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POISON SEDGE CAN KILL STOCK

POISON SEDGE was first suspected of being toxic to livestock in Western Australia nearly 80 years ago. Sudden deaths of sheep grazing areas on which poison sedge grew have been reported from many regions from Geraldton to Scott River. This article reports a case of poisoning in the field, and the experimental reproduction of poison sedge toxicity in pen-fed sheep.

By M. E. NAIRN, T. E. H. APLIN, D. S. PETTERSON, and A. J. BRIGHTON

Stock deaths caused by eating poison sedge are not common, but feeding trials at the Animal Health Laboratory confirmed that 210 sheep which died on a Scott River property last summer were killed by poison sedge. Chemical analysis failed to identify the toxic principle.

Poison sedge is thought most likely to be toxic soon after rain has fallen, or after burning.

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

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
Massive accumulation of fluid in the thoracic cavity of a sheep which died after eating 500 grams (about 18 oz.) of poison sedge.

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 the Scott River in the south. It has also been recorded in the Salmon Gums district, south of Kalgoorlie. It favours slight depressions where a certain amount of sub-surface moisture may be present during the summer. It is usually associated with sand heath formations, containing occasional tall shrubs and trees, or with stunted forests.

Poison sedge is a short-lived perennial which usually sets seed in its second year of growth. It is most commonly seen after fires or after the land has been cleared. It may be eradicated by normal clearing and cropping methods and is rarely present in established pastures.

**Farm deaths**

In March, 1970, 210 of 1,000 sheep were found dead on a Scott River property within a few days of being allowed access to newly cleared land containing poison sedge. In some animals there was evidence of acute respiratory difficulty before death. Forced exercise of sheep showing depression resulted in laboured breathing and death.

Post-mortem examination of carcasses revealed large amounts of pale yellow fluid in the thoracic cavity. This fluid tended to clot readily upon exposure to air, indicating that it contained a high level of the plasma protein, fibrinogen. The trachea and bronchi in most animals were filled with large quantities of frothy fluid and the lungs were wet and heavy due to the accumulation of fluid (oedema). There were no signs of damage to other organs.

**Feeding trials**

Because poison sedge was suspected of killing the sheep, plants were taken to the Animal Health Laboratory for a pen feeding trial and chemical analyses. The leafy parts were coarsely chopped and weighed amounts fed to two 2-tooth Merino wethers. One animal died within 24 hours, after eating 285 grams (about ½ lb) of poison sedge. The second wether died after eating 500 grams (just over 1 lb) of poison sedge over 30 hours. Neither animal showed marked respiratory difficulty before death but post-mortem examination revealed frothy fluid in the trachea and massive accumulation of fluid in the thoracic cavity. The experimental disease was therefore considered identical to the field cases.

**Chemical investigation**

Plants were tested for the more common natural compounds which may cause such rapid death. These included cyanide (prussic acid), long thought to be the toxic agent in sedge; fluoroacetate, the toxic principle of *Gastrolobium* and *Oxylobium* species; and alkaloids, which can cause a variety of symptoms before death.

None of these was detected.

Poison sedge plants were still toxic after three days' storage, which suggests that cardiac glycosides, such as those found in the foxglove plant, are unlikely to be the toxic agent in poison sedge.
Other common poisons tested for were oxalates, nitrates and nitrites, none of which was detected.

Following a case of suspected poison sedge toxicity at Carnamah in 1952 it was reported that prussic acid was the probable toxic principle. However, there are no records of plant analyses or detailed post-mortem examinations.

In last year's investigation we were unable to detect prussic acid in poison sedge and the material fed to experimental animals was still toxic several days after collection. This and the presence of thoracic fluid are not consistent with prussic acid poisoning.

It seems that poison sedge is not always toxic, as subsequent feeding of more than 1,000 grams of plant material collected from the Scott River property at a later date failed to induce symptoms of poisoning in two sheep.

It may be significant that the material shown to be toxic was collected not long after rain had fallen. It has also been reported that most poison sedge mortalities normally occur in autumn following the burning of vegetation.

Treatment

Sheep affected by poison sedge should be denied further access to the plant and disturbed as little as possible, to avoid precipitating respiratory distress. There is no known antidote to the poison sedge toxin.