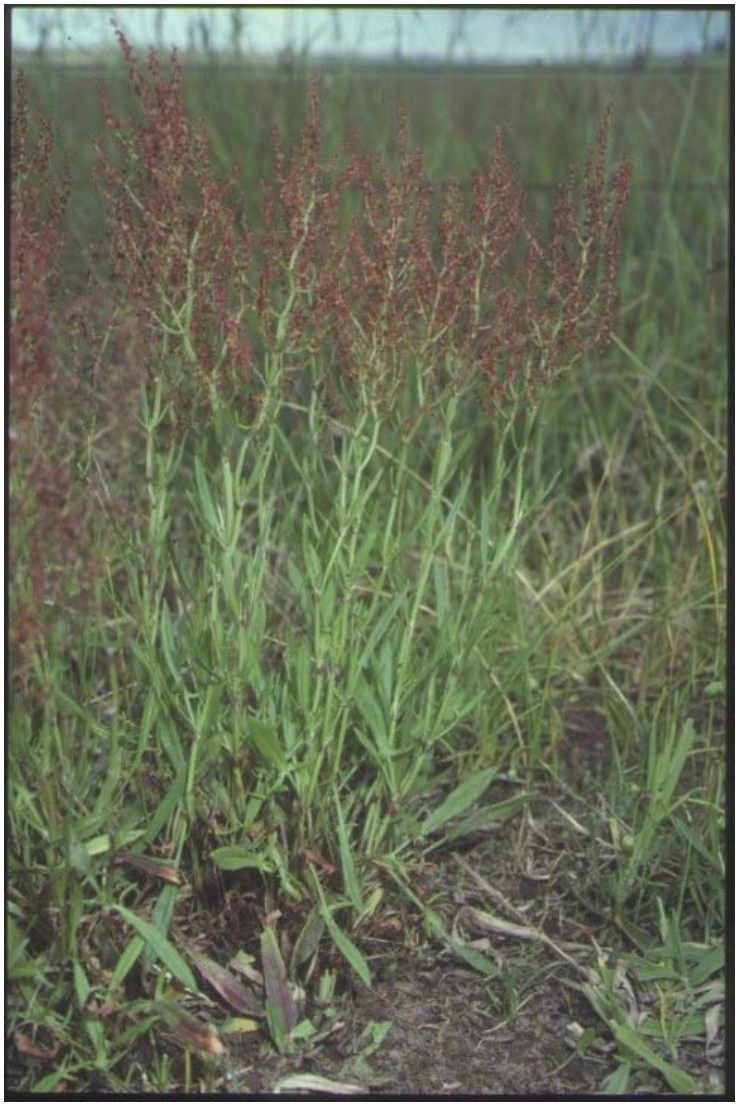
Oxalis pes-caprae (soursob). [RAM Photo]

Family Poaceae (grasses) - associated with *acute* oxalate poisoning from soluble oxalates; see notes on calcium oxalate crystals (non-raphide) for anti-nutritive effects

Setaria sphacelata (setaria) [DM54; Seawright *et al.* 1970]
Cenchrus ciliaris (buffel grass) [DM51; McKenzie *et al.* 1988]
Panicum antidotale (blue panic)

Family Polygonaceae

Acetosella vulgaris *(Rumex acetosella)* (sheep sorrel) (Ev913)
Emex australis (spiny emex, doublegee, three-cornered jack) (Ev897)
Rheum rhaponticum (rhubarb) (Ev913); cultivated
Rumex spp. (docks) (Ev913)
Rumex brownii (swamp, brown, hooked or slender dock) (Ev913)
Rumex conglomeratus (clustered or sharp dock) (Ev913)
Rumex crispus (curled dock) (Ev913)
Rumex vesicarius (Acetosa vesicaria) (ruby dock, rosy dock, bladder dock)



Acetosella vulgaris (sheep sorrel). [RAM Photo]

Family Portulacaceae

Portulaca spp. (pigweed, munyeroo) [DM108]
Portulaca oleracea (pigweed, purslane)
Portulaca sp.aff. *oleracea* (inland pigweed, munyeroo)
Calandrinia spp. (parakeelya) (Ev892)

Calandrinia balonensis (parakeelya)
Calandrinia polyandra

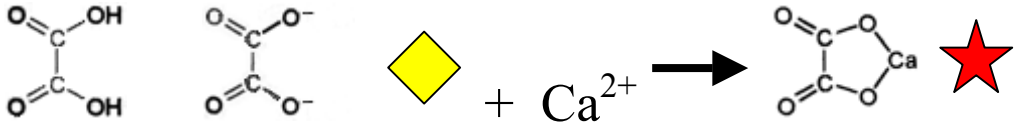
Oxalate content of some plants from Queensland (% dry matter). Data from Mathams & Sutherland (1952); Assay method of Moir (1953) - = none detected; ND = not assayed

Botanical name	Total oxalate	Soluble oxalate	No. of specimens assayed
FAMILIES WITH SPECIES CONTAINING SIGNIFICANT SOLUBLE OXALATE CONCENTRATIONS			
Aizoaceae			
Tetragonia expansa	2.9-13.4	2.3-12.0	13
Trianthema portulacastrum	9.6	7.7	1
Amaranthaceae			
Alternanthera nodiflora	2.5-3.7	0.6-1.3	2
Amaranthus mitchellii	7.2	4.6	1
Chenopodiaceae			
Atriplex muelleri	6.2-7.0	4.0-4.5	2
Atriplex semibaccata	1.9-10.2	0.6-7.4	10
Bassia anisacanthoides	5.7	0.5	1
Bassia echinopsila	2.4-7.9	0.0-5.1	4
Bassia quinquecuspis	2.4	2.2	1
Bassia tricuspis	2.5	-	1
Chenopodium auricomum	4.6	2.1	1
Chenopodium hubbardii	6.5-8.7	1.4-6.3	4
Chenopodium triangulare	5.5	4.3	1
Enchylaena tomentosa	6.7-7.2	6.5-6.7	3
Kochia coronata	2.0	-	1
Salsola kali	5.0-15.7	2.0-10.7	7
Threlkeldia proceriflora	3.6-8.6	2.0-6.2	2
Oxalidaceae			
Oxalis corniculata	7.0	4.1	1
Poaceae			
Aristida latifolia	0.0-0.2	-	2
Astrebla lappacea	-	-	1
Astrebla spp. (mixed species)	-	-	1
Bothriochloa ewartiana	-	-	1
Brachiaria purpurascens	1.1	0.5	1
Dactyloctenium radulans	0.5	-	1
Dichanthium sericeum	-	-	1
Enneapogon flavescens	-	-	1
Eragrostis setifolia	-	-	1
Eriochloa pseudoacrotricha	0.0-0.9	0.0-0.4	3
Iseilema membranaceum	-	-	1
Iseilema vaginiflorum	-	-	1
Leptochloa digitata	-	-	1
Panicum antidotale	4.9	3.5	1
Panicum maximum	1.5	0.5	1
Paspalidium caespitosum	1.2	0.9	1
Triraphis mollis	0.1	-	1
Portulacaceae			
Portulaca oleracea	3.5-11.1	4.3-9.4	13
Zygophyllaceae			
Emex australis	6.0	2.0	1
Tribulus terrestris	0.4	ND	1
FAMILIES WITHOUT SPECIES CONTAINING SIGNIFICANT SOLUBLE OXALATE CONCENTRATIONS			
Apiaceae			
Daucus glochidiatus	0.8-2.5	-	3
Asteraceae			
Brachycome curvicarpa	0.5	-	1
Calotis hispidula	0.0-trace	-	2
Cassinia laevis	-	-	1
Gnaphalium japonicum	-	-	1
Minuria integerrima	-	-	1
Pterigeron odorus	0.5	-	1
Pterocaulon sphacelatum	0.4	-	1
Senecio lautus	-	-	1
Sphaeranthus hirtus	1.2	-	1
Verbesina encelioides	0.4	-	1
Vittadenia pterochaeta	-	-	1

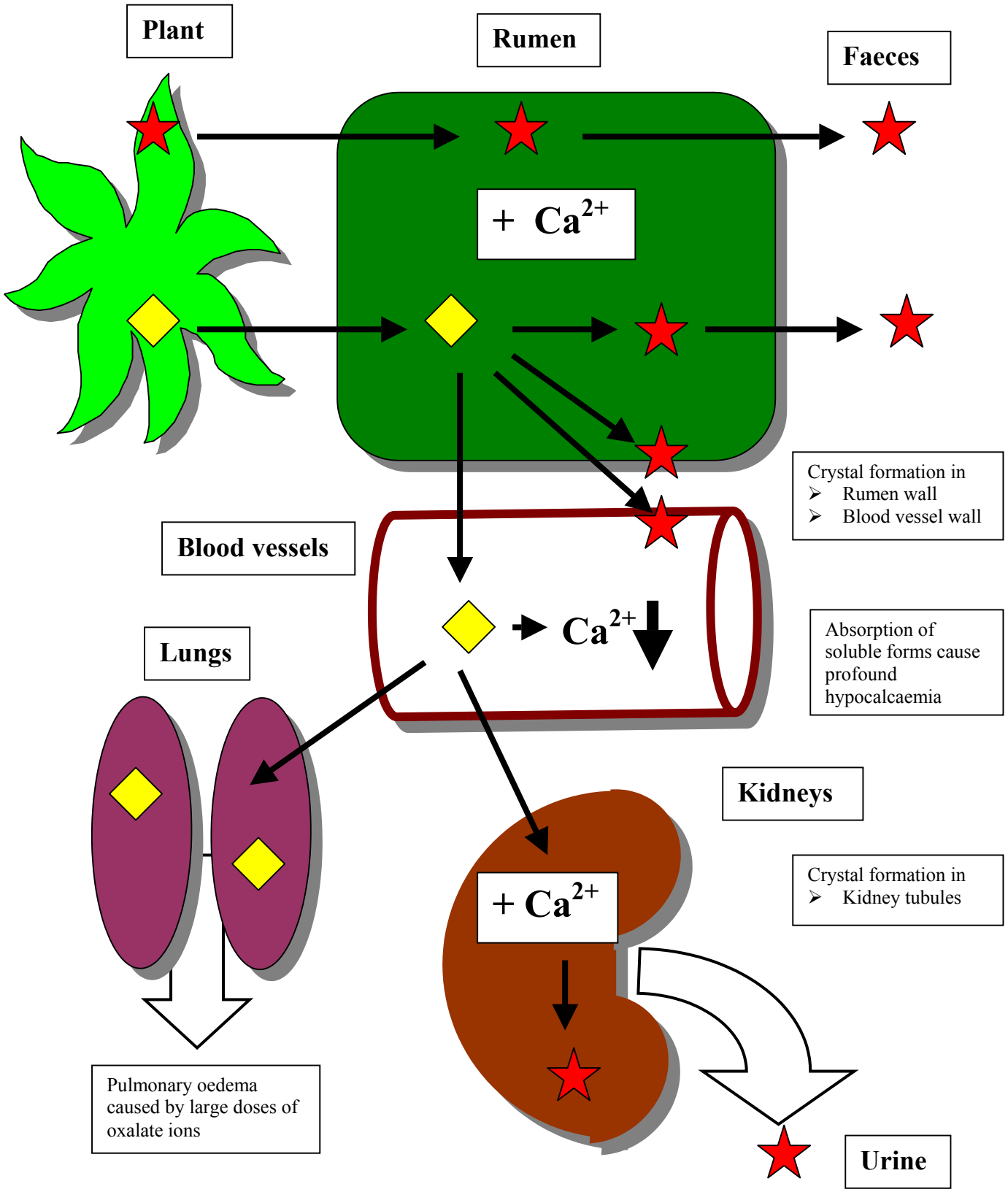
Oxalate content of some plants from Queensland (% dry matter). Data from Mathams & Sutherland (1952); Assay method of Moir (1953) - = none detected; ND = not assayed

Botanical name	Total oxalate	Soluble oxalate	No. of specimens assayed
Vittadenia triloba	0.5	-	1
Brassicaceae			
Lepidium hyssopifolium	-	-	1
Caesalpiniaceae			
Cassia occidentalis	-	-	1
Cucurbitaceae			
Cucumis myriocarpus (fruit)	-	-	1
Cucumis trigonus (fruit, dry vine)	-	-	1 each
Cyperaceae			
Cyperus dactylotes	-	-	1
Cyperus gilesii	-	-	1
Euphorbiaceae			
Euphorbia drummondii	0.0-0.5	-	3
Phyllanthus maderaspatensis	-	-	2
Fabaceae			
Psoralea cinerea	0.8	-	1
Goodeniaceae			
Goodenia strangfordii	-	-	1
Lamiaceae			
Basilicum polystachyon	0.8	-	1
Teucrium integrifolium	2.0	-	1
Liliaceae			
Bulbine bulbosa	-	-	1
Malvaceae			
Abutilon malvifolium	1.5	ND	1
Abutilon otocarpum	0.0-1.7	-	2
Abutilon oxycarpum	5.9	-	1
Malvastrum spicatum	0.5-2.5	-	4
Sida fibulifera	0.0-2.0	-	3
Sida trichopoda	0.0-1.1	-	2
Meliaceae			
Owenia acidula	3.4	-	1
Mimosaceae			
Acacia cambagei	-	-	1
Neptunia gracilis	0.9	-	1
Myoporaceae			
Eremophila maculata	2.1	-	1
Nyctaginaceae			
Boerhavia diffusa	3.6	0.5	1
Plantaginaceae			
Plantago varia	-	-	7
Solanaceae			
Nicotiana megalosiphon	-	-	1
Solanum esuriale	2.4-2.8	-	2
Solanum nigrum	-	-	1
Verbenaceae			
Verbena officinalis	0.0-2.6	-	3

- certain species of *Aspergillus*, *Penicillium* and other fungi growing on feedstuffs (e.g. hay) may produce significant amounts of oxalic acid (Wilson & Wilson 1961, Hodgkinson 1977)



Soluble forms (oxalic acid, oxalate ion) Insoluble form (calcium oxalate crystals)



Toxicity:
- toxicity depends on the **forms of oxalate** present in plants
    - water-**soluble** oxalate ion or acid oxalate (usually with Na, K or NH_4) → acute toxicity
    - water-**insoluble** calcium oxalate (as **crystals** - raphides, prisms, druses) →
        - ➢ buccal mucosa irritation (raphides) or
        - ➢ interfere with Ca availability from feed (others)
- oxalate ions have a high affinity for Ca
- clinical manifestation of oxalate-animal interactions depend on **where** the combination of calcium with oxalate takes place
    - **soluble** oxalates are absorbed and combine with Ca **in the body** (rumen wall, blood, kidney tubule lumen)
    - insoluble oxalates combine with Ca **in the plant** as crystals

- syndromes from **soluble oxalates**
    - acute oxalate poisoning
        - sheep, cattle
        - very rare in horses (experimental cases - Stewart & MacCallum 1944)

        One case of sub-acute oxalate poisoning of horses grazing a lush pasture of *Setaria sphacelata* cv. Kazungula has been reported from central Queensland in 1981 (McKenzie 1985). Two females, 1 and 3 years old, were affected. Both horses became depressed, developed oedema of the head, neck and ventral abdomen and had urinary incontinence. Hypocalcaemia and increased serum creatinine concentrations were detected. Death occurred 5 days after onset. Necropsy revealed nephrosis with heavy deposits of calcium oxalate crystals in renal tubules. Kazungula setaria can contain up to 6% soluble oxalate.

    - chronic oxalate poisoning – sheep grazing ***Oxalis pes-caprae*** (soursob)
    - abortion - cattle (foetal renal oxalosis) [Gopal *et al.* 1978, Moffatt 1974, Schiefer & Moffatt 1974, Schiefer *et al.* 1976]
    - hypocalcaemia without kidney damage (McKenzie 2001)
- syndromes from **calcium oxalate crystals**
    - nutritional secondary hyperparathyroidism - horse (*q.v.*)
    - buccal irritation - dog, cat, human (livestock) (*q.v.*)

Mode of action (acute poisoning):
- oxalate ions absorbed into the circulation

→ combine with plasma Ca → **hypocalcaemia**
→ combine with Ca in renal tubular lumens → **nephrosis**, crystals → physical blockage
→ direct irritant action on lung capillaries → pulmonary oedema
→ combine with Ca in rumen wall (mucosa and capillary walls) → rumenitis (in severe cases)

Conditions of poisoning:
- soluble oxalate concentrations **greatest in actively-growing, lush leaf material**
- well fertilised plants contain greater concentrations of soluble oxalates (e.g. around stock yards)
- factors predisposing to acute poisoning from soluble oxalates
    - soluble oxalate content **> 2.0 – 2.5% of dry matter** (often > 10%)
    - hungry animals → **rapid intake of large dose**
    - **rumen flora not adapted to catabolise oxalate** (not recently exposed to oxalate-containing plants); ruminants acquire the capacity to tolerate large intakes of oxalates if fed gradually-increasing amounts of oxalate-containing feed and this tolerance depends on catabolism of oxalates by ruminal microbes, principally anaerobes (Allison 1978)

Clinical signs:
- acute oxalate poisoning
    - dyspnoea
    - staggering, stiff gait
    - **collapse → coma → death without struggling**
    - sometimes mild tetany, mild hypersensitivity
- chronic oxalate poisoning → chronic weight loss→ emaciation, polyuria

Pathology:
- acute oxalate poisoning
    - **hypocalcaemia**
    - ↑ urea, ↑ creatinine
    - pulmonary congestion and oedema
    - hyperaemia of forestomach walls; rumenitis in severe cases
    - pale swollen kidneys → **nephrosis with calcium oxalate crystals** in tubules
- chronic oxalate poisoning → shrunken fibrotic kidneys; ↑ serum urea & creatinine, ↓ protein

Diagnosis:
- access + hypocalcaemia + calcium oxalate crystals in kidney tubules
- assay plants for soluble oxalate content (laboratory method: Roughan & Slack 1973)
    - hazardous plants contain > 2.0 - 2.5% soluble oxalate in dry matter
    - N.B. other conditions of poisoning must be satisfied before poisoning can take place

Therapy:
- acute oxalate poisoning - **calcium borogluconate** IV or SC

Therapeutic protocol for acute oxalate poisoning

- IV or SC 25% calcium borogluconate @ 50-100 ml (sheep), 300-500 ml (cattle)
- oral fluids (?)
- prognosis always guarded (because of risk of nephrosis)

Prevention & control:
- avoid access to hazardous plants when animals are hungry
- maintain low level access to oxalate in feed to ensure ruminal flora remains adapted, e.g. on *Setaria sphacelata* pastures

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☑ *Calcium oxalate raphide crystals*

Core data

Common sources: members of arum plant family (*Dieffenbachia, Zantadeschia, Alocasia* and others)
Animals affected:

- herbivores
- young cats & dogs

Mode of action: microscopic needle-like crystals penetrate buccal mucosa → local irritation
Poisoning circumstances:

- herbivores: access to native or naturalised species in pasture
- pets: young animals' exploratory behaviour (chewing various objects)

Main effects: local buccal irritation
Diagnosis: syndrome + access
Therapy: non-specific anti-inflammatory drugs
Prevention: deny access

Microscopic needle-shaped crystals of calcium oxalate, raphides are often contained under pressure in explosive ejector cells (idioblasts) of the leaves of plants, particularly those in the Family Araceae (arum family). Not all Araceae family members contain idioblasts – those with idioblasts below are indicated by ♠ (Genua & Hillson 1985).

Sources:

- popular **ornamental and house plants**
- Family Araceae (arum family) (the family contains about 110 genera and 1800 species):
  - ***Dieffenbachia* spp.** (dumb cane)♠ - houseplants
  - ***Zantedeschia* spp.** (arum lily, calla lily)♠ – naturalised in WA
  - ***Alocasia brisbanensis*** (cunjevoi) – Australian native rainforest species (previously classified erroneously as *A. macrorrhizos* or '*macrorrhiza*' (Hay & Wise 1991)) [the common name cunjevoi (or cunje) is also used for a common Australian littoral tunicate (sea-squirt), *Pyura stolonifera*, popular as a fish bait] (Beare 1926, Macpherson 1929)
  - *Alocasia macrorrhizos* (Lin *et al.* 1998)
  - *Monstera deliciosa* (monstera, fruit salad plant) (Rakovan *et al.* 1973)
  - *Philodendron* spp.♠
  - *Anthurium* spp. (flamingo flower)
  - *Arum* spp. (lords-and-ladies, cuckoo-pint)
  - *Spathiphyllum* spp.
  - *Colocasia* spp.♠ (taro)
  - *Xanthosma* spp. (elephant's ear) (Wood *et al.* 1997)
  - *Caladium* spp.
  - *Syngonium* spp.♠
  - and probably others
- Family Araliaceae:
  - *Schlefflera* spp. (umbrella trees)
- Family Arecaceae (the palms)
  - *Caryota mitis* Lour. [= *C. furfuracea* Blume, *C. griffithii* Becc., *C. sobolifera* Wallich ex Mart.] (fishtail palm, Burmese fishtail palm, clustered fishtail palm, tufted fishtail palm) – fruit pulp, fruit juice and the fibres of the base of the leaf stalks contain crystals (Spoerke & Smolinske 1990)

Toxicity:

- herbivores, cats, dogs
- humans (Lin *et al.* 1998, Pedaci *et al.* 1999)

Mode of action:

- damage to leaves (mastication) → injection of raphide crystals into in-contact tissues (buccal mucosa) → local mechanical irritation + possibly → portal of entry for irritant

soluble chemicals (including oxalic acid, proteolytic enzymes) (Arditti & Rodriguez 1982)
- it has been suggested that the abundant druse crystals of calcium oxalate may also be mechanical irritants of tissues (Genua & Hillson 1985)

Conditions of poisoning:
- herbivores: access to native or naturalised species in pasture
- pets: young animals' exploratory behaviour (chewing various objects)

Clinical signs:
- congestion ± swelling of buccal mucosa/tongue
- excessive salivation/drooling
- pain reactions (e.g. pawing at mouth)

Pathology: not described
Diagnosis: syndrome + access
Therapy:
- non-specific anti-inflammatory therapy
- the intoxication is unlikely to be serious unless oedema of the buccal mucosa extends to the pharynx and threatens patency of the airway

Prevention & control: deny access
References:

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☑ *Calcium oxalate crystals (non-raphide)*

Summer's steers grow fat
On buffel, para, panic –
Killers of horses
Haiku (1983). R.A. McKenzie

Core data

Syndrome names:
- nutritional secondary hyperparathyroidism
- osteodystrophia fibrosa
- big head

Common sources:
- tropical pasture grasses
- particularly *Cenchrus ciliaris, Setaria sphacelata*

Animals affected: horses

Mode of action: Ca oxalate crystals in grass leaves undigested by horses → no Ca absorption → negative Ca balance

Poisoning circumstances: horses grazing pure or near-pure swards of tropical pasture species

Main effects:
- lameness
- loss of weight
- swelling of jaws
- osteodystrophia fibrosa
- pitted joint surfaces

Diagnosis: pasture type + syndrome

Therapy:
- move to non-hazardous pasture
- mineral supplement (Ca:P 2:1) 2 kg / horse / week for 6 months
  - rock phosphate in molasses or mixture of 1/3 limestone + 2/3 DCP in molasses

Prevention:
- graze non-hazardous grasses
- maintain legume in pasture (Ca source)
- if non-hazardous pasture unavailable: feed supplement (1 kg / horse / week)

Strictly speaking, this syndrome is **anti-nutritive** rather than toxic.

Syndrome names:
- equine nutritional secondary hyperparathyroidism
- *osteodystrophia fibrosa*
- **big head** of horses

Sources:

Known hazardous grasses (Family Poaceae) are:

Cenchrus ciliaris (buffel grass) [DM51]
Cenchrus setiger (birdwood grass) (Allen 1999)
Setaria sphacelata (setaria) [DM54]
Setaria incrassata (purple pigeon grass) (McKenzie 1988)
Panicum maximum var. *trichoglume* (green panic)
Panicum maximum (Guinea grass)
Pennisetum clandestinum (kikuyu)
Pennisetum polystachyon (mission grass)
Pennisetum purpureum (Napier grass)
Digitaria eriantha ssp. *pentzii* [= *Digitaria decumbens*] (pangola grass)
Urochloa mutica [= *Brachiaria mutica*] (para grass)
Brachiaria decumbens (signal grass)

Toxicity [*sic*]:

Horses only are affected.

Weaned foals and nursing mares are most at risk as those classes of horse with the greatest calcium demand.

Hazardous grasses have **Ca : total oxalate ratios < 0.5**. This usually occurs when their **total oxalate content** is **> 0.5%** of dry matter.

Mode of action:

Most calcium is present in hazardous grass leaves as calcium oxalate crystals (prismatic & druses, not raphides).

Oxalate crystals are undigested in horses before reaching the colon, thus no Ca is absorbed through the major site for Ca absorption in duodenum → negative Ca balance → hyperparathyroidism.

Horses fed grass hays (setaria, buffel grass) in mineral balance experiments lost up to 40 mg Ca/kg live weight/day and up to 20 mg P/kg day/day (Blaney *et al.* 1981a).

In contrast, ruminal microbes destroy oxalate through using it as an energy source, thus releasing Ca for absorption.

Cenchrus ciliaris (buffel grass) (left); *Setaria spacelata* (right) [RAM Photos]

Oxalate content of tropical pasture grasses grown in Australia [species associated with field cases of NSH in grazing horses are given in **bold type**]

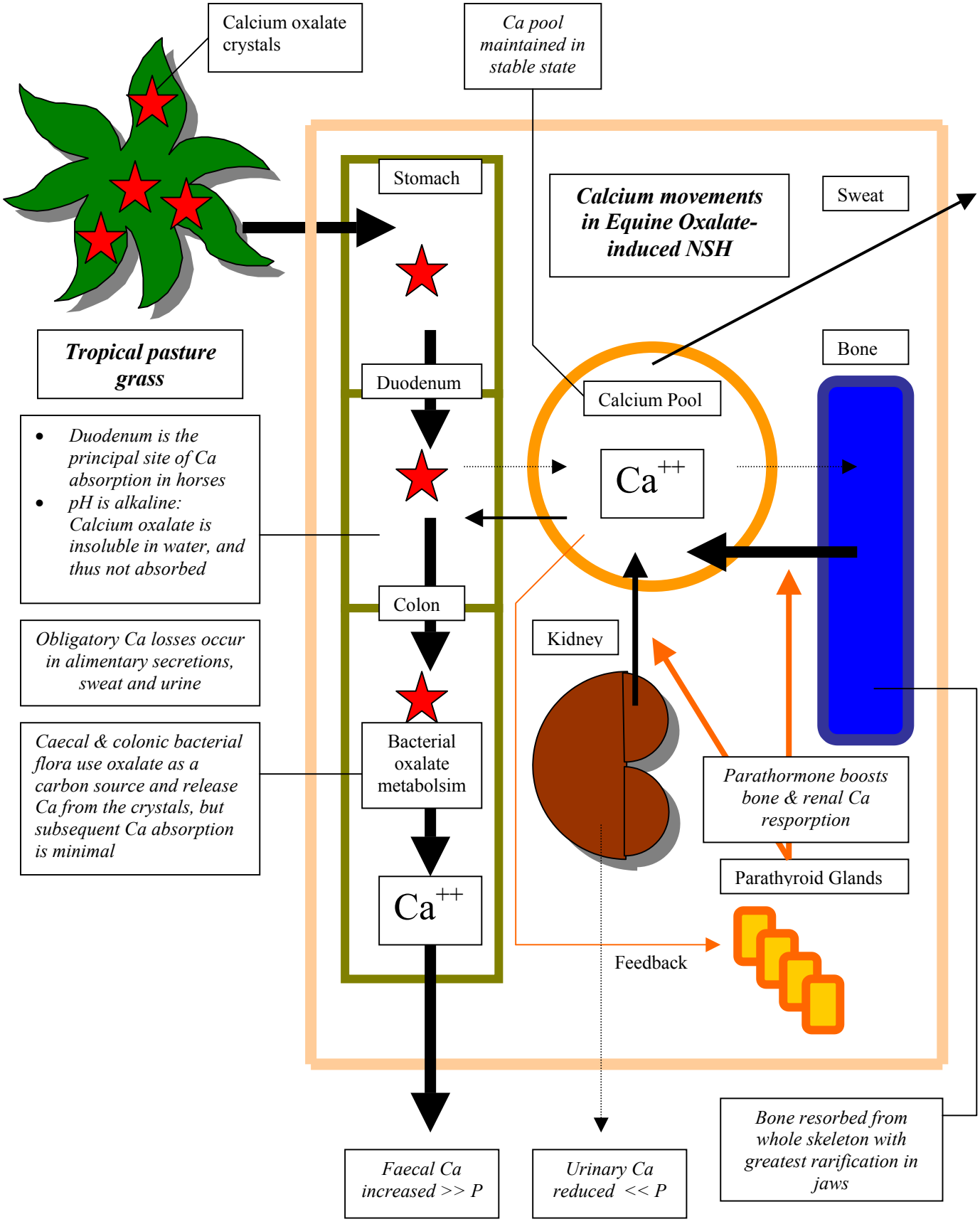
Botanical name	Common name	Total oxalate (% d.m.)	Soluble oxalate (% d.m.)	References
Aristida latifolia		0.2		Mathams & Sutherland 1952
Astrebla spp.	Mitchell grasses	<0.5		Mathams & Sutherland 1952
Avena sativa	oats	0.08	0.04	Blaney *et al.* 1981a
Axonopus compressus	carpet grass	0.02		Garcia-Rivera & Morris (1955)
Bothriochloa ewartiana		<0.5		Mathams & Sutherland 1952
Bothriochloa insculpta	creeping blue grass	0.14		BJ Blaney, unpublished data 1979
Bothriochloa pertusa	Indian blue grass	0.24	0.04	BJ Blaney, unpublished data 1979
***Brachiaria* sp.**		1.15±0.02	0.60±0.20	Ndyanabo 1974
Brachiaria brizantha		1.07±0.08	0.47±0.02	Ndyanabo 1974
		2.0	0.85	Sood & Chopra 1973
Brachiaria decumbens	**signal grass**		0.13-0.89	Jones & Ford 1972
		1.10±0.20	0.47±0.20	Ndyanabo 1974
		0.46-0.48 (leaf)		Middleton & Barry 1978
Brachiaria mutica	**para grass**	0.85	0.43	Walthall & McKenzie 1976
		0.75	0.37	Blaney *et al.* 1981a
		1.68	0.84	Sood & Chopra 1973
Brachiaria purpurascens		1.1	0.5	Mathams & Sutherland 1952
Cenchrus ciliaris	**buffel grass**		1.4-1.8	Jones & Ford 1972
		1.99±0.52	1.30±0.43	Walthall & McKenzie 1976
		3.5		RA McKenzie, unpublished data ("Ba Downs")
		2.7-4.0	1.8-2.9	McKenzie *et al.* 1988
		1.06-1.73	0.31-1.19	Blaney *et al.* 1981a
		2.5-4.2		McKenzie *et al.* 1981a
		0.52-2.92	0.11-1.35	Sood & Chopra 1973
		6.50	5.00	McKenzie 2001 [01-189830]
Cenchrus segiter	**Birdwood grass**		1.4	Jones & Ford 1972
		1.01		Allen 1999
		2.33	1.26	Sood & Chopra 1973
Chloris gayana	Rhodes grass		0.07-0.10	Jones & Ford 1972

Oxalate content of tropical pasture grasses grown in Australia [species associated with field cases of NSH in grazing horses are given in **bold type**]

Botanical name	Common name	Total oxalate (% d.m.)	Soluble oxalate (% d.m.)	References
		0.44±0.04	0.38±0.17	Ndyanabo 1974
		0.45	0.12	Blaney *et al.* 1981a
Chloris inflata		0.43		Garcia-Rivera & Morris 1955
Cynodon dactylon		0.02-0.16		Garcia-Rivera & Morris 1955
		1.10±0.001	-	Ndyanabo 1974
	African star grass	0.6		BJ Blaney, unpublished data 1981
		0.88	0.31	Sood & Chopra 1973
Cynodon plectostachyum		0.09		Garcia-Rivera & Morris 1955
Dactyloctenium radulans	button grass	0.5	-	Mathams & Sutherland 1952
Dichanthium aristatum	Angleton grass		0.07-0.08	Jones & Ford 1972
		0.21		BJ Blaney, unpublished data 1979
Dichanthium sericeum		<0.5		Mathams & Sutherland 1952
Digitaria eriantha* ssp. *pentzii ***(D. decumbens)***	**pangola grass**	0.89		Garcia-Rivera & Morris 1952
			0.35-0.65	Jones & Ford 1972
		1.22-1.80 (leaf)		Middleton & Barry 1978
		1.14		RA McKenzie, unpublished data 1979
		0.92	0.37	Blaney *et al.* 1981a
		0.49	0.42	Sood & Chopra 1973
Digitaria didactyla	blue couch		0.53	Jones & Ford 1972
Digitaria smutsii			0.36-0.57	Jones & Ford 1972
Enneapogon flavescens		<0.5		Mathams & Sutherland 1952
Eragrostis setifolia		<0.5		Mathams & Sutherland 1952
Eriochloa polystachya		0.22		Garcia-Rivera & Morris 1952
Eriochloa pseudoacrotricha		0.9	0.4	Jones & Ford 1952
		<0.5		Mathams & Sutherland 1952
Hymenachne acutigluma		0.2 (stem) 0.4 (leaf)	0.1 (stem) 0.4 (leaf)	R.A.McKenzie, unpublished data 2001[01-131773]; Ca 0.04%
Iseilema spp.	Flinders grasses	0.25	0.04	Blaney *et al.* 1981a
Iseilema membranaceum		<0.5		Mathams & Sutherland 1952
Iseilema vaginiflorum		<0.5		Mathams & Sutherland 1952
Leptochloa digitata		<0.5		Mathams & Sutherland 1952
Melinis minutiflora	molasses grass	0.41		Garcia-Rivera & Morris 1955
		2.0		BJ Blaney, unpublished data 1981
Panicum antidotale	blue panic	4.9	3.5	Mathams & Sutherland 1952
		3.06	1.86	Sood & Chopra 1973
		2.42-3.01	0.95-1.34	Sood *et al.* 1980
Panicum coloratum			0.17-0.50	Jones & Ford 1972
Panicum maximum	Guinea grass / panic	1.5	0.5	Mathams & Sutherland 1952
		1.05-2.26		Garcia-Rivera & Morris 1955
			0.36-0.80	Jones & Ford 1972
		0.28±0.19	0.61±0.18	Ndyanabo 1974
		1.38-1.59 (leaf)		Middleton & Barry 1978
		0.8-2.0	0.25-1.10	Talapatra *et al.* 1942
		1.27	0.88	Sood & Chopra 1973
		2.32-2.39	0.37-0.84	Sood *et al.* 1980
Panicum maximum cv. Makueni		1.03-1.07 (leaf)		Middleton & Barry 1978
Panicum maximum* var. *trichoglume	**green panic**	1.26	0.33	Walthall & McKenzie 1976
		1.16		RA McKenzie, unpublished data 1979
		0.81	0.43	Blaney *et al.* 1981a
Panicum purpurascens		1.24		Garcia-Rivera & Morris 1955
Paspalidium caespitosum		1.2	0.9	Mathams & Sutherland 1952
Paspalum commersonii	scrobic		0.05-0.09	Jones & ford 1972
		0.23±0.001		Ndyanabo 1974
Paspalum dilatatum	paspalum		0.10-0.12	Jones & Ford 1972
Paspalum fasciculatum		0.02		Garcia-Rivera & Morris 1955
Paspalum nicorae cv. Blue Dawn [= cv. Amcorae]		< 0.01	< 0.01	Cook BG [DPI Principal Scientist (Sown Pastures)], personal communication, 4 Feb 2000
Paspalum plicatulum	plicatulum	0.02		Garcia-Rivera & Morris 1955
			0.08-0.13	Jones & Ford 1972
Pennisetum clandestinum	**kikuyu**		0.72	Jones & Ford 1972
		0.8	0.3	Walthall & McKenzie 1976
		2.1	1.4	RA McKenzie, unpublished data 1974

Oxalate content of tropical pasture grasses grown in Australia [species associated with field cases of NSH in grazing horses are given in **bold type**]

Botanical name	Common name	Total oxalate (% d.m.)	Soluble oxalate (% d.m.)	References
		1.14		RA McKenzie, unpublished data 1979
		1.3	0.79	Blaney *et al.* 1981a
		3.04	1.31	Sood *et al.* 1980
Pennisetum ciliare	"buffel grass"	0.83		Garcia-Rivera & Morris 1955
Pennisetum purpureum	Napier or elephant grass	2.48-2.57		Garcia-Rivera & Morris 1955
		3.35-3.63	2.38-2.63	Lal *et al.* 1966
		3.10±0.17	2.59±0.24	Ndyanabo 1974
		0.65-3.05	0.40-1.01	Talapatra *et al.* 1942
		1.60-2.73	0.83-1.39	Sood & Chopra 1973
Pennisetum typhoides		1.51-2.31	0.34-1.37	Lal *et al.* 1966
Pennisetum typhoides x *P. purpureum*		1.26-3.06	0.25-2.39	Lal *et al.* 1966
Setaria incrassata cv. Inverell	purple pigeon grass	0.67-1.90		McKenzie 1988
Setaria sphacelata	**setaria**		2.8-4.2	Jones & Ford 1972
		3.79±0.16	3.22±0.17	Ndyanabo 1974
		2.8	2.5	Walthall & McKenzie 1976
		1.37-4.07		Hacker 1974
Setaria sphacelata cv. Bua River		5.4-6.9		Jones *et al.* 1970
Setaria sphacelata cv. Kazangula		7.8		Jones *et al.* 1970
		up to 6.3	1.2-6.0	Jones & Ford 1972
		5.9-8.6		Roughan & Warrington 1976
		5.2-10.4		Smith 1972
		1.30-3.78	0.99-2.06	Blaney *et al.* 1981a
Setaria sphacelata cv. Nandi		3.7		Jones *et al.* 1970
		5.4	4.59	Groenendyk & Seawright 1974
		2.1-2.6		Jones & Ford 1972
		4.7-8.7		Smith 1972
Setaria sphacelata cv. Narok		4.7-8.8		Smith 1972
		1.81	1.21	Blaney *et al.* 1981a
Setaria splendida		3.15-5.46		Middleton & Barry 1978
Triraphis mollis	purple plume grass	0.1	-	Mathams & Sutherland 1952
Urochloa mosambicensis	sabi		0.44-0.67	Jones & Ford 1972
		2.01	0.86	Sood & Chopra 1973


Calcium oxalate crystals
Ca pool maintained in stable state
Stomach
Calcium movements in Equine Oxalate-induced NSH
Sweat
Tropical pasture grass
Duodenum
Calcium Pool
Bone
Ca++
Duodenum is the principal site of Ca absorption in horses
pH is alkaline: Calcium oxalate is insoluble in water, and thus not absorbed
Colon
Kidney
Obligatory Ca losses occur in alimentary secretions, sweat and urine
Caecal & colonic bacterial flora use oxalate as a carbon source and release Ca from the crystals, but subsequent Ca absorption is minimal
Bacterial oxalate metabolsim
Parathormone boosts bone & renal Ca resporption
Parathyroid Glands
Ca++
Feedback
Faecal Ca increased >> P
Urinary Ca reduced << P
Bone resorbed from whole skeleton with greatest rarification in jaws

Conditions of poisoning:

- **horses grazing tropical pasture grasses as sole or major part of the diet**
- cases have been reported in horses grazing pastures dominated by forbs/herbs with a significant oxalate content e.g. *Atriplex* spp. (saltbushes)
- tropical pasture grass-associated cases have been described in northern Australia mainly, but also in the Pacific islands, South-eastern Asia, South America and southern Africa (McKenzie 1994). Ronen *et al.* (1992) describe probable cases in grazing horses in southern Africa without relating the disease to the pasture species or oxalate content.

Cattle are not affected by the syndrome, but the oxalate content of tropical grasses does reduce the availability of calcium from tropical grasses to a maximum of 50%, that is 20% less than would be the case if oxalates were not present (Blaney *et al.* 1982).

Clinical signs:

- **shifting lameness**, stiff gait – usually first sign
- **weight loss**
- **swelling of maxillae, mandibles** or both
- signs may occur together or separately in individual horses

Pathology:

- ↑ parathormone, ± ↑ serum alkaline phosphatase; **normal serum Ca, P**
- **osteodystrophia fibrosa** in swollen bones, osteoporosis in long bones
- parathyroid gland hyperplasia & hypertrophy
- **erosions, pitting of joint surfaces**

Diagnosis:

- nutritional history + syndrome
- bone biopsy + histopathology (hardly necessary)
- urinalysis for Ca, P and osmolality or specific gravity, interpreted using the Atwood Urinalysis Guide for assessing Ca and P nutrition of horses (Caple 1981, Caple *et al.* 1982) could be used to assess mild cases. Note: erroneous results may be obtained if the urine sample is obtained after the horses has been removed from its diet for several hours. The diet needs to be fed for at least 3 days before an assessment is made.
- assay parathyroid hormone (if available) (Roussel *et al.* 1987, Ronen *et al.* 1992)

Therapy:

- remove affected horses to non-hazardous pasture
- to aid remineralisation of bones, feed a **mineral supplement with a Ca:P ratio of 2:1**:
  - 2 kg supplement in 3 kg molasses to each horse each week for about 6 months [other carriers may be substituted for molasses if intake can be maintained]
  - alternative supplements (others may be formulated as well)
    - 2 kg rock phosphate (fluorine content <2.0%)
    - 2 kg mixture of 1 part ground limestone ($CaCO_3$) to 2 parts dicalcium phosphate (DCP) [0.6 kg $CaCO_3$ + 1.4 kg DCP]
    - 1.4 kg dolomite + 1.4 kg DCP
  - supplements in molasses will be consumed in a few days, but provide sufficient Ca and P for the week
- use of parenteral vitamin D or Ca preparations alone is **insufficient** to correct the lesions
- delivery of Ca through water sources alone is insufficient to meet Ca intake requirements because Ca cannot be dissolved in sufficient quantity, but any Ca from water will (of course) make a positive contribution to intake.
- therapy with mineral supplementation should result in a return to normal form and function. Lamenesses resolve well. Horses with severe jaw swelling may have some residual swelling after therapy, but in most cases the lesions are re-mineralised and re-modelled back to a normal appearance.

Prevention & control:

- graze horses on hazardous pastures for **no more than 1 month** at a time without providing mineral supplement
- **maintain** a significant **legume** component in the pasture if possible (good Ca sources). Legumes tend not to persist with vigorous grasses such as *Cenchrus ciliaris* and *Setaria sphacelata*, so this is a difficult option to achieve, although it is the best long-term solution in many cases. There is a report of persistence of *Stylosanthes scabra* cv. Seca (seca stylo; shrubby stylo) in buffel grass pasture under cattle grazing pressure for 10

years after aerial sowing into brigalow scrub soil over shale (J & L Gommersall, personal communication 21 May 1999). After renovation, the seca stylo formed 10-20% of pasture mass (rough estimate) in one paddock that had been locked up for a short period. There was no observation on palatability for horses, but it was well accepted by cattle and grazed almost to the point of invisibility.

- graze **non-hazardous pastures:**
  - *Chloris gayana* (Rhodes grass)
  - *Paspalum* spp.
  - *Cynodon* spp. (couch grasses)
  - *Bothriochloa insculpta* (creeping blue grass)
  - temperate pastures grasses (*Lolium, Phalaris, Festuca*, etc.)
  - all native grass species
  - if non-hazardous pasture is unavailable, feed half the therapeutic dose of **supplement** weekly for each horse (1 kg in 1.5 kg molasses); supplying an equivalent amount of Ca and P from good quality lucerne would need 20kg lucerne/horse/week. The risk of chronic fluorosis from long-term feeding of Christmas Island rock phosphate (1.5%F) or DCP (0.4%F) as mineral supplements has been assessed as very small to negligible (Blaney *et al.* 1981c).

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