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Annual ('Wimmera') ryegrass toxicity

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Because annual ryegrass is one of the most widespread pasture plants in Western Australia, annual ryegrass toxicity is a threat to livestock in many agricultural areas—even though the area now affected is small.

The problem is being intensively studied by the Department of Agriculture and a working group has been set up to investigate potential control measures.

In this article the group reviews the problem and reports progress in its investigations to the end of 1973.

The members of the group are:

Biological Services Division:
- G. A. Pearce (Convenor)—Senior Adviser, Weed Control, Olga M. Goss—Senior Plant Pathologist.

Animal Division: P. H. Berry—Veterinary Surgeon, Katanning.

Wheat and Sheep Division: J. L. Wise—Agricultural Adviser, Katanning.

"Wimmera ryegrass toxicity" is an unusual condition in which annual ("Wimmera") ryegrass is attacked by a nematode, which infects the grass heads with a bacterium and the mature dry grass becomes toxic to stock.

A yellow bacterial slime may form on the heads and on the leaves ensheathing the heads of infected plants.

The disease was first noticed in livestock about 20 years ago on two properties in South Australia. By 1965 it had been recorded on other properties in the area and stock losses of up to 30 per cent had been attributed to it. Since 1972 it has been blamed for some substantial losses, although it has not spread beyond a 20-kilometre radius from its origin.

In Western Australia the problem was probably first noticed in 1959 on a property in the Katanning area. However, the first confirmed outbreak was in 1968 on a property near Gnowangerup, which had been losing stock since 1964. In the summer of 1970-71, eight properties in the Katanning-Gnowangerup area recorded losses and a total of 450 sheep died. In the following summer 10 properties lost 750 sheep and it is now known that 30 properties in Western Australia have this disease. Both sheep and cattle have been affected.

"WIMMERA" OR "ANNUAL" RYEGRASS?
The annual pasture grass known to farmers in Western Australia as "Wimmera" ryegrass is the species Lolium rigidum, the correct common name of which is annual ryegrass.

At least two cultivars (varieties) of annual ryegrass are found on farms in this State—"Wimmera" and "Merredin early". Both may be involved in the annual ryegrass toxicity problem.

The description "Wimmera ryegrass toxicity" should therefore be "annual ryegrass toxicity" and this name is used in this article, except where the name "Wimmera" is used to ensure identification of the disease.
Nematode gall replaces mature ryegrass seed.

Annual ryegrass toxicity has only been seen on pastures or stubbles containing mature annual ryegrass. Another ryegrass disease is perennial ryegrass "staggers", which is a disease of sheep and cattle grazing green perennial ryegrass, and related to annual ryegrass toxicity.

Ryegrass staggers is confined to high rainfall areas in which perennial ryegrass is grown.

Research
Since 1971 a considerable amount of research has been carried out to isolate the disease organisms and investigate their biology.

The disease in South Australia has been intensively studied at the Waite Agricultural Research Institute by Dr. P. C. Price, who isolated Corynebacterium species bacteria from the yellow bacterial slime on infected annual ryegrass heads.

His studies of the nematode indicated that it was a new species of Auguina for which he proposed the name Auguina lollii. In his experiments nematode larvae readily transmitted the bacterium to ryegrass plants and he considered the nematode an essential vector for the bacterium in the field.

Following a feeding trial with infected material he concluded that "there is considerable evidence that the infection of annual ryegrass by Auguina lollii and/or the Corynebacterium sp. results in the formation of a toxin which causes the neurological disease of stock", and that the toxin was contained in infected ryegrass seedheads.

In Western Australia, the Department of Agriculture's Animal Health Laboratory has grown Corynebacterium bacteria from toxic plant material and is studying the process of toxin development.

Seed taken from a toxic area of mature ryegrass and fed to sheep and guinea pigs has caused typical symptoms and death within about six days. Other animals fed ryegrass seed from non-toxic areas have remained normal and this appears to confirm that the toxic material is in the seedhead of mature ryegrass.

The only practical measures that can be recommended at present to avoid annual ryegrass toxicity concern removal of the ryegrass from the pastures and crops.

Three main approaches to practical control are being investigated in current Department of Agriculture field trials:

- Treatment with a nematicide in an effort to kill the nematode. Trials in 1973 failed to give effective control and earlier treatment is planned for the 1974 season.
- Use of a bactericide to eliminate the bacteria. Chemicals tested in 1973 showed indications of reducing the level of bacterial infection and tests with these will be continued.
- Herbicidal treatments to control the ryegrass. This is the most practical approach available at present. In one trial herbicidal treatment eliminated about 95 per cent of flowering stalks of ryegrass.

Life cycle
Soon after the opening rains when the ryegrass has germinated the nematode larvae begin to be freed in the soil from the nematode galls.

They are very small eel-like organisms which make their way to the young plant. When a film of moisture is present they crawl over the surface of the leaf sheaths and in between them, eventually reaching the growing point. The larvae are freed from the galls over a period of about three months if adequate moisture is present, so that even late tillers may be infected.

The larvae themselves are often infested with a bacterium and introduce it to the plant. The bacterium is apparently not capable of infecting the plant in the field without a nematode vector.

The larvae remain passive at the growing point and are carried up as it develops. They do not feed until the grass begins to flower, when they bore their way into the developing flower. The nematode larvae mature into adult worms within the flower, which fails to develop properly. No anthers are formed and the ovary does not develop to produce a seed, but is replaced by an eelworm gall. Inside the gall the adult worm lays eggs.
which hatch into larvae, which remain inside the gall. Galls differ from normal seeds in both size and shape, being much smaller and pointed, or pear shaped (see diagram). They then remain dormant or inactive until the following season when they again hatch out in the soil.

The bacteria introduced to the plant by the nematode also begin to multiply rapidly at the flowering stage of the ryegrass and the heads and leaves ensheathing the heads may be covered with yellow slime. There are therefore no visible symptoms of attack by the disorder until the flowering stage.

Usually when the bacteria multiply they kill the nematode and prevent development of a new generation of larvae. Although the nematode introduces the bacteria to the plant, it is therefore rare to find a gall containing both organisms.

The animal disease

The disease is characterised by staggering and convulsions and affected animals collapse, convulse for some minutes then may regain their feet and stagger away with a stiff legged “rocking horse” gait. After a few more minutes they may rejoin the flock or mob and become apparently normal.

Signs of the disease are usually most obvious when a flock or mob is disturbed.

Mortality rates as high as 53 per cent in sheep and 45 per cent in cattle have been observed.

Symptoms may appear as early as two days or as long as 12 weeks after sheep or cattle have been introduced to an infected paddock. Stock appear to be more quickly affected in pasture than stubble areas.

Diagnosis

Other diseases which cause staggering, convulsions and death must be eliminated as possible causes of losses before annual ryegrass toxicity is diagnosed.

Other diseases which must be differentiated from annual ryegrass toxicity in sheep and cattle are polio encephalomalacia (cerebrocortico necrosis), hypocalcaemia and hypomagnesaemia, ketosis, botulism and Gastrolobium sp. and Oxylobium sp. poisoning.

Autopsy findings in stock which have died are not very specific to the disease.

The most constant feature is a pale, friable liver while haemorrhages are sometimes seen in various organs, such as the thymus and small intestine.

The presence of the nematodes in the pasture can be confirmed by forwarding samples of seed heads to the Department of Agriculture for testing. If nematodes are present this is a clear indication of disease risk.

Treatment

No successful treatment of affected stock is available. However, careful nursing of affected stock, and the provision of shade and water to recumbent animals may reduce losses.
When an outbreak of the disease occurs stock should be removed from the affected paddock with a minimum disturbance. Badly affected animals should be left in shade and given water and not disturbed until the symptoms have disappeared.

**Control measures**

Fortunately, ryegrass seed has very little long-term dormancy. Plants growing in any year come mostly from seed formed the previous year.

Because of this, control is most readily obtained if seed formation is prevented as the first stage in a programme. There is no single treatment which will completely eliminate ryegrass. However, there are a number of techniques for controlling ryegrass and farmers who wish to remove it are advised to include as many of these techniques as possible in a control programme.

**Other pasture species**

If the ryegrass is to be eliminated from the pasture, it is desirable to have other pasture plants present, particularly legumes, to provide grazing for stock. The best variety to grow will depend on the soil type and climate. It should provide soil cover to discourage seed germination.

**Autumn burn**

A hot fire will destroy any ryegrass seed lying on the soil surface. If seed has been trampled into the soil by grazing stock, this may escape the effects of the fire. Often summer grazing can be restricted to ensure there is sufficient material to provide a good burn.

**Cropping**

It is extremely difficult to kill more than about 90 per cent of a ryegrass population when the area is cropped.
This allows adequate seed to both maintain the disease in the stubble and ensure a ryegrass population the following year.

If it is necessary to crop a particular area, shallow cultivation to a depth of about 3 centimetres (one inch) at or just before the germinating rains will be helpful. Covering the seed with soil ensures a fast and even germination of the weed seeds, which can then be more effectively handled by cultivation. The land should be ploughed about 14 days after the germinating rains.

Avadex can be used as a pre-emergence herbicide to control ryegrass in crops. The crop should be sown to about 5 centimetres (2 inches), using covering harrows to level out combine tyne ridges before Avadex is applied. The chemical should be applied within two days of seeding to a well-prepared seed bed. The rate of application is 2.0 litres per hectare (1.5 pints per acre).

Dosanex is another herbicide which will kill ryegrass in crops. It is only registered at present for use in wheat. This chemical can be applied after the wheat has three leaves and up to tillering—an obvious advantage. The recommended rate of application is 1.7 kg per hectare (1.5 lb per acre).

Heavy grazing
Ryegrass can be prevented from seeding by maintaining heavy grazing with sheep during the growing period. This often becomes difficult with the spring flush of growth and extra sheep may be necessary on the infected areas. It is essential to maintain the grazing pressure until all the plants have completely dried off. Showers of rain late in the season are often sufficient to produce seed heads close to the ground.

The effectiveness of burning and heavy grazing are illustrated in Table 1, which shows the results of a trial carried out at Newdegate Research Station in 1970/71.

**Table 1—The number of plants of annual ryegrass in May 1971 on plots receiving heavy grazing in 1970 and autumn burning in 1971**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Total of 60 counts each of 400 sq cm*</th>
<th>Plants per 400 sq cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Heavy spring grazing 1970</td>
<td>494</td>
<td>8.2</td>
</tr>
<tr>
<td>2. Autumn burnt 1971</td>
<td>1336</td>
<td>22.3</td>
</tr>
<tr>
<td>3. Normal grazing 1970</td>
<td>5827</td>
<td>97.1</td>
</tr>
</tbody>
</table>

A combination of treatments 1 and 2 would obviously reduce the seed survival still further. 400 sq cm is about 1 sq link, the unit used in the original measurements.

**Table 2—The number of flowering stalks of ryegrass on 23/10/73 following various treatments of Gramoxone**

<table>
<thead>
<tr>
<th>Gramoxone treatment</th>
<th>Flowering stalks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total 3.2 sq metres</td>
</tr>
<tr>
<td>1. 1.41/ha 10/7/73</td>
<td>669</td>
</tr>
<tr>
<td>2. 1.151/ha 3/10/73</td>
<td>38</td>
</tr>
<tr>
<td>3. Nil</td>
<td>1571</td>
</tr>
</tbody>
</table>

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**Table 2—The number of flowering stalks of ryegrass on 23/10/73 following various treatments of Gramoxone**

General
The treatments described are only stop-gap measures to control the disease by removing the host plant. The development of an effective nematicide treatment or anti-toxin might be a far simpler approach to the problem. Investigations are continuing with these objectives in mind.

In the meantime the Department of Agriculture recommends these measures to prevent the spread of the disease to unaffected areas:

- Carefully check annual rye pastures at flowering for signs of yellow slime. If this is present the grass is likely to become toxic as it dries off.
- Be careful to clean down, near the gate, any machinery used on each paddock known to be infested before moving to another paddock. All soil, dust and grass residues should be removed, preferably by swabbing down or spraying with formalin solution at 1 part formalin to 40 parts of water.
- When cereals are being harvested, do everything possible to stop blowing of residues from the harvester. Eelworm galls are so light that in a strong wind they can be carried a considerable distance in dustblow from harvesters. Carefully bag up and burn residues which can be cleaned from the harvester or seed graders used in the area.
- Areas showing the disease in the spring should be burnt before the close of the burning season.
- Infected areas should be treated as separate units without moving stock and machinery through them.
- Hay or small seeds (clover and ryegrass) should not be harvested from infected areas.
- Mature seed heads should be collected from areas suspected of being infected and sent to the Department of Agriculture for testing.