3: Introduction to Plant, Fungal and Cyanobacterial Poisoning

The honey of poison-flowers and all the measureless ill.
From *Maud* (1855). Alfred, Lord Tennyson (1809-1892)

Plant poisoning – lessons from the past

Listen carefully to the Reverend Gilbert White writing in England in the late eighteenth century. The comments in parentheses are mine:

" In a yard, in the midst of the street, till very lately grew a middle-sized female tree of the same species [*Taxus baccata*, English yew], which commonly bore great crops of berries. By the high winds usually prevailing about the autumnal equinox, these berries, then ripe, were blown down into the road, where the hogs ate them. And it was very remarkable, that, though barrow hogs and young sows found no inconvenience from this food, yet milch-sows often died after such a repast; a circumstance that can be accounted for only by supposing that the latter, being much exhausted and hungry, devoured a larger quantity [= dose-response relationship & possible variation in susceptibility through differing metabolic states].

While mention is making about the bad effects of yew-berries, it may be proper to remind the unwary that the twigs and leaves of yew, though eaten in a very small quantity, are certain death to horses and cows, and that in a few minutes. An horse tied to a yew-hedge, or to a faggot-stack of dead yew, shall be found dead before the owner can be aware that any danger is at hand; and the writer has been several times a sorrowful witness to losses of this kind among his friends; and in the island of Ely had once the mortification to see nine young steers or bullocks of his own all lying dead in an heap from browsing a little on an hedge of yew in an old garden, into which they had broken in snowy weather [= animals poisoned in circumstances of nutritional stress?]. Even the clippings of a yew-hedge have destroyed a whole dairy of cows when thrown inadvertently into a yard [= prunings may be more palatable than when on the plant itself, or more accessible]. And yet sheep and turkeys, and, as park-keepers say, deer will crop these trees with impunity [= species variation in susceptibility].

Some intelligent persons assert that the branches of the yew, while green, are not noxious; and that they will kill only when dead and withered, by lacerating the stomach; but to this assertion we cannot by any means assent, because, among the number of cattle that we have known fall victims to this deadly food, not one has been found, when it was opened, but had a lump of green yew in its paunch [= necropsy observation for diagnosis; conclusions drawn from multiple observations leading to consistent findings]. True it is, that yew-trees stand for twenty years or more in a field, and no bad consequences ensue; but at some time or other cattle, either from wantonness when full, or from hunger when empty (from both which circumstances we have seen them perish), will be meddling, to their certain destruction [= poisoning is the result of the presence of toxic material + appropriate circumstances leading to its consumption in toxic amounts]; the yew seems to be a very improper tree for a pasture field [= knowledge of toxic properties of plants allows effective prevention]."

Gilbert White (1788) *The Natural History and Antiquities of Selbourne*. Folio Society edition (1994) pp.289-290.

☑ *Poisonous plants, fungi and cyanobacteria in an Australian context*

Organisms

Broadly, the term "poisonous plant" can be used to include poisonous species of **cyanobacteria** (cyanophytes, blue-green algae), **macrofungi** ("mushrooms"), **moulds**, **ferns**, **gymnosperms** (cone-bearing plants) & **angiosperms** (flowering plants). In strict terms, these organisms belong to three of the currently recognised kingdoms of living beings – plants, fungi and one of the prokaryotic groups

Plants. In the strict sense of the term, poisonous plants in Australia include species of introduced crop, pasture & garden plants, exotic weeds and Australian native plants. The Australian flora (native and naturalised plants) contains about 20 000 flowering plant species, 110 gymnosperm species and 400 fern species (Orchard 1999). Cultivated (but not yet naturalised) plants are additional to these totals. About 1000 of all these (about 5%) are known or suspected to be toxic.
The major toxic vascular plant groups (plant families) include grasses (Poaceae), legumes (Fabaceae, Mimosaceae & Caesalpiniaceae), daisies (Asteraceae), radish/cabbage family (Brassicaceae), saltbushes (Chenopodiaceae), nightshades (Solanaceae), rice-flowers (Thymeleaceae), cycads (Cycadaceae, Zamiaceae & Stangeriaceae) and spurges (Euphorbiaceae).

Fungi. The fungal flora of Australia is relatively poorly known with an estimated 250,000 species occurring here (Pascoe 1990). Known or suspected poisonous species account for very few of these.

Cyanobacteria. The cyanobacteria (cyanophytes, blue-green algae) listed as occurring in Australia number some 400 species (Day *et al*. 1995). Of these, some 20 (about 5%) are known or suspected to be toxic.

Occurrence

All native and naturalised toxic species live in **populations** integrated into particular broad **plant communities** or **vegetation types**, often identified as alliances with particular species of *Eucalyptus, Corymbia* or *Acacia*, the dominant plant genera of the continent (Beadle 1981, Read 1987, Beard 1990). These form a mosaic across the continent, their distribution depending on **soil type** (influenced by underlying geology), and on **climate** (influenced by geographical location).

Each native toxic species is **an integrated part of particular ecosystems**. All methods imposed to prevent animal toxicity events must take serious account of this if they are to be compatible with the sustainability of these natural systems.

References:

Beadle NCW (1981) *The Vegetation of Australia.* Cambridge University Press, Cambridge.
Beard JS (1990) *Plant Life of Western Australia.* Kangaroo Press, Kenthurst NSW.
Day SA, Wickham RP, Entwisle TJ, Tyler PA (1995) *Bibliographic Checklist of Non-Marine Algae in Australia.* Flora of Australia Supplementary Series No. 4. , Australian Biological Resources Study, Canberra. pp. 199-221.
Orchard AE (editor) (1999) *Flora of Australia*. Volume 1, Introduction 2nd edition, ABRS/CSIRO, Melbourne, p.1.
Pascoe IG (1990) History of systematic mycology in Australia. In Short PS (ed.) *History of Systematic Botany in Australasia.* Australian Systematic Botany Society. pp. 259-264.
Read IG (1987) *The Bush. A Guide to the Vegetated Landscapes of Australia.* Reed Books, Sydney.
White ME (1986) *The Greening of Gondwana.* Reed Books, Sydney.

☑ How are plants, fungi and cyanobacteria known to be toxic?

By one or a combination of

- being associated with multiple cases of consistent syndromes under field conditions
- yielding positive results from feeding experiments in target animal species (important historically, but now limited by animal welfare considerations and the restricted availability of funds for research)
- having known toxins isolated or detected in hazardous amounts

Of course, for an organism to be reliably known to the community as toxic requires that evidence in the above categories be published in the scientific literature. See Building the Australian Veterinary Toxicology Knowledge Base (above).

☑ How do I recognise a poisonous plant?

No simple morphological features distinguish toxic from non-toxic plants or fungi. So, it is necessary to learn to recognise the known toxic species individually (use reference books; submit herbarium specimens to confirm tentative identifications from the field)
F**ertile** specimens are required for accurate identification, that is, specimens bearing reproductive structures (flowers, fruit, cones or spores depending on the type of plant)
Features of plants to concentrate on when trying to identify plants include growth habit (type of plant), flower shape & colour, fruit shape and leaf shape.

☑ Identification: The primacy of the scientific name

Obtaining the correct scientific (botanical) name for a plant, fungus or cyanobacterium is the key to the literature on its toxicity and thus to effective understanding and management of the toxic risk

Common names can be confusing. **One common name can refer to more than one plant species, each with very different toxins** e.g. in Australia, potato weed can mean *Heliotropium europaeum* or a *Solanum* sp.; castor oil can mean *Ricinus communis* or *Datura stramonium*. **A common name can give a misleading idea of the toxic properties of a plant.** For example, in some places, *Solanum nigrum* (*s.l.*) is called deadly nightshade, a name more properly applied to the much more toxic *Atropa belladona*. Again, *Solanum mauritianum* may be called tree tobacco or wild tobacco, but is not in the genus *Nicotiana* (the tobaccos) and does not contain nicotine or other tropane alkaloids, but glycosidic steroidal alkaloids (steroidal glycosides). See Kanis *et al.* (1999) for a discussion of common *vs.* formal botanical names.

An accurate identification provides access to knowledge of the plant's properties and thus to effective diagnosis and management of poisonings. This knowledge resides in the published scientific literature and in unpublished data in state herbariums, state departments of agriculture (primary industries) and universities. The professional identifiers of plants are **botanists**. Those most available to help veterinarians and other members of the public with plant identification are employed in state herbariums throughout Australia. Contact details for Australian state herbariums are given above (Information Sources in Veterinary Toxicology).

A number of CD-ROM publications are available for computer-assisted identification of plants in Australia. To date these are of only limited application to the identification of poisonous species and are given above (Information Sources in Veterinary Toxicology).

A useful guide to the technique for identification of flowering plants for the interested amateur, written using Australian examples, clearly illustrated with line drawings and a selection of coloured photographs, and with a glossary of botanical terms is Clarke & Lee (1987) and subsequent editions.

Taxonomic botanists agree to change scientific plant names from time to time to better reflect our current understanding of the detailed evolutionary relationships between plants. "Taxonomies are active theories about the causes of natural order, not objective, unchanging, and pre-existing stamp albums for housing nature's obvious facts" (Gould 2002). Some plants are in taxonomically unstable situations and are more likely to undergo name changes than others. New names of plants can always be linked to their previous names through the scientific literature by consulting a herbarium.

References:

Clarke I, Lee H (1987) *Name that Flower. The Identification of Flowering Plants.* Melbourne University Press, Melbourne. xii + 256 pp. ISBN 0 522 84335 2

Gould SJ (2002) *I Have Landed. The end of a beginning in Natural History.* Jonathan Cape, London. p.303.

Kanis A, Crisp MD, Orchard AE (1999) Classification, phylogeny and the *Flora of Australia.Flora of Australia* 2nd edition **1**:125-147.

☑ Why are plants poisonous?

Different animal susceptibility defines toxicity, that is, whether we perceive a plant to be "toxic" or not. A plant that poisons one animal species does not necessarily poison another species. Among mammals, there are sometimes quite striking differences in toxicity of the same plant or plant toxin to ruminants compared with monogastrics. And there is often a much wider gap between the toxicity of certain plants or toxins to insects compared with mammals.

Plants, being unable to escape by movement from the invertebrate and vertebrate animals that feed on them, have developed both physical and chemical defense methods to minimise the damage done to them by herbivory. We now look on many of the products of this chemical defense effort as toxins. Populations of herbivores, both invertebrate and vertebrate, and the flora with which they evolved over geological time have reached a state of dynamic equilibrium between the plants' chemical composition and the animals' capacity to ingest it unscathed.

This equilibrium of plant chemical defense with herbivore resistance is overturned when a population of herbivores is displaced from its natural habitat to encounter a novel flora. This is the experience of European peoples migrating to the Americas, Africa, Australia and New Zealand and bringing their domestic livestock with them. Deaths and illness among introduced flocks and herds from the effects of toxic chemicals in the indigenous floras were common and widespread in each of these continents.

Plant species develop hazardous amounts of toxic chemicals for various known reasons including

- **defence against attack by herbivores** (principally insects and molluscs), e.g. cyanogenic glycosides, bitter compounds such as alkaloids. There is a biological "arms race" between a plant species' defence chemicals and the capacity of its herbivores to detoxify these chemicals, moderated by natural selection. Poisoning of mammals can be thought of as "collateral damage" – an outcome beyond the stimulus-response cycle between insects and plants that lead to the development of the toxins.
- **competitive advantage** through the inhibition of the growth of competing plants by allelopathic chemicals, e.g. atractyloside group
- **disturbance of normal plant physiology** by environmental conditions, e.g. nitrate

Why do different plants share the same toxins?

When plants in the same or closely-related families share the same complex organic toxins, this is evidence for common ancestry with the capacity to produce the toxins being conserved during the evolution of new species through natural selection. Explaining the occurrence of the same toxin in plants of widely different families, for example galegine (*q.v.*) in *Galega officinalis* (dicots of the Family Fabaceae), *Verbesina encelioides* (dicots of the Family Asteraceae) and *Schoenus asperocarpus* and *Schoenus rigens* (monocots of the Family Cyperaceae), may represent evidence of common ancestry *or* parallel evolution - a shared solution, independently developed, to a common environmental (evolutionary) challenge in the same way that insects, birds and bats independently evolved wings.

Note that this is in some contrast with complex organic toxins shared by animals. In certain cases, such as ciguatoxins and tetrodotoxins, the diversity of animals sharing the same toxin reflects the ultimate microbial source of these toxins with their subsequent transfer to the animals either from microbes resident in their alimentary tracts or through bioaccumulation via the food web.

Reference:

Jones JS (1999) *Almost like a Whale.* The Origin of Species *Updated.* Chapter XIII. Mutual Affinities of Organic Beings. Doubleday, London. pp. 297-315.

☑ *Why do plant poisonings occur?*

Many, if not most, plant poisonings result from a **disturbance of the balance in the ecosystem** concerned, either through **human interventions** or through the impact of **natural fluctuations**. Humans impose cultivation and monocultures, remove major components of ecosystems such as trees, introduce alien plants and animals or confine and concentrate animals by fencing and providing artificial water supplies. Nature imposes flood, fire, tempest and drought. The poisonings described in this work reflect the consequences of these processes. I believe that the successful prevention of poisoning should be underpinned by an understanding of the ecological context in which it may occur. Prevention of plant poisoning is part of the development of sustainable agriculture in this country. Serious and widespread plant poisoning can be a message to the community that ecological relationships are faulty. We veterinarians can be so focused on getting the diagnosis correct and saving lives where possible, that other issues get less attention. Gathering the details and pondering the implications of the circumstances leading to poisonings need our best efforts if we are to discharge fully our responsibilities for preventing animal disease *and* establishing and maintaining stable ecosystems.

In broad terms, **ecosystem modification or disturbance** can be thought of as responsible for many incidents of poisoning in domestic animals; the unnatural combining of plants with herbivores or reduced choices for herbivores being the immediate causes.

Agents of ecosystem disturbance can be **natural** or **anthropogenic** (resulting from human activity).

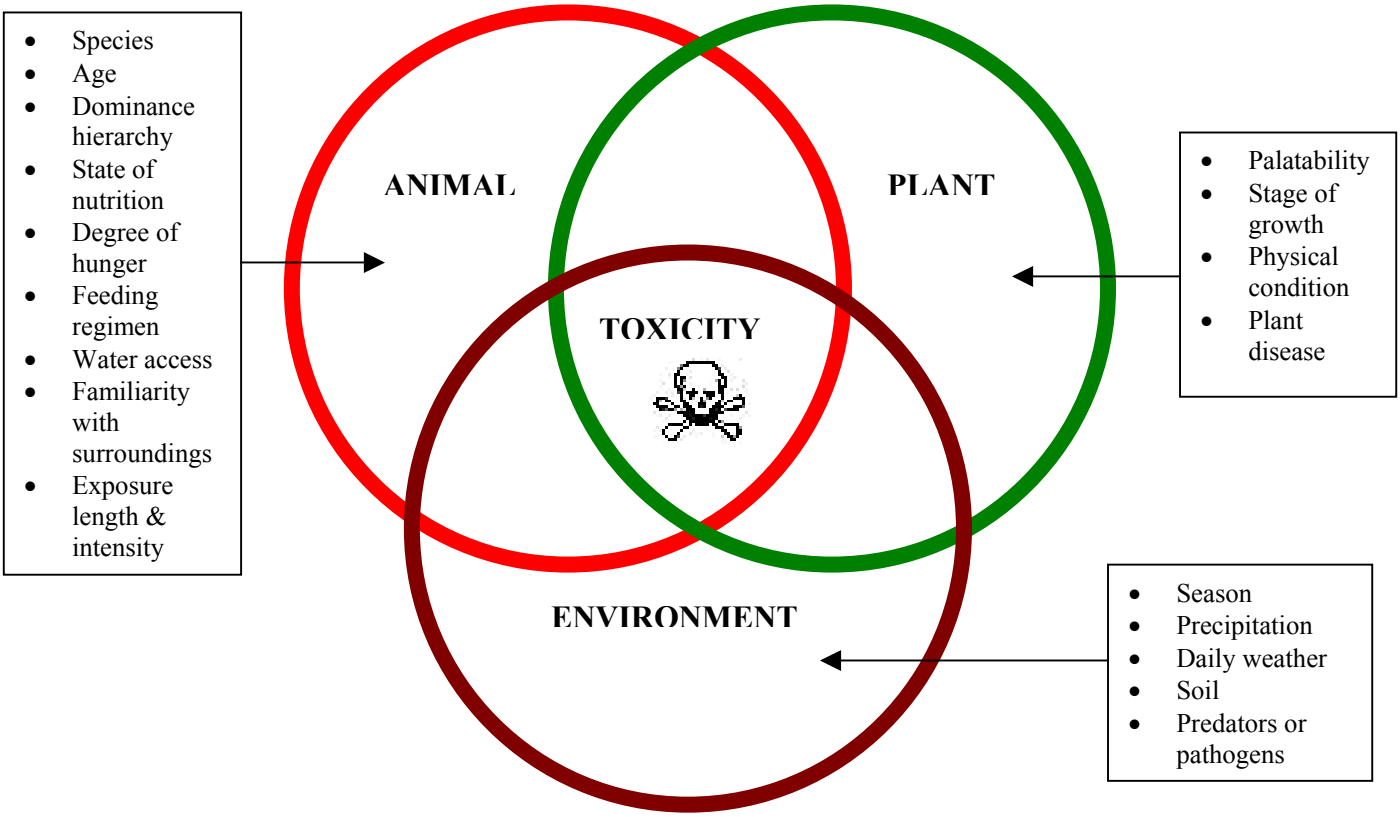
Natural causes

- drought
- fire
- storm
- flood

Human causes

- cultivation
- establishment of crop or pasture monocultures
- removal of major ecosystem components (trees)
- introduction of alien plants & animals
- confinement & concentration of animals through fencing and controlled food & water supplies
- fire
- flood

Understanding the ecological basis of poisoning incidents may allow the introduction of rational preventive measures.



☑ *Plant Factors affecting plant toxicity*

- **Palatability** and thus attractiveness to animals varies significantly among known poisonous plants. Fluoroacetate-containing plants are palatable, making them particularly dangerous to browsing animals. Applying herbicides may temporarily increase the palatability of poisonous weeds, boosting the hazard. This is known to occur with variegated thistle (*Silybum marianum*). Plants containing alkaloids are generally bitter and non-palatable. Cardiac glycoside-containing plants are usually unpalatable. These plants are likely to be eaten only when other feed is scarce or lacking, or animals are very hungry.
- **Stage of growth** affects the concentration and distribution of toxins in plants. Toxin concentrations vary in different plant parts or at different stages of maturity. Nitrate concentrates in stems. Cyanogenic glycosides and soluble oxalates concentrate in young leaves. Defense chemicals are concentrated in the plant's most vulnerable parts, particularly in its seeds, cotyledons (seed leaves) and young shoots. Not all parts of all poisonous plants are toxic. In some plants, only the seeds may be poisonous, e.g. *Ixiolaena brevicompta*, *Castanospermum australe*, in others, only the young leaves, e.g. *Sorghum* spp. Root suckers may be the most hazardous part

of toxic trees, e.g. *Erythrophleum chlorostachys* and *Alstonia constricta*, because these are readily accessible to terrestrial browsing animals.

- **Physical condition and plant disease (pathology)** may boost toxicity. Wilting, herbicide damage, insect damage, bacterial or virus infections all influence concentrations of toxins e.g. nitrates, steroidal saponins

☑ Animal Factors affecting plant toxicity

- **Species**
  - Metabolic processes, including the efficiency of detoxication processes, differ between ruminant mammals, monogastric mammals and birds causing differences in susceptibility to particular toxins. These differences can be very wide.
  - **The rumen microflora** – the bacteria and single-celled animals (protozoa) living in the paunch of animals such as cattle, sheep and goats - can either **detoxify or potentiate** ingested toxins. This underlies some of the major differences in susceptibility to intoxication between ruminants and other mammal species.
- **Age.** Young animals are generally more susceptible than adults because they have less effective detoxification mechanisms established in their body organs.
- **Position in the dominance hierarchy.** Dominant animals are more at risk of large rapid intakes which may lead to poisoning where more moderate intakes would not. Dominant animals may take more of certain normally choice plant foods, for example the high protein seed pods legumes, and thus be more likely to be poisoned when these have the highest concentration of toxins, such as fluoroacetate in *Acacia georginae*.
- **State of nutrition.** Poorly fed animals are generally more susceptible than well fed animals because they have available reduced amounts of substrates necessary for effective detoxification mechanisms.
- **Degree of hunger.** Animals are more hungry after transport or yarding and this may result in more rapid intake of a large amount of toxic material and less discrimination in the choice of plants eaten
- **Type of feed on offer.** Hand-fed animals are open to poisoning when food sources are contaminated with such material as
  - toxic weed seeds in feed grains, e.g. *Heliotropium europaeum* seeds containing pyrrolizidine alkaloids contaminating feed wheat or
  - toxic weeds contaminating hay, e.g. one-leaf cape tulip (*Homeria flaccida*) containing cardiac glycosides or mint weed (*Salvia reflexa*) containing nitrate

  and they are unable to choose not to eat the toxic component
- **Access to drinking water.** Some toxins may not be released from eaten plants and absorbed from the animal's digestive tract until after the animal has drunk water.
- **Familiarity with surroundings.** Newcomers may make less discriminating food choices. This is confounded by the increased degree of hunger experienced by animals after being transported.
- **Length and intensity of exposure**
  - chronic toxicities take time to be manifest, e.g. pyrrolizidine alkaloids accumulate in the liver and illness does not result until damage exceeds a certain threshold.
  - tolerance may develop to otherwise lethal doses of certain toxins if the body is exposed to lesser doses over a period of time e.g. nitrates, oxalates, and detoxication processes either of the rumen flora or the liver have time to develop greater efficiency.

☑ Environmental Factors affecting plant toxicity

- **Season of year** dictates
  - the **presence or absence** of particular **annual** plants in the habitat. Australia can be divided roughly on the basis of rainfall pattern into summer-rainfall-dominant (northern) and winter-rainfall-dominant (southern) zones. Annual toxic plants occur seasonally in the environment according to this rainfall pattern. In arid regions where rainfall is largely unpredictable, they occur sporadically after significant rain.
  - the **stage of growth & physiological state** of plants (the presence or absence of flowers, fruit, seeds or young leaves). Perennial toxic plants will be influenced by rainfall pattern in their production of reproductive structures and new leaf growth.

The arid zone constitutes 70% of the Australian land mass (van Oosterzee & Morrison 1991) with the remainder of the country, that carries most of the human and introduced animal populations, lying around the northern, eastern and southern rim of the continent. The sporadic nature of rainfall events in the arid zone overwhelmingly influences the occurrence of local plant populations, making plant poisoning there a consequence of rain more than of any other factor.

- **Precipitation history** (rain or snowfall)
  - dry conditions produce wilting which may increase the concentrations of some toxins (e.g. steroidal saponins)
  - droughts decrease the food choices for grazing animals by decreasing the diversity and biomass of food plant species. Animals without adequate nutritious grasses may turn to eating shrubs and trees which may be toxic.
  - floods decrease food choice to grazing animals by decreasing the area of land available for foraging
  - heavy snow decreases the food choice to grazing animals by decreasing access to food plant species
- **Daily weather conditions**
  - air temperatures influence
    - the water balance of plants, high temperatures leading to wilting and higher concentrations of certain toxins in plant tissues
    - the rate of metabolic processes in plants, for example cold temperatures reduce the rate of conversion of nitrate into proteins, thus increasing the concentration of toxic nitrates in some plants
  - rain – plants containing cyanogenic glycosides appear to be more dangerous during conditions of light rainfall or drizzle
  - fog is reputed to boost the toxicity of *Phalaris aquatica*
  - cloud cover, if heavy and prolonged, decreases photosynthesis and thus energy production by the plant and so slows or stops the conversion of nitrates to proteins, thus boosting the concentration of toxic nitrates in plants prone to
- **Soil**
  - mineral content e.g. selenium-accumulating plants will be most toxic when growing on soils rich in selenium
  - absolute amounts of plant nutrients e.g. soils high in nitrogen predispose plants to develop large concentrations of nitrates and oxalates
  - proportions of plant nutrients e.g. the balance of nitrogen and other minerals, particularly phosphorus, sulphur and molybdenum, can influence plant nitrate content. If soil is rich in nitrogen but poor in one of the other elements, nitrate ions are taken up by plants such as *Salvia reflexa* and *Silybum marianum*, but are not readily converted to ammonium ions and thus to proteins, leaving the plants with large toxic nitrate concentrations.
- **Presence of predators or pathogens**
  - insect damage may increase the concentration of defense chemicals such as cyanogenic glycosides (e.g. in *Sorghum* spp.) and alkaloids
  - microbial damage may
    - increase the concentration of defense chemicals such as the phytoalexins including furanocoumarins
    - interfere with normal physiological processes and increase the concentration of certain toxic chemicals such as nitrate as an incidental by-product

References:

Burbidge NT (1960) The phytogeography of the Australian region. *Aust. J. Bot.* **8**:75-211.
van Oosterzee P, Morrison R (1991) *The Centre. A Natural History of Australia's Desert Regions.* Reed Books, Sydney.
White ME (1986) *The Greening of Gondwana.* Reed Books, Sydney.

☑ Establishing a diagnosis of plant poisoning

Gathering sufficient evidence to diagnose plant poisoning with high probability requires meeting some or all the following conditions. **In most cases the first 4 conditions are mandatory**. Not all conditions need be met or are even technically possible in all cases - assay methods for suspected toxins may be unavailable and given the current knowledge of plant poisonings, feeding experiments are very rarely needed for final confirmation.

- The suspected plant has been **identified** accurately, allowing meaningful reference to the published literature
- Epidemiology, clinical signs, clinical pathology, necropsy and histopathology findings in the case **match** those known to be produced by the suspected plant or contained toxin
- The patient had **access** to the suspected plant in quantities capable of producing poisoning of the observed severity or extent
- There is evidence that the suspected plant has been **eaten** in quantities capable of producing poisoning of the observed severity or extent, either from examination of the remaining vegetation in the animal's environment or examination of the animal's stomach contents or both
- The suspected plant contains sufficient **toxin** to account for the observed syndrome
- The suspected **plant** has been **found** in the alimentary tract (rumen, stomach) of poisoned animals
- The plant **toxin** or known metabolite(s) has been **detected** in alimentary tract (Lang & Smith 1998) or tissues of poisoned animals. Detection methods are limited to specific toxins and in many cases to particular laboratories. Examples include multi-residue alkaloid screening tests (Holstege *et al.* 1998).
- A feeding trial with the suspected plant reproduces the syndrome under investigation. This step is rarely required and difficult to prosecute to a satisfactory conclusion.

References:

Holstege DM, Galey FD, Booth MC (1998) Development and validation of a multiresidue alkaloid screen. Chapter 48 in *Toxic Plants and Other Natural Toxicants*, edited by T Garland and AC Barr, CAB International, Wallingford UK, pp. 233-238.

Lang DG, Smith RA (1998) The chemical identification of plant toxins in ingesta and forage. Chapter 46 in *Toxic Plants and Other Natural Toxicants*, edited by T Garland and AC Barr, CAB International, Wallingford UK, pp. 223-226.

Field investigation of a suspected plant poisoning – guidelines for a thorough step-by-step approach

- Complete history taking, clinical examinations and necropsies as appropriate
- Collect from affected live animals for laboratory examination
  - blood in EDTA + thin blood smears (haematology)
  - clotted blood or blood in lithium heparin (clinical chemistry)
  - other samples as indicated by clinical findings (urine, faeces, CSF *etc.*)
- Consider collecting similar specimens from normal in-contact animals of the same class & age for comparison
- Collect from necropsied animals for laboratory examination
  - organ samples for histopathology (see above for minimum recommended set), guided by clinical and necropsy evidence – do not forget the CNS
  - sample of rumen or stomach contents (about 500 g preserved with a few mls 10% formalin, well stirred in)
- Inspect the environment of affected animals in the period before illness/death to identify known toxic plant, macrofungi or cyanobacterial species. Check for evidence of consumption of the plants/fungi/cyanobacteria. Compare their known properties with the evidence gathered from your clinical & pathological investigations. Collect appropriate specimens for submission to a herbarium to confirm your tentative field identification (you may have got it wrong!).
- If no plants, fungi or cyanobacteria are recognised that have the capacity to produce the syndrome under investigation, more detailed investigation may be undertaken as follows:
- Collect representative pressed specimens (fertile where possible) *in duplicate* of all plants in the environment of the affected animals, recording those with evidence of being eaten and the relative abundance of the species.
- Submit one set of the duplicates to the local state herbarium for identification; keeping the second set of duplicates to aid interpretation of the herbarium's report and future identification of species in the field.
- Check the results against the toxicological literature for the species and plant families identified.
- Consider a comparative study of plants on adjacent or near-by paddocks or properties using the same agricultural/pastoral system/s and class/es of animals and where the syndrome has *not* been recorded; subsequent comparison of plant lists and/or plant population densities may suggest certain species to be more available to affected than to unaffected animals.
- If the situation is serious enough and financial support is available, obtain the services of a professional botanist to undertake the field surveys in collaboration with you and the owners of affected animals.

- Consider feeding experiments using the target species and class of animal and suspected plant species; first effectively justify the experiments by balancing animal welfare considerations against the importance of the syndrome under investigation, and second design the experiments with the production of publication-standard results firmly in mind (use controls, adequate numbers, adequate statistical comparison of treatments, realistic dose rates, conditions mimicking those of the field as closely as possible, maximum data harvesting, thorough observation and recording). It is mandatory to deposit voucher specimens of the actual plant(s) used in experiments in a state herbarium to confirm the identity of the material and to guard against future changes in plant taxonomy (McKenzie 1993, Wagstaff *et al.* 1999a,b).

References:

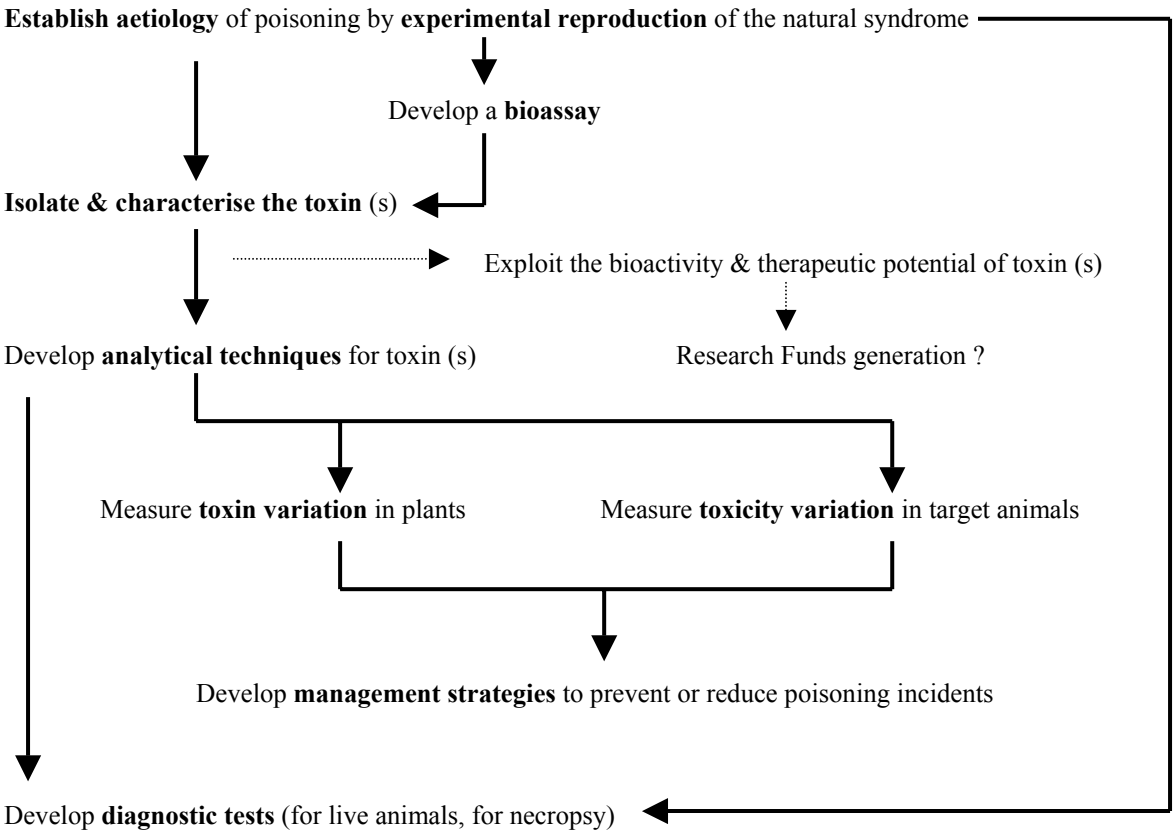
McKenzie RA (1993) Plant poisoning? Which plant?! *Aust. Vet. J.* **70**:201-202.

Wagstaff DJ, Lellinger DB, Wiersema JH (1999a) Retrospective searching for poisonous plant vouchers. *Vet. Human Toxicol.* **41**:158-161.

Wagstaff DJ, Wiersema JH, Lellinger DB (1999b) Poisonous plant vouchers. *Vet. Human Toxicol.* **41**:162-164.

Strategy for rational research leading to effective diagnosis & management of plant poisonings

[modified from a chart by Russell Molyneux, USDA Agricultural Research Service 1993]



The sequence of events that take place or may take place during research investigation of plant poisoning syndromes directed at developing rational control measures [modified from a chart by Russell Molyneux, USDA Agricultural Research Service 1993] is

- Establish the aetiology of poisoning by experimental reproduction of the natural syndrome using the suspected plant species
- Develop a bioassay to guide attempts at toxin isolation
- Isolate & characterise the toxin or toxins – see Colegate & Molyneux (1993) for detailed discussions of approaches and methods

    - ❖ Side issue: Exploit the bioactivity & therapeutic potential of toxin(s) for research funds generation
- Develop analytical techniques for the toxin(s)
- Determine the natural variation in toxin concentrations in the source plants
- Determine the natural variation in toxicity in the target animal species
- Develop management strategies to prevent or reduce poisoning incidents using the data gathered. These may include
    - ❖ controlled access of susceptible animals to hazardous plants at times when the hazard is minimal, for example seasonal grazing of pastures infested by toxic weeds
    - ❖ use of natural or engineered ruminal bacteria to detoxify hazardous plants
    - ❖ development of immunogens to protect susceptible animals
- Develop diagnostic tests for live affected animals and for necropsy investigations

The most effective way of carrying out this type of research is to use multi-discipline teams of scientists comprising mainly chemists and veterinarians with input from other disciplines such as botanists, immunologists and rumen ecologists. Prime examples of such team were those at Murdoch University in Western Australia that isolated the toxins swainsonine, stypandrol and iforrestine (Dorling *et al.* 1993), the USDA Agricultural Research Service Poisonous Plants Research Laboratory at Logan, Utah (James 1994) and the looser associations of scientists represented by the Queensland Poisonous Plants Committee (McKenzie 1995).

References

Colegate SM, Molyneux RJ (eds.) (1993) *Bioactive Natural Products. Detection, Isolation, and Structural Determination.* CRC Press, Boca Raton, Florida. 528 pp. ISBN 0 8493 4372 0

Dorling PR, Colegate SM, Huxtable CR (1993) Plants affecting livestock: an approach to toxin isolation. Chapter 21 in Colegate SM, Molyneux RJ (eds.) *Bioactive Natural Products. Detection, Isolation, and Structural Determination.* CRC Press, Boca Raton, Florida. pp. 481-506.

James LF (1994) Solving poisonous plant problems by a team approach. Chapter 1 in Colegate SM, Dorling PR (eds.) *Plant-Associated Toxins. Agricultural, Phytochemical and Ecological Aspects.* CAB International, Wallingford, Oxon UK. pp.1-6.

McKenzie RA (1995) The Queensland Poisonous Plants Committee: its history and functions. *Aust. Vet. J.* **72**:10-17.

Which are the most important poisonous plants and mycotoxins in Australia?

This issue has been addressed for plants and mycotoxins poisonous to livestock for economic reasons. The lists of plants & mycotoxins generated will vary in extent and ranking with the perspective adopted, the geographical region surveyed and with time.

Focus of study	Important plants/toxins	Reference
Food safety & trade risks from contamination of Australian animal products	Pyrrolizidine alkaloids Corynetoxins Aflatoxins Phomopsins Ochratoxins Fumonisins Trichothecenes (particularly DON, nivalenol) Ergot alkaloids Alternarial toxins (particularly tenuazonic acid) Zearalenone Patulin Ptaquiloside Shellfish biotoxins Ciguatoxin	SCARM Working Group on Natural Toxins, Report, July 2000
Death & production losses (Australia-wide)	Phomopsins Pyrrolizidine alkaloids (*Heliotropium*) Phyto-oestrogens (*Trifolium subterraneum*) Corynetoxins *Lantana camara* *Phalaris aquatica* *Pimelea* spp. (St.George disease)	Culvenor (1985)
Death & production loss (Queensland)	*Lantana camara* *Cenchrus ciliaris* (Equine NSH) *Pimelea* spp. (St.George disease) Fluoroacetate (*Acacia georginae*) Sawfly larval poisoning	Culvenor (1985)

Focus of study	Important plants/toxins	Reference
Death & production loss (New South Wales)	Pyrrolizidine alkaloids *Ixiolaena brevicompta* *Phalaris aquatica* *Pimelea* spp. (St.George disease) *Tribulus* spp.	Culvenor (1985)
Death & production loss (Victoria)	Pyrrolizidine alkaloids Ptaquiloside (*Pteridium esculentum*) Sporidesmin Phomopsins Nitrate-nitrite *Phalaris aquatica* Lolitrems Phyto-oestrogens (*Trifolium subterraneum*)	Culvenor (1985)
Death & production loss (South Australia)	Corynetoxins Pyrrolizidine alkaloids Cardiac glycosides (*Homeria* spp.) Phomopsins Oxalates (*Oxalis pes-caprae*) *Phalaris aquatica* *Pimelea* spp. (Marree disease) Phyto-oestrogens (*Trifolium subterraneum*)	Culvenor (1985)
Death & production loss (Western Australia)	Corynetoxins Phomopsins Sporidesmin Phyto-oestrogens (*Trifolium subterraneum*)	Culvenor (1985)
Death & production loss (Tasmania)	Ptaquiloside (*Pteridium esculentum*) Lolitrems Pyrrolizidine alkaloids	Culvenor (1985)
Significant risks to livestock production (Queensland)	*Lantana camara* Pyrrolizidine alkaloids (*Crotalaria, Senecio, Heliotropium*) Ptaquiloside (*Pteridium, Cheilanthes*) Cycads (*Cycas, Macrozamia*) *Pimelea* spp. (St.George disease) Fluoroacetate *Erythrophleum chlorostachys* Oxalate Nitrate-nitrite Cyanogenic glycosides Cardiac glycosides (*Bryophyllum* spp.) *Trema tomentosa* Carboxyatractylosides (*Cestrum, Wedelia, Xanthium*) *Indigofera linnaei* Swainsonine (*Swainsona, Ipomoea*) *Ixiolaena brevicompta* Thiaminase (*Marsilea*) *Myoporum* spp. Hydrolysable tannins (*Terminalia*) Galegine (*Verbesina*) *Ageratina adenophora* *Xanthorrhoea* spp.	McKenzie (1991)
Frequency & intensity of poisoning reports – first & second rank plants (Northern Territory)	**First rank:** *Cycas* spp. *Erythrophleum chlorostachys* Fluoroacetate (*Acacia georginae*) *Indigofera linnaei* **Second rank:** Pyrrolizidine alkaloids (*Crotalaria*) Cyanobacteria Oxalate Nitrate Swainsonine (*Swainsona, Ipomoea*) Thiaminase (*Marsilea*) *Atalaya hemiglauca* *Isotropis atropurpurea* *Trachymene* spp.	McKenzie *et al.* (1995)

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Control of plant poisoning

Approaches to control of poisoning (McKenzie 1991) include

- **promoting knowledge** in owners of animals of the potentially poisonous plants and circumstances of poisoning in their region
- general management:
  - **prevent access** to plants under circumstances likely to lead to poisoning
  - **provide adequate nutrition at all times** as the basis for preventing the eating of many less-palatable poisonous species
- **specific management strategies**: currently available for *Swainsona* spp., *Leucaena leucocephala* in ruminants, *Indigofera linnaei* in horses. Others could be devised from study of the epidemiology and pathophysiology of poisonings.
- **therapy** (usually unrewarding) can be helpful in some cases such as cyanide, nitrite, thiaminase, oxalates, cardiac glycosides, *Lantana camara.* Cyclodextrins are promising candidates for sequestration of toxin molecules (Edgar 1998)
- **plant control** (see below)
- **immunisation**: restricted to a small number of candidate toxins (McKenzie 1994, Edgar 1998)
- **manipulation of rumen flora** (*q.v.*) to detoxify ingested plants. There has been limited success against mimosine and fluoroacetate and hopes of success against pyrrolizidine alkaloids (Edgar 1998).
- creation of **conditioned aversion** in susceptible animals to particular toxic plants in their environment (Ralphs & Olsen 1998).

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Control of poisonous vascular plants

Methods applicable to weedy species

Effective methods will vary with species, location, habitat, population density and other factors

- Physical removal: hand pulling/cutting, ploughing
- Fire
- Herbicides
- Grazing with non-susceptible herbivores: goats (Allen *et al.* 1993, Simmonds *et al.* 2000), sheep, heavy stocking rates → individual plant intake below the toxic dose
- Biological control using insects and microbial pathogens (Menz *et al.* 1984, Julien & White 1998): applicable to only exotic, naturalised plant species; complex screening procedures to ensure that introduced agents (microbes, insects) are specific to the intended target; unforseen non-target effects are an increasing concern (Hamilton 2000)

Biological control of weeds (Julien & White 1997):
Biological control aims to introduce natural insect herbivores, microbial pathogens or both for a target weed that will reduce the population density of the weed to a size that is acceptable and to maintain the population density at that size. Two techniques of biological control are used – classical and non-classical.

Classical biological control is the most commonly employed technique and involves introduction of natural "enemies" of the target weed from their native range into an exotic range where the host plant has become a weed.
Non-classical biological control can be divided into *inundative* and *augmentative* techniques. Inundative control involves release of large numbers of the agent, such as fungal pathogens acting as mycoherbicides, to control target weeds. Augmentative control involves mass rearing and release of large numbers of a control agent that cannot be grown *in vitro*.
Steps in a biological control program are

- Initiation: review the literature and compile existing knowledge about the target weed and its natural "enemies"
- Approval to work: seek approval and funds
- Foreign exploration: locate the native range of the target weed and search for its natural "enemies".
- Survey the exotic range of the weed: survey fauna attacking the weed and determine their origin
- Ecology of weed and natural "enemies": study the weed and its natural "enemies" including their host ranges
- Host specificity studies: prepare lists of test plants and conduct host testing trials
- Approval of agents: submit reports of host testing to appropriate authorities to obtain approval to release
- Importation: obtain certified clean material or eliminate parasites and pathogens before release
- Rearing and Release: mass rear and make field releases
- Evaluation: field studies to determine establishment spread and effect on target weed
- Distribution: distribute the agents widely; collaborate with other institutions

Biological control of weeds

- is environmentally friendly: reduces the use of herbicides and thus reduces environmental contamination and health risks to primary producers and weed control specialists
- is relatively cheap
- is self-sustaining
- is useful for weeds that cannot otherwise be controlled such as environmental weeds invading national parks and nature reserves
- takes 5-10 years to achieve control
- requires government support
- generally cannot be sold and thus does not attract industry support (except mycoherbicides)
- is unsuitable for rapid short-term control, such as in cash crops

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4: Introduction to Mycotoxin Poisoning

☑ What is a mycotoxin?

Mycotoxin are low molecular weight, non-antigenic chemicals produced by fungi. The fungi usually considered as sources of mycotoxins are the **mould fungi** – those that act as agents of plant decay – but also include the **endophytic fungi** – the cryptic and symbiotic inhabitants of many plants – and the **ergot fungi** – parasites of grass seeds. The toxins produced by mould and ergot fungi may be used by the toxigenic fungus as a defense mechanism against other microbes in the same nutrient source. Mycotoxins produced by endophytic fungi may help the host plant resist insect attack. While ergot fungi and their toxicity have been known for centuries (Matossian 1989), other mycotoxins were first recognised in 1960 through aflatoxicosis of turkeys (turkey X disease) in England and trout in California (Culvenor 1974). Currently over 300 mycotoxins are recognised as capable of intoxicating mammals (Fink-Gremmels 1999). The **major genera** of mycotoxin-producing fungi are ***Aspergillus, Claviceps, Diaporthe, Fusarium, Neotyphodium, Pithomyces*** and ***Penicillium***.

☑ Where do mycotoxins & mycotoxicoses occur?

Toxigenic fungi with impact on humans and animals of significance to them grow on or in various substrates, usually

- **standing crops** (e.g. lupins, maize)
- **particular pasture species** (e.g. ryegrass, tall fescue)
- **stored feeds** (e.g. maize, sorghum, peanuts, oilseeds, bread, feed pellets, dry dog food)

Mycotoxins in stored feeds cause disease in pigs, poultry, dairy & feedlot cattle. Those in pastures cause disease in grazing animals. Perhaps 25% of the world's crop production is contaminated to some extent by mycotoxins (Fink-Gremmels 1999). The situation in feed in the USA is reviewed by Meronuck & Xie (1999).

In **Australia**, mycotoxicoses of animals *from stored feed* are uncommon and have only a small impact on animal health. Pasture and standing crop-associated mycotoxicoses are more important. Culvenor (1974) and Blaney (1996) have reviewed the mycotoxin-producing fungi in Australia and their effects on animals. Hocking & Pitt (1996) have reviewed mycotoxin-producing fungi and mycotoxins in human foods in Australia.

Suitable **growth conditions** are required (temperature & moisture content) for mycotoxin production by fungi e.g. *Aspergillus flavus* produces toxins best at 25-32^{o}C. Factors affecting mycotoxin accumulation in storage include (Abramson *et al.* 1992)

- moisture
- temperature
- time
- intergranular oxygen levels
- mechanical damage to the grain
- invertebrate vectors (e.g. grain beetles. weevils)
- composition of substrate
- fungal abundance
- prevalence of toxigenic strains
- spore load
- microbiological interactions

Variation in substrate & growth condition requirements for particular fungi lead to a **regional distribution of mycotoxicoses** e.g. T-2 trichothecene poisoning from over-wintered grain in cold climates

The **severity of mycotoxin contamination of feedstuffs** is heavily influenced by major environmental factors (Meronuck & Xie 1999) including

- excessive moisture content in the field and in storage
- temperature extremes
- humidity
- drought
- variations in harvesting practices
- insect infestations

Which mycotoxins and mycotoxicoses occur in Australia?

The table below is based on that of Blaney and Williams (1991a) and contains additional data

Mycotoxin recorded from Australia	Fungal source	Prevalence in Australia	Natural case(s) of toxicosis recorded	References
Aflatoxins	*Aspergillus flavus* *Aspergillus parasiticus*	Common in peanuts; uncommon in sorghum and maize; occasionally in other grains, oilseeds and mixed feeds	Yes	Blaney 1984, 1985; Bryden *et al.* 1975, 1980; Connole *et al.* 1981
Cyclopiazonic acid	*Aspergillus flavus*	Unknown naturally; produced in culture	No	Blaney *et al.* 1989
Ochratoxin A	*Aspergillus ochraceus*	Occasionally in maize and mixed feeds	No	Connole *et al.* 1981; Ketterer *et al.* 1982
Lolitrems	*Neotyphodium lolii*	Common in perennial ryegrass pastures, mostly in southern Australia	Yes	
Fumonisins	*Fusarium moniliforme*		Yes	
Zearalenone	*Fusarium graminearum*	Common in maize and sorghum in wetter regions; occasionally in wheat and triticale	Yes	Blaney *et al.* 1984a,b, 1986, 1987; Williams *et al.*1986
Deoxynivalenol	*Fusarium graminearum* Groups 1 and 2	Occasionally in wheat and triticale	Yes	Blaney *et al.* 1987; Moore *et al.* 1985; Tobin 1988
Nivalenol and derivatives	*Fusarium graminearum* Group 2	Common in maize in northern Queensland	No	Blaney & Dodman 1988, unpublished data
T-2 and HT-2 toxins	*Fusarium* spp.	Unknown naturally; produced in culture	No	Blaney, Dodman, Tyler & Moore, unpublished data
Cytochalasins H and J	*Phomopsis longicolla*	Unknown naturally; produced in culture	No	Allen *et al.* 1989
Phomopsins	*Diaporthe toxica*	Common in lupin stubble & seeds	Yes	Wood & Petterson 1986
Sporidesmin	*Pithomyces chartarum*	Sporadic in pastures in southern Australia	Yes	Walsh 1966; Gardiner & Nairn 1962
Ergot alkaloids	*Claviceps purpurea* *Claviceps africana* *Neotyphodium coenophialum*	Sporadic in pastures and grains contaminated with annual ryegrass seed; common in sorghum in northern Australia	Yes	Connole & Johnston 1967;
Paspalitrems	*Claviceps paspali*	Common in *Paspalum* spp. pastures	Yes	Noble 1985
Alternariols	*Alternaria alternata*	Common in sorghum, occasionally in wheat	No	Bryden *et al.* 1984; Blaney *et al.* 1987; Williams *et al.* 1986
Altertoxins, Altenuene, Tenuazonic acid	*Alternaria alternata*	Unknown naturally; produced in culture	No	S. Andrews, personal communication to Blaney & Williams 1991a
Penitrem A	*Penicillium crustosum*	Mouldy garbage (consumed by dogs)	Yes	Hocking *et al.* 1988
Patulin	*Penicillium expansum*	Apple juice	No	G. Fazekas, personal communication to Blaney & Williams 1991a
Viriditoxin	*Paecilomyces varioti*	Unknown naturally; produced in culture	No	Green *et al.* 1989

Mycotoxin recorded from Australia	Fungal source	Prevalence in Australia	Natural case(s) of toxicosis recorded	References
Diplodia maydis toxin	*Diplodia maydis*	One suspected natural case (Darling Downs, Q)	Presumed yes	Darvall 1964

How important are mycotoxins & mycotoxicoses of domestic animals in Australia?

Blaney (1986) estimated that moulds and mycotoxins cost the animal industries in Queensland $2-5 million annually (1986 values) from deaths and lowered production.

☑ *Preventing mycotoxin production in stored feeds - general principles*

- **store at low moisture content**; dry before storage if required
- prevent weather damage / **wetting** of stored feed
- prevent **physical damage** to feed grains during harvest and drying processes; damaged kernels are more susceptible to fungal infection
- prevent **insect infestation**; insects are the commonest cause of damage to kernels and these are more susceptible to fungal infection

A discussion of aspects of prevention of mycotoxin production in feed grains in South-east Asia is provided in Highley & Johnson (1996)

☑ *A caution on diagnosing mycotoxicoses*

- Toxigenic strains of fungi do not produce toxins in clinically significant amounts unless growth conditions are suitable, so **isolation of a known mycotoxin-producing fungal species from a feed source is not, of itself, diagnostic of the mycotoxicosis**
- Mycotoxins are often unevenly distributed in a feed source, so **failure to detect a mycotoxin does not rule it out as a diagnosis**. The most contaminated parts of the feed should be sampled. The most important step in assay of feed for mycotoxins is not the assay itself, but the method used to provide the sample for analysis.
- The availability of assays for mycotoxins is **not** universal in diagnostic laboratories and should not be assumed. Enquire at your local laboratory.

☑ *General effects of mycotoxins on animals*

Subclinical effects have the most economic impact. Clinical effects are "the tip of the iceberg".

- reduced growth efficiency
- lowered feed conversion rates
- lowered reproductive rates
- impaired resistance to infectious disease
- reduced efficiency of vaccinations
- pathological damage to organs (liver, kidney, *etc.*)

☑ *Managing mouldy feeds*

Not all mouldy feeds contain mycotoxins dangerous to animals. Methods for utilising mouldy feeds are available (see Chapter 2) (Blaney & Williams 1991 a,b). Blaney (2000) reviews mouldy feed utilisation in the poultry industry. Wilkinson (1999) reviews animal health concerns from mouldy silage.

Blaney & Williams (1991a) suggest the following general approach to using mouldy feeds:

- ❖ Take representative samples for analysis of nutrients, mycotoxins and fungi present to best assess feeding options.
- ❖ Discard visibly-mouldy and caked feed (bearing in mind the health risks of human exposure) and mix and dry the remaining feed well before use.

- ❖ Include damaged feed at lesser rates initially, feeding it preferably to non-breeding older animals.
- ❖ Consider the relative susceptibilities of animal species and age groups when allocating such feed: fish, ducklings and turkeys are often most susceptible; pigs, calves and fowls are intermediate; adult ruminants are often the most tolerant.
- ❖ Attempt to lessen the effect of "off" aromas and flavours with sweeteners such as molasses.
- ❖ Consider using adsorbents such as alumino-silicate clays (bentonite, zeolite) to reduce absorption of mycotoxins.
- ❖ Do not feed moulded feeds to animals within 2 weeks of slaughter to reduce the risk of carcase residues.

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