

Problems in livestock grazing weeds.

Luzia Rast

Senior Regional Animal Health Manager-Southern Slopes,

NSW Department of Primary Industries, Wagga Wagga Agricultural Institute, Pine Gully Road, Wagga Wagga, NSW 2650.

Introduction

If the definition of a weed is "a plant that one does not want or desire", there are many that can adversely affect livestock. They can include crop and pasture plants such as sorghum, forage brassicas, lucerne, lupins, ryegrass as well as the more commonly known weeds such as Paterson's curse, St. John's wort, heliotrope or fireweed.

The toxic effects of these plants cause three major syndromes in livestock:

- i) Stagers;
- ii) Sudden (or almost sudden) deaths;
- iii) Photosensitisation (sensitivity to sunlight) and hepatopathies (disease of liver).

The above listed syndromes are the most commonly encountered on farms, however there are still a wide variety of weeds that can cause other miscellaneous syndromes.

Little quantitative research and information is available on the short and long-term effects of weed consumption on productivity of livestock. Once there are obvious clinical signs or even deaths, ingestion of toxic plants may have been occurring for some time and it is then very difficult to retrospectively measure

production losses. Furthermore, subclinical effects are very difficult to quantify in livestock.

Plants/weeds that cause stagger syndrome

Stagers means a dysfunction of the locomotor system. This can result from nervous, muscular or metabolic disorders caused by toxic weeds or plants (Table 1). Signs of stagers in livestock are typically, knuckling over, paresis (weakness of limbs), falling, tremors and convulsions. Diagnosis is often difficult and mostly reliant on observation rather than on laboratory tests.

Observable symptoms can occur in days (eg stagger weed, phalaris) to months (eg *Tribulus* spp.) after grazing the offending plant. Effects of these weeds on livestock vary. Most stagger syndromes resolve once livestock are removed and prevented access to the offending weed or plant and given time to recover. In other cases the effect can be permanent or cause death, although death is mostly through misfortune (i.e. *T. terrestris*). The growth stage of the weed also affects toxicity (ie stagger weed is most poisonous to livestock as a seedling) as does the age and species of livestock (i.e. cattle and horses are not affected by *tribulus*, while merino sheep are less susceptible than

Table 1 Weeds that cause stagers in livestock.

Stagger symptoms	Plant (common name)	Plant (botanical name)
Weakness (paresis) of the limbs	Billy buttons, marshmallow, wild parsnip, cat head, grasstree; various peas, Oxalate plants	<i>Ixolaena brevicompta</i> , <i>Malva parviflora</i> , <i>Trachymene ochracea</i> , <i>Tribulus terrestris</i> , <i>Xanthorrhoea</i> spp., <i>Lathyrus</i> spp., <i>Oxalis</i> spp.
Weakness (paresis) of the limbs with knuckling	Onion grass, fenugreek, stagger weed, branched onion weed, yellow vine, cycad leaves, nodding blue lily, weir vine	<i>Romulea rosea</i> , <i>Trigonella foenum-graecum</i> , <i>Stachys arvensis</i> , <i>Trachyantra divaricata</i> , <i>Tribulus micrococcus</i> , <i>Macrozamia</i> spp., <i>Stypandra glauca</i> , <i>Ipomea</i> spp.
Falling	Darling pea, Narrawa butt	<i>Swainsona</i> spp., <i>Solanum cinereum</i>
Falling and tremor	Broadleaf lupin, tree lucerne, phalaris, perennial ryegrass, sorghum, paspalum infected with the fungus <i>claviceps paspali</i>	<i>Lupinus albus</i> , <i>Chamaecytisus proliferus</i> , <i>Phalaris</i> spp., <i>Lolium perenne</i> , <i>Sorghum</i> spp., <i>Paspalum</i> spp. infected by <i>claviceps paspali</i>
Convulsion	Nardoo fern, rock/mulga fern, bracken, tobacco, nightshade, white cedar, thornapple, beard grass, annual ryegrass, blow away grass infected by the fungus <i>Rathayibacter toxicu</i>	<i>Marsilea drumondii</i> , <i>Cheilanthes seiberi</i> , <i>Pteridium esculentum</i> , <i>Nicotiana</i> spp., <i>Solanum</i> spp., <i>Melia azedarach</i> , <i>Datura</i> spp., <i>Polypogon monspeliensis</i> , <i>Lolium rigidum</i> , <i>Agrastis avenacea</i> infected by <i>Rathayibacter toxicus</i>

British and cross breeds). Some specific examples of stagger syndrome include:

Tribulus stagers

Sheep grazing cat head (*T. terrestris*) or yellow vine (*T. micrococcus*) can develop weakness of the limbs. Many months of grazing on these weeds are required before symptoms develop. It is important to note that sheep affected by *T. terrestris* may not show signs until some time after ceasing to graze the weed, but nevertheless will not recover and generally, will eventually die of misfortune. In contrast sheep affected by *T. micrococcus* can fully recover within a few weeks after ceasing to graze the weed. There is variation between sheep breeds in their susceptibility to developing tribulus stagers. British breed and crossbred sheep are more severely affected than pure merinos. The condition has not been reported in cattle or horses.

Stagger weed (*Stachys avensis*)

Prior application of knockdown herbicide increases the risk of toxicity of stagger weed to livestock. All herbivores are susceptible, however younger stock are more susceptible, particularly those still feeding on their mother. Plants are most toxic at the seedling growth stage which usually occurs in late November to January. Stagger signs usually start about seven days after access. Recovery usually occurs within a few weeks of removing stock from the weed.

Phalaris stagers

Only ruminants are susceptible to phalaris stagers. Animals grazing pastures containing the phalaris species *P. aquatica*, *P. minor*, *P. arundinacea* and/or *P. canariensis* can be affected. Sheep and cattle are affected in slightly different ways. Sheep generally develop weakness of the limbs and falling with tremors. In addition to this, cattle also develop chewing and swallowing problems and therefore eventually show weight loss.

Stagers is caused by indole alkaloids in the plants. Sufficient cobalt in the diet can combat the effect of alkaloids. Cobalt allows the rumen flora to 'neutralise' the toxin into harmless compounds. Hence sufficient cobalt in the diet can contribute to a reduction in the incidence of phalaris stagers.

Soils derived from basalt parent material are generally higher in cobalt than soils originating from other parent material. Therefore the prevalence of phalaris stagers tends to be lower on soils derived from basalt. Legumes contain higher levels of cobalt than many other pasture species. Adequate legume content in phalaris pastures can assist in alleviating the incidence of phalaris stagers. The indole alkaloid content in phalaris is highest in autumn and lowest in

late winter/early spring. On the other hand soil cobalt availability is highest in autumn and lowest in late winter. Outbreaks of stagers typically occur in late winter/early spring.

Stock may have had access to the plants for only a few days or several months before signs become apparent. Phalaris stagers is an example of a health problem where several factors need to interact simultaneously for clinical effects to occur (soil type, plant growth stage, stock species, season, pasture plant combination).

Phalaris stagers if known to occur in certain paddocks can be managed in several ways including increasing the content of pasture species other than phalaris, supplementing stock with cobalt (slow release pellets once a year in autumn/late summer) or using a small test group of animals to determine if the paddock is likely to cause phalaris stagers.

Plants or weeds that cause sudden deaths

Many plants have the potential to cause sudden death in large numbers of individuals in a herd or flock (Table 2). This provides a dramatic situation for the farmer, attending veterinarian or advisor when faced with large numbers of dead livestock that were apparently normal 24 to 48 hours earlier. Fortunately, plants that do have this potential rarely cause large outbreaks because the plants are either not palatable or not present in large enough quantities to affect large numbers of animals. Some plants only are toxic in some seasons of some years because several environmental factors are required to occur at the same time to make the plant toxic or grow in the first place (eg blue-green algae).

Ingestion of these plants causes relatively quick death in livestock. Death is caused due to toxic effects on the nervous, cardiovascular, respiratory or gastrointestinal system. Identifying the signs preceding death, can give vital clues in identifying the plants responsible. Even if animals are found dead, observations such as signs of struggle or presence of diarrhoea can give an indication of the likely cause.

Numerous garden plants may also cause sudden death but are too numerous to list in this paper. They only become a problem when trimmings are thrown over the garden fence into livestock paddocks, or if stock accidentally gain access to gardens. Examples of sudden death syndrome include:

Nitrate poisoning

When it rains after extended dry periods, plants can accumulate significant quantities of nitrate. Once ingested, the rumen microflora converts nitrates to

Table 2 Weeds that cause death in livestock.

Signs preceding death	Plant (common name)	Plant (botanical name)
Nervous signs	Phalaris, annual ryegrass, blown grass, common thornapple, tobacco bush, deadly nightshade, corkwood, blue-green algae, white cedar berries, grass dominant lush pasture.	<i>Phalaris aquatica</i> , <i>Lolium rigidum</i> , <i>Agrostis avenacea</i> , <i>Datura stramonium</i> , <i>Nicotiana glauca</i> , <i>Atrapa belladonna</i> , <i>Duboisia</i> spp., <i>Anabaeba</i> spp., <i>Melia azedarach</i>
Cardiac signs	Phalaris, cotton bush, cape tulip, tufted honey flower, mother of millions	<i>Phalaris aquatica</i> , <i>Gomphocarpus</i> spp., <i>Homeria</i> spp., <i>Melanthus comosus</i> , <i>Bryophyllum</i> spp.
Gastrointestinal signs	Kikuyu grass, castor oil seeds, red camomile, legumes	<i>Pennisetum clandestinum</i> , <i>Ricinus communis</i> , <i>Adonis macrocarpa</i> (check species name possibly <i>microcarpa</i>)
Liver damage signs	Bathurst burr seedlings, noogoora burr seedlings, green cestrum, rough dogs tail, blue-green algae	<i>Xanthium spinosum</i> , <i>Xanthium occidentale</i> , <i>Cestrum parqui</i> , <i>Gynosurus echinatus</i> , <i>Microcystis aeruginosa</i>
Respiratory signs	Prickly lettuce, crown beard, cyanide releasing plants, nitrate accumulating plants, ergot of rye	<i>Lactuca serriola</i> , <i>Verbesina encelioides</i> ,

nitrites and then ammonia. If the rumen is gradually exposed to rising nitrate levels then it can adapt and large nitrate loads can be safely handled. This is why nitrate poisoning most commonly occurs when hungry stock are put into a small paddock or yard that contains large quantities of nitrate accumulating plants (especially young stalky plants).

Nitrate poisoning affects oxygen transport and signs include many deaths, acute respiratory distress, diarrhoea, inability to stand (recumbency) and nervous signs. Horses and pigs are not susceptible.

Due to the sudden onset, death usually occurs before treatment can be given.

Cyanide poisoning

Many plants can accumulate cyanide, but sorghum, lotus and linseed plant families are the most common offenders. Ruminants are unable to adapt to the high levels of cyanide accumulated in offending plants as they can only detoxify cyanide at a fixed rate. Therefore if large amounts of cyanide are ingested, the animal is unable to detoxify the additional cyanide and will die. Ruminants are more susceptible to cyanide poisoning than horses and pigs.

Signs include many deaths, acute respiratory distress, inability to stand and nervous signs.

As with nitrate poisoning, due to the rapid progression to death after ingestion, treatment rarely can be given in time.

As a rough guide, plant levels of cyanide up to 200 mg/kg dry matter are fairly safe, 200–600 mg/kg dry matter is safe if fed with caution, >600mg/kg dry matter is very risky or considered unsafe.

Weeds or plants causing photosensitisation and hepatopathies (liver diseases)

Photosensitisation is the development of abnormally heightened reactivity of the skin to sunlight. Photosensitisation in livestock caused by plants may occur in response to the plant, or as a result of damage caused to the liver by the plant.

Photosensitisation is caused by compounds that circulate in the blood vessels of the skin that can become activated by sunlight. These compounds are slowly metabolised in all species and hence cause problems. Susceptibility varies within and between livestock species. An individual's susceptibility to photosensitisation will depend on its ability to prevent sunlight from reaching the skin. This is affected by skin thickness, skin density, length and density of fibre cover and the degree of skin pigmentation.

The most common photosensitising compound is phylloerythrin formed from ingested plant chlorophyll. Once the compound is activated it causes cellulitis of the skin which results in swelling and intense irritation.

Liver diseases can be acute (causing deaths) or chronic (causing ill thrift). Acute liver disease in cattle can lead to brain disease (encephalopathy). If this is the case, nervous signs will dominate (i.e. depression, aimless wandering, reduced awareness, aggression). One exception is a chronic liver disease that causes liver copper accumulation and then sporadic deaths. A wide range of plants can cause photosensitisation and associated liver disease (Table 3). Photosensitisation and liver disease examples include:

Table 3 Weeds that cause photosensitisation and/or liver disease in livestock.

Syndrome	Plant (common name)	Plant (botanical name)
Photosensitisation	St John's wort, buckwheat, barrel medic, burr medic, Persian clover, lucerne, birds foot trefoil, forage brassica, brassica, alligator weed, biserrula, members of the Apiaceae family including celery, carrot, parsley, parsnip	<i>Hypericum perforatum</i> , <i>Fagopyrum esculentum</i> , <i>Medicago truncatula</i> , <i>Medicago polymorpha</i> , <i>Trifolium resupinatum</i> , <i>Medicago sativa</i> , <i>Lotus corniculatus</i> , <i>Brassica</i> spp., <i>Alternanthera philoxeroides</i> , <i>Biserrula pelecinus</i> ,
Liver disease leading to photosensitisation	Cat head and a second factor, blue-green algae, lantana, sweet grass, hairy panic	<i>Tribulus terrestris</i> , <i>Microcystis aeruginosa</i> , <i>Lantana camara</i> , <i>Panicum gilvum</i> , <i>Panicum effusum</i>
Liver disease without photosensitisation	Green cestrum, Paterson's curse, Bathurst and noogoora burr, heliotrope, rattlepods, fireweed, cycad fruits	<i>Cestrum parqui</i> , <i>Echium plantagineum</i> , <i>Xanthium</i> spp., <i>Heliotropium</i> spp., <i>Crotalaria</i> spp., <i>Senecio madagascariensis</i> , <i>Macrozamia</i> spp.

Paterson's curse (*Echium plantagineum*)

Paterson's curse is a common plant in cultivated paddocks, herbicide treated paddocks, overstocked pastures or fire damaged pasture in central and southern NSW.

Alkaloids (pryrolizidine alkaloids, PA) are produced in all parts of the plant including seeds but are highest in young plants.

Ingestion of PA will cause chronic liver disease. Chronic liver disease in cattle and horses can lead to the development of nervous signs as the liver becomes so damaged that it is unable to remove toxic substances from the blood. These toxic substances then impair brain function. The condition is then known as hepatic encephalopathy. Once this stage of disease is reached stock do not recover.

In sheep, chronic liver disease can lead to large amounts of copper being accumulated in the liver. If liver damage becomes severe, large amounts of copper can be released into the bloodstream and results in sudden death of the animal. Sheep with compromised livers are more susceptible to accumulating excessive amounts of copper when they are on legume dominant pasture.

In all species chronic liver damage will lead to ill thrift.

Other plants such as heliotrope, fireweed, amsinckia (*Amsinckia* spp.) and rattlepods can all produce PA causing the same conditions as described above.

Merino and goat wethers are least susceptible to PA poisoning and can be used to control these weeds, but the groups used should be changed every 2-3 years.

Spray grazing is a common management method used to control Paterson curse. While some herbicides increase the palatability of weeds (eg Paterson's curse, tribulus), their toxin content remains and hence the risk to livestock increases, because larger quantities are ingested as plants become more palatable.

This method of weed control needs to be carefully

managed; only stock destined for slaughter within a relative short time after grazing sprayed paddocks should be used. However, there is also a risk of chemical residues in the carcass and the withholding periods as per label recommendations must be adhered to.

St. John's wort (*Hypericum perforatum*)

This weed commonly grows on steep country along the NSW Great Dividing Range.

St John's wort contains the toxin hypericin. Narrow-leaf wort contains twice as much hypericin as does the broad-leaf wort. The amount of toxin is highest between mid spring and mid autumn. Control of this weed is most economically achieved by strategic grazing with livestock. The most hypericin tolerant animal is a fully pigmented steer, but the most effective defoliators of wort are sheep or goats. The most tolerant sheep are fine wool merino wethers with at least four months fleece growth.

Affected animals experience hyperthermia and severe photosensitisation. It takes around five days for affected animals to unload any ingested hypericin and they require full shade during this recovery period.

Panicum grasses

Panicum coloratum (bambatsi panic), *P. maximum* (guinea grass), *P. miliaceum* (millet panic), *P. gilvum* (sweet or hairy panic) are all capable of causing severe acute liver disease leading to photosensitisation in juvenile sheep and goats. Adult sheep and goats and cattle are less likely to be affected.

Outbreaks of this particular type of liver disease are extremely sporadic because two factors need to be present concurrently before toxicity occurs.

The first factor is a steroidal saponin produced by these plants and the second factor is likely to be an unrelated hepatotoxic (liver damaging) compound produced at the same time by either the plant or by a pasture fungus associated with the plant. Activity

of the second factor seems to be favoured by short rainfall events during otherwise dry summers.

Miscellaneous weeds/plants poisoning syndromes

Some specific examples of plants that cause poisoning syndromes include:

Brassica poisoning

Wild radish (*Raphanus raphanistrum*), canola (*Brassica napus*), and forage brassica, such as chou moellier, cabbage, broccoli and turnips can be poisonous to livestock in some situations.

The risk of livestock poisoning is increased when the plants are flowering and setting seeds, when they have been affected by drought or frost, or when they put on fresh growth after rain.

Brassica syndromes are numerous and include: goitre in young livestock, reproductive problems, digestive irritation, rumen dysfunction, anaemia, respiratory distress, blindness and nervous symptoms. In addition brassicas can also cause photosensitisation, bloat, nitrate poisoning and oxalate poisoning.

To avoid stock poisonings, brassicas should not be grazed during high risk periods.

Desert rice flower (*Pimelea simplex*)

Pimelea grows in north western NSW, southern Queensland and northern parts of South Australia. Around ninety species of pimelea are known to occur in Australia and of these; nine are known to be poisonous.

Poisoning occurs in cattle if animals eat dry or green plants, inhale dried plant fragments or ingest soil containing dry fragments. The green plant is quite unpalatable but stock newly introduced to an area will often ingest the plant.

The poison in pimelea is called simplexin and causes constriction of blood vessels taking blood from vital organs such as lung and heart. Clinical signs include death (from intense diarrhoea) or signs of congestive heart failure after inhalation of dry plant fragments (this syndrome is also called St. George disease). The signs of congestive heart failure are large accumulations of fluid in the brisket and lower jaw (oedematous swelling) as well as ill thrift.

This poisoning is still relatively poorly understood. Some cattle appear to recover if removed from plants and given supportive care.

Rock fern (*Cheilanthes seiberi*) and bracken fern (*Pteridium esculentum*)

Both ferns cause similar poisoning problems. Both produce the toxins thiaminase and ptaquilosides but different populations of plants produce different

amounts of each compound. Therefore the severity of poisoning in livestock due to these ferns may vary significantly from place to place.

Stock generally only eat these ferns if no other feed is available or if the ferns are the only green feed available.

Thiaminase can cause thiamine deficiency which causes aimless wandering, incoordination and staggers in affected sheep and horses (the syndrome is called polyoencephalomalazia) and can occur after several weeks of plant ingestion.

Ptaquilosides are carcinogenic (cause cancer) and destroy bone marrow. Affected animals will bleed and display greater susceptibility to infections. The following syndromes can all be caused by this toxin:

- i) An acute haemorrhagic (uncontrollable bleeding) syndrome: this occurs usually in cattle after several months of ingestion and causes bloody faeces, bloody nose as well as internal bleeding.
- ii) A chronic haemorrhagic syndrome: this also usually occurs in cattle after many months of ingestion and leads to red urine, cystitis (inflammation of the bladder), anaemia (lack of red blood cells and therefore oxygen) and ill thrift.
- iii) Cancer: long-term ingestion has been associated with cancers in the bladder.
- iv) In the UK a retinal degeneration syndrome called 'bright blindness' in sheep has been described.

Conclusion

A multitude of weeds and plants can be toxic to livestock and lead to minor and major production losses. However for severe poisonings to occur, generally a number of factors need to interplay (growth stage of plant, soil type, season, species of livestock ingesting plants and production stage of animals). The need for several factors to occur at the same time, make predicting and hence preventing of plant intoxication in livestock difficult.

Quite often a severe poisoning, leading to high mortality rates may not occur again for a long time period despite the occurrence of apparently similar conditions.

Plant poisoning in livestock is dose dependant. In grazing situations, intake of toxic plants may vary considerably between individuals in a mob of animals. This depends on the amount and location of toxic plants within a paddock as well as the nutritional status and grazing behaviour of individual animals.

Due to the large variability in climatic conditions, soil conditions, plant population and animal grazing behaviour it is firstly, difficult to research and quantify production losses due to poisonings, and secondly, to

make definitive recommendations on management of specific weeds or plants. Often management of toxic weeds depends on local knowledge and experience. Local agronomists and animal health staff can provide advice. Careful and frequent observation of stock during risky periods or grazing on risky pastures is also important

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