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CYANOCENGETIC PLANTS OF WESTERN AUSTRALIA

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This article deals with the more important cyanogenetic plants found in Western Australia. It also deals with plants which have not been proved as cyanogenetic but which do produce toxic effects similar to those produced by cyanogenetic plants. Symptoms, post mortem appearances and the treatment for affected stock are also dealt with.

Cyanogenetic plants release the toxic substance hydrocyanic acid (HCN) or prussic acid when eaten by stock. In Western Australia, cyanogenetic species include both indigenous and introduced plants, many of which are very important to the Animal Industry.

Some other plants are also dangerous, but are less important as HCN producers. Such plants can cause mortalities in stock, especially in hungry stock, on rare occasions. Some of these less important cyanogenetic plants are Acacia dilatata R. Br., Dactyloctenium radulans (R. Br.) Beauv., Digitaria sanguinalis (L.) Scop., Eleusine indica (L.) Gaertn., crowsfoot grass, Eriobotrya japonica Lindl., loquat, Heterodendron oleifolium Desf., rosewood, Linum spp., flax or linseed, Lotus australis Andr. austral trefoil, Passiflora spp., passion-flower, Poranthera microphylla Brongn., Pililotus polystachyus (Gaud.) F. Muell., mulla-mulla, Trema aspera Blume, peach-leaf poison-bush and Zea mays L., maize.

Important cyanogenetic or cyanogenetic-like plants in Western Australia are:

Chenopodium species
Chenopodium blackianum Aellen. C. carinatum R. Br. (keeled goosefoot), C. cristatum F. Muell. (crested goosefoot), C. myriocephalum (Benth.) Aellen and C. rhadinostachyum F. Muell. have been suspected of causing stock mortalities and are cyanogenetic. C. melanocarpum (J. M. Black) J. M. Black (black goosefoot) and C. pumilio R. Br. (common goosefoot) have been suspected of poisoning stock and are possibly cyanogenetic.

Eremophila species
Eremophila maculata F. Muell. This native fuchsia is found from the Gascoyne River eastwards to Wiluna and southwards to Widgiemooltha. It has been suspected of poisoning sheep and cattle and is one of the most strongly cyanogenetic plants known.

Eucalyptus species
Eucalyptus cladocalyx F. Muell. (sugar gum and E. viminalis Labill. (manna gum) are native to Eastern Australia but are cultivated in this State. Sugar gum is commonly planted on farms as a shade tree. Both species are cyanogenetic and have been suspected of causing stock mortalities.
Gascoyne spurge has been incriminated in sheep and cattle losses along the Gascoyne stock route.
Mat spurge has been shown to contain prusiac acid but is not always considered poisonous.
**Euphorbia species**

*Euphorbia boophthona* C. A. Gardn. (Gascoyne spurge) is common along the Gascoyne River and its principal tributaries. It is found as far north as Mt. Herbert in the Hamersley Range and eastwards to Laverton and has been incriminated in sheep and cattle losses along the Gascoyne stock route. Affected animals show symptoms strongly suggestive of HCN poisoning.

*E. clutioides* (Forst. f.) C. A. Gardn. (desert spurge) is found on sandy soils most of northern Western Australia to as far south as Lake Barlee. It is less significant as a toxic plant than Gascoyne spurge but its effects suggest HCN poisoning.

*E. drummondii* Boiss. (mat spurge) is found over much of the more arid region of Western Australia. On occasions it has been responsible for mortalities in sheep and cattle but travelling stock are more often affected. The plant is cyanogenetic although only 12 per cent. of the 900 plants tested chemically have given positive results for HCN.

*E. terracina* L. (false caper or Geraldton carnation weed) is high in HCN and is therefore dangerous to stock, particularly when other feed is scarce. This plant is a declared primary noxious weed for the whole State.

**Clover-leaf poison**

*Goodia lotifolia* Salisb. Commonly known as clover-leaf poison, this plant occurs in the south-west from Manjimup eastwards to beyond Esperance and northward to Kondinin and Lake Grace. It has been involved in stock losses and has yielded dangerous amounts of HCN.

**Sorghum species**

*Sorghum alnum* Parodi (Columbus grass), *S. halepense* (L.) Pers. (Johnson grass), *S. sudanese* Staph (Sudan grass) and *S. vulgare* Pers. (sorghum) are either cultivated or found naturalised but are all cyanogenetic. They have been reported to cause mortalities in stock and *S. alnum* is a declared primary noxious weed for the Ord River irrigation area.

**Sandplain woody pear**

*Xylomelum angustifolium* Kipp. The sandplain woody pear has been shown to contain HCN and has been responsible for mortalities in sheep. Only the sappy young leaves are considered to be harmful.
Geraldton carnation weed or false caper, a primary noxious weed, is dangerous to stock.
Poison sedges

Probably the most important cyanogenetic or cyanogenetic-like plant found in Western Australia is poison sedge (*Schoenus asperocarpus* F. Muell.).

In 1952, poison sedge was reported as probably causing some 200 out of 800 sheep deaths in the Carnamah district. This plant has been suspected of being poisonous to stock as far back as 1892 and stock losses due to poison sedge continue up to the present day. Most losses occur in the Midlands region but some have been recorded from centres as far south as Waroona.

Poison sedge is a tussocky grass-like sedge, with a strong fibrous rooting system. Each clump is roughly 2 to 3 in. across at the base. The leaves are very narrow, almost thread-like, up to 10 inches long, erect, dark green in colour and smooth. The expanded leaf bases form sheaths which are obtusely lobed at the top and chestnut to reddish brown in colour.

The flowering stalks or culms of poison sedge are longer than the leaves, from 15 to 18 in. long, with partial inflorescences or flower clusters borne alternately along the upper part of the culm. Each flower cluster or spikelet is enveloped by shiny brown bracts, which, like the leaf sheaths, are obtusely lobed at the top. When these bracts are removed the axis of each spikelet is seen to have a zig-zag appearance. The pale rounded seeds are somewhat rough in texture and are borne on this zig-zag axis.

Poison sedge grows in both siliceous and calcareous sands from the Murchison River in the north to Margaret River in the south. The species has also been recorded in the Salmon Gums district, southwards of Kalgoorlie. It is often found in slight depressions where a certain amount of subsurface moisture may be present during the dry summer months. Usually it is associated with sand heath formations, containing occasional taller shrubs and trees.

Poison sedge can be eradicated by burning during clearing. In areas where native scrub is present, the burn follows the chaining or rolling of original plant material. Where no scrub is present the sedges are ploughed in at a depth of 4 to 5 in. to get under the root zone of the plants. The area is then root-raked or fire-harrowed to remove fragments of root and dried sedges. These operations clear native vegetation, including poison sedge, and provide conditions suitable for subsequent or pasture establishment.

Other suspect species

*Gyrostemon ramulosus* Desf. (fire bush) has been suspected of causing sheep losses in Western Australia. The plant has given a positive reaction for HCN.

*Haloragis* spp. (raspwort) occur in the south-west and the more arid parts of Western Australia. A species present in New South Wales was found to contain HCN and another species was suspected of causing sickness in lambs in Queensland.

*Loudonia aurea* Lindl. and *L. roei*, Schlecht. (both commonly known as yellow pop-flower) have been suspected of causing sheep losses in Western Australia. Both species have given positive reactions for HCN.

*Prunus armeniaca* L. (apricot), *P. laurocerasus* L. (cherry laurel) and *P. persica* Stokes (peach) have also been shown to be cyanogenetic.
Poison sedge, one of the most important of native cyanogenic species, has caused considerable stock mortalities particularly in the midlands.
Yellow pop-flower. There are two species of Loudonia which are cyanogenetic.

Toxicity of cyanogenetic plants

The toxicity of cyanogenetic plants is determined by a number of factors. Climate is most important. Toxicity generally increases when favourable conditions for plant growth follow unfavourable conditions such as drought or frost. Plants are more toxic in the early stages of development or when making new growth following setbacks. Soil fertility, especially a favourable nitrogen status, increases the toxicity of cyanogenetic plants. Toxicity also varies according to the genetic makeup and some strains of a particular species are more toxic than others.

The HCN bound up in cyanogenetic plants is generally released when the plant is cut and allowed to dry. Thus a toxic standing crop becomes harmless and can provide good fodder for stock, if made into hay.

Cyanogenetic plants normally have their HCN bound to sugar molecules to form chemical compounds known as cyanogenetic glycosides. These compounds are not toxic by themselves, but they may be acted upon in the stomach of the animal by other chemical substances known as enzymes or "keys." This action unlocks the HCN component from the glycoside and leads to symptoms of poisoning and perhaps the death of the animal.

Stock losses following eating of cyanogenetic plants are affected by the quantity and rate of consumption of the plants by the level of cyanogenetic glycoside present, by the presence of appropriate enzymes or "keys" to release the HCN in the stomach, and by the rate at which the HCN is rendered harmless by the animal.

Symptoms

Cyanogenetic plants can cause very rapid death. Hungry or travelling stock are most susceptible to this form of poisoning and stock that are well fed and undisturbed are less likely to be affected. If considerable quantities of toxic material are consumed death occurs very rapidly without the appearance of any symptoms.

Animals suffering from HCN poisoning suddenly develop deep, rapid breathing, bloat and frothing at the mouth. The mucous linings of the mouth and eyes are bright red at first, but rapidly change to a purple-blue colour typical of cyanide poisoning. The breath may have a faint smell of "bitter almonds."

Animals which go down may have their heads turned around towards their flanks. Both sheep and cattle are nearly always bloated in the final stages, and have a short period of convulsions before death.

Animals which have not received a lethal dose recover, although the symptoms may be prolonged. Diarrhoea occasionally results from an irritant gastro-enteritis.

Post mortem

Blood released from blood vessels during an autopsy turns a cherry red colour when exposed to the air. Small haemorrhages are often seen in the lining membrane of the windpipe and congestion and haemorrhages on the lungs and inner linings of the abdominal wall may be seen. In acute cases there may be severe inflammation of the abomasum (fourth stomach) and the first few feet of small intestine. The smell of "bitter almonds" may be detected from the contents of the first stomach.

Treatment

Animals suffering from HCN poisoning are usually given compounds containing sulphur, such as sodium hyposulphite ("hypo") or sulphuric ether, as rapidly as possible. Cattle should be given "hypo" at
the rate of two ounces dissolved in one pint of water. Sheep should be drenched with one-fifth of this amount. The treatment should be repeated in half an hour if the symptoms do not abate.

Bloating may be relieved by puncturing the left flank with a trocar and cannula and subsequently administering the “hypo” directly through the cannula.

Summary

A number of plants are cyanogenetic or cyanogenetic-like in action. These can, on occasions, contain toxic levels of HCN, particularly when in the early stages of development or when making new growth following damage by frost, by drought or by fire.

Graziers and pastoralists can prevent stock losses due to HCN poisoning by eradicating cyanogenetic plants or by deferring grazing of these plants at the time when they are most dangerous to stock. Cyanogenetic plants are not toxic when cut and dried as hay. Hungry and travelling stock are most likely to be seriously affected and should not be allowed to graze cyanogenetic plants until they have fed and rested in safe pastures.

Accidental poisonings due to cyanogenetic plants can occur. Stock owners are therefore advised to have on hand a quantity of sodium hyposulphite (“hypo”) so that immediate treatment may be given to affected stock.

References

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