Controlling annual ryegrass toxicity

C H. Trotman
IMPORTANT DISCLAIMER

This document has been obtained from DAFWA's research library website (researchlibrary.agric.wa.gov.au) which hosts DAFWA's archival research publications. Although reasonable care was taken to make the information in the document accurate at the time it was first published, DAFWA does not make any representations or warranties about its accuracy, reliability, currency, completeness or suitability for any particular purpose. It may be out of date, inaccurate or misleading or conflict with current laws, polices or practices. DAFWA has not reviewed or revised the information before making the document available from its research library website. Before using the information, you should carefully evaluate its accuracy, currency, completeness and relevance for your purposes. We recommend you also search for more recent information on DAFWA's research library website, DAFWA's main website (https://www.agric.wa.gov.au) and other appropriate websites and sources.

Information in, or referred to in, documents on DAFWA's research library website is not tailored to the circumstances of individual farms, people or businesses, and does not constitute legal, business, scientific, agricultural or farm management advice. We recommend before making any significant decisions, you obtain advice from appropriate professionals who have taken into account your individual circumstances and objectives.

The Chief Executive Officer of the Department of Agriculture and Food and the State of Western Australia and their employees and agents (collectively and individually referred to below as DAFWA) accept no liability whatsoever, by reason of negligence or otherwise, arising from any use or release of information in, or referred to in, this document, or any error, inaccuracy or omission in the information.
Controlling annual ryegrass toxicity

By C. H. Trotman, Information Branch

Eight years of intensive work has changed annual ryegrass toxicity from an unknown killer to a manageable problem. Almost out of the blue the eelworm Anguina sp. and a bacteria, Corynebacterium sp., have joined forces to make toxic an increasing area of one of Western Australia's commonest pasture plants. The result has been death for many of the State's sheep and cattle grazing on annual ryegrass, Lolium rigidum.

Since the 1970 summer when annual ryegrass toxicity disease was first fully described, the disease has spread from nine infected properties in three shires to 128 properties in 10 shires. It has moved from almost adjacent properties to properties stretching over 450 kilometres. It has caused the deaths of some 250 cattle and 12 000 sheep, and drastically reduced the areas available for grazing on some properties.

This article attempts to review the scope and success of research and extension work generated by annual ryegrass toxicity disease since 1970. The article cannot describe or even list all work by the Western Australian Department of Agriculture, and W.A.I.T. Institute, South Australian Department, CSIRO and Murdoch University.

But it should indicate the reasons for, and directions taken by research. Also it may raise the question of whether we can rightly expect more rapid research progress with a problem as complex as annual ryegrass toxicity disease is proving to be.

Symptoms in affected livestock

Annual ryegrass toxicity affects sheep and occasionally cattle and horses. Symptoms appear as early as two days, or as late as 12 weeks after introduction to the toxic area, leading to mortality rates as high as 90 per cent for sheep and 45 per cent for cattle.

Affected sheep develop convulsive spasms and stiffness in their limbs, then fall down to lie with the limbs stiff, their muscles quivering and their eyes tending to roll inwards. Cattle are similarly affected and may wander aimlessly or go down with convulsions which often include the head being thrown back violently.

Most animals return to normal after a few minutes but symptoms reappear if they are moved again. Affected sheep move with a characteristic 'rocking horse' gait. Seriously affected animals remain on the ground having occasional spasms until they die—perhaps 24 hours or more later. Even those that regain their feet may stagger aimlessly about and cattle in particular may die through accidents such as drowning in dams or becoming caught in trees or fences. Pregnant ewes grazing toxic annual ryegrass may also abort their lambs.

Toxicity signs in the paddock

Knowing that animals are affected by annual ryegrass toxicity is much easier than learning whether a paddock is likely to cause toxicity. However there are signs, usually appearing in September when ryegrass seedheads appear, that indicate potential toxicity in pastures.

The first sign is an impression of yellowness in patches of ryegrass seen at a distance of a few metres. Bacterial infection causes the yellowing by producing a slime in the seedhead. The slime may ooze down the stem and when first produced is sticky, moist and easy to see because it glistens. It is much more difficult to see once it dries and changes to an orange then brown colour.

At this stage potential toxicity can still be detected by looking for nematode (eelworm) infection in single seedheads. Normal seeds are rounded at the top and evenly pigmented green, purple or buff depending on the stage of maturity. Infected seeds are pointed, shorter than normal and contain a black or yellow gall in place of the seed. Yellow galls indicate that the seedhead has been invaded by bacteria as well as nematodes; and that they are likely to be highly toxic to livestock grazing them. Some infected ryegrass seedheads are also distorted and this can help recognition of infected areas.

Finding apparently infected ryegrass is not always positive proof that paddocks will become toxic—usually four to six weeks after the appearance of the bacterial slime. More importantly however the reverse is also true; infection and toxicity can develop without noticeable distortion or discoloring of the ryegrass seedheads. Also of course the infection may be in small portions of a paddock such as along fencelines, creeks or headlands, which are easily missed.
Recently cropped paddocks are the biggest risk

Pastures requiring closest examination are those cropped 12 months previously (Table 1). In 1977, such pastures caused nearly 80 per cent of the reported 864-sheep losses. For cattle the relationship was even more marked with 91 per cent of the 56 cattle deaths occurring on paddocks cropped in 1976.

One reason for the big losses on paddocks one year out of crop is the increased seed production of ryegrass plants which survive weed killing operations during cropping. Such plants have good supplies of moisture and fertiliser and are not continually beheaded by grazing. The result is more ryegrass tillers, more ryegrass seedheads, and more ryegrass seed to germinate during the following pasture year.

Another possibility is that cultivation during seeding buries the growing point of some ryegrass plants. This may then allow infecting nematodes to enter the plant directly from the soil instead of having to climb up the plant as they do in the pasture situation.

The end effect is likely to be more plants infected with nematodes carrying more bacteria than is likely under pasture conditions. In the following year under pasture, the soil’s nematode and bacterial populations are thus likely to be very high.

Infected material remains toxic for a long time and has caused deaths in laboratory animals fed such material more than 12 months after collection. Toxicity is generally lost in the paddock situation because galls, like seeds, shed and fall to the ground. However hay made late enough to include infected seedheads will remain toxic during storage and can cause deaths during feeding out.

![Closer view of infected seedheads. A yellow slime is easy to see at first because it is moist and glistens. It is much harder to see once it dries and changes to an orange and then brown colour.](image)

Fig. 1.—Map showing the spread of annual ryegrass toxicity. Blue triangles indicate outbreaks recorded until 1973/74; red squares show outbreaks since then. Obviously the occurrence of outbreaks is not to scale.
Sheep affected by toxicity develop convulsive spasms and stiffness in their limbs.

Table 1.—Relationship between paddock toxicity and previous cropping history

<table>
<thead>
<tr>
<th>Pasture last cropped in—</th>
<th>Sheep lost on such paddocks in 1977/78</th>
<th>Percentage of total 1977/78 losses</th>
</tr>
</thead>
<tbody>
<tr>
<td>1972</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1973</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1974</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1975</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1976*</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1977*</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Totals</td>
<td>966</td>
<td>100</td>
</tr>
</tbody>
</table>

* About half the sheep lost in the 1977/78 summer occurred on land cropped in both 1976 and 1977, and on which no chemical weed control had been carried out.

Table 2.—Occurrence of annual ryegrass toxicity disease in Western Australia 1970/71 to 1977/78

<table>
<thead>
<tr>
<th>Shire</th>
<th>Properties known to have annual ryegrass toxicity disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gnowangerup</td>
<td>3</td>
</tr>
<tr>
<td>Katanning</td>
<td>4</td>
</tr>
<tr>
<td>Broomehill</td>
<td>2</td>
</tr>
<tr>
<td>Brookton</td>
<td>1</td>
</tr>
<tr>
<td>Dumbleyung</td>
<td></td>
</tr>
<tr>
<td>Tambellup</td>
<td></td>
</tr>
<tr>
<td>Kent</td>
<td></td>
</tr>
<tr>
<td>Wongan/Ballidu</td>
<td></td>
</tr>
<tr>
<td>Quairading</td>
<td></td>
</tr>
<tr>
<td>Total properties affected</td>
<td>9</td>
</tr>
<tr>
<td>Total losses</td>
<td>Sheep</td>
</tr>
<tr>
<td></td>
<td>Cattle</td>
</tr>
</tbody>
</table>

Disease history in Western Australia

The first recorded Western Australian report of ryegrass toxicity in sheep grazing annual ryegrass is related to weaners at Dongara in 1959.

Before this however, a similar disease had been observed at Kulin, and among rams at Rocky Gully although these had been grazing seedling ryegrass rather than the mature ryegrass reported at Dongara.

Because the symptoms were similar to those reported from New Zealand for sheep, cattle and horses affected by perennial ryegrass, these early reports referred to the disease as ryegrass staggers, the name used in New Zealand.

Clarification of the Western Australian situation was probably delayed by a dramatic outbreak of ryegrass staggers at Two People's Bay, east of Albany, in autumn 1961. In this instance some 75 of 300 crossbred weaners grazing perennial ryegrass were affected, showing fairly similar symptoms to those we now associate with annual ryegrass toxicity.

A 1971 report from the Katanning area then listed sporadic cases of deaths amongst sheep selectively grazing annual ryegrass which was infected with an unidentified bacteria and a nematode identified as an Anguina sp. The report implied that the Western Australian disease was different from ryegrass staggers and was causing deaths by affecting animals' livers and perhaps other tissues including the brain.

It was apparently similar to a disease reported from South Australia and since 1968 in Gnowangerup. In Katanning districts it had caused deaths of 360 out of 2 100 sheep.

The sparcity of reports to 1970 cannot be taken as evidence that the disease was virtually non-existent before then, but certainly, since 1971, the disease appears to have spread rapidly.

Figure 1 clearly pictures this spread and Table 2 summarises the associated stock losses.
History in South Australia

In South Australia the disease's history has been remarkably similar to that in Western Australia. Deaths were first associated with annual ryegrass in the Black Springs area in 1956 and associated with the nematode (Anguina sp.) and bacteria (Corynebacterium sp.) by 1958. The disease then remained quiet but new losses were reported in the 1967 summer when 19 properties were declared infected. Toxic areas were restricted to the Black Springs region until about 1974; since then it has spread to about 100 km north and 150 km south of the initial infection area. The situation in 1976/77 was that South Australia had 90 infected properties of which 51 reported losses totalling 3,000 sheep, 78 cattle and two horses.

Research history

Early Western Australian research, in 1971, soon confirmed that annual ryegrass toxicity disease in this State was associated with the same Corynebacterium and Anguina bacterial and nematode organisms as those identified in South Australia. Other work established the quantities of toxic material needed to cause the disease and establish the fact that the disease can appear about six days after animals graze toxic materials. Field observations discovered that infected ryegrass seedheads could be identified by distortion or the presence of yellow slime from which both the nematodes and the bacteria could be isolated.

Knowing that the disease was associated with sheep grazing dense stands of mature annual ryegrass, early research work into disease control concentrated on reducing the amount of mature ryegrass available to grazing livestock.

Techniques tested during 1971 and 1972 included heavy grazing in late-winter and spring to prevent the grass from seeding, burning and cropping for two years before grazing, and displacing the seed of mature ryegrass seedheads by chaining before grazing. Heavy grazing in particular proved reasonably successful at reducing ryegrass stands in succeeding years.

The potential seriousness of the disease was fully recognised in 1972 with the formation of a Department of Agriculture working party to establish research priorities and coordinate investigations. The CSIRO also began to look at the nature of the toxin involved and two Western Australian research workers were sent to study the South Australian approach to the problem.

Top priorities were seen by the Working Party as eradication and replacement of ryegrass in affected areas, finding the chemical nature of the toxin, and recognition of those changes in the ryegrass seedhead which could lead to recognition of potential toxicity and development of grazing management and control methods.

During the next three years this research programme expanded as described below.

Ryegrass control

Experimental and field scale trials of ryegrass control methods including heavy grazing, burning, mowing, and the application of pre-emergence, early and late post emergence herbicide treatments. The complexity of some of this work is shown in the following 1973 experimental schedules which included; heavy grazing and winter applications of Gramoxone, heavy grazing and spring applications of Gramoxone, and heavy grazing with both winter and spring applications of Gramoxone.

Recommended control measures developed from this research were put into operation on 12 commercial farms known to be infected with annual ryegrass toxicity. Results to date suggest that management methods developed from these recommendations can be completely successful at preventing stock losses from the disease.

Associated with the ryegrass control programme is a project aimed at replacing annual ryegrass as the pasture grass used in areas affected by ryegrass toxicity disease.
The project is examining some 2,000 grass accessions being tested at the Wongan Hills and Mt Barker Research Stations. About 300 of these are new ryegrass types some of which may prove as useful as ryegrass but without associated problems of toxicity and being a weed in the crop.

Part of the ryegrass replacement project has been the development of a means of testing possible replacements for their resistance to toxin development. This has meant the development of glasshouse techniques for the artificial inoculation of grasses with bacterial galls, nematode galls and bacterial slime.

A recent find is that autumn burning can reduce the number of nematode galls that become colonised by bacteria in subsequent ryegrass plants by as much as 85 per cent. However, further work is being done to confirm and find the best conditions for burning.

**Disease effects and control in animals**

Research focusing on animals probably consumes more than half the research effort associated with annual ryegrass toxicity. Besides a large staff input it involves time-consuming biochemical processes, expensive equipment and chemicals, and large numbers of laboratory animals.

Included in animal research has been the development of tests to determine the toxicity of pasture materials, or fractions of such materials including nematode galls and bacterial slime. Such work required the repeated testing of various materials on animals such as sheep, rats, mice and guinea pigs until a standard test was developed to give guaranteed results. Included are the efforts of the Department and CSIRO to isolate the chemical which is acting as the toxin and determine its exact molecular structure.

Alongside such biochemical workers are veterinary researchers at the Department and Murdoch University determining the effects of the toxin on various tissues in the different animals involved. Major effects include liver damage and vascular damage in the brain.

Such findings have encouraged the continued search for treatments which might halt or reverse the disease's effects, and eventually for a vaccine to protect animals from ryegrass toxicity. Drugs tested on sheep and laboratory animals have included chloral hydrate, magnesium sulphate, pentobarbitone, sodium acetyl, promazine, xylazine, L-dopa, amylobarbbitone, dilantin, librium, valium, chlorpromazine and impromine. A drug which showed some success in the laboratory, librium, was widely tested on sheep and cattle in the field during 1977-78 with moderate success. Similar drugs in the librium group are now being tested. Detailed field work with sheep has also been carried out. It is aimed at determining the effect of stocking rate on the development of toxicity, and at monitoring blood changes in sheep grazing toxic pastures.

**Life cycle**

Early work with annual ryegrass toxicity showed that ryegrass seed containing 50 per cent immature nematode larvae was likely to be toxic. It was also known that the nematodes were parasites of annual ryegrass normally present in the soil over summer and early winter. The nematodes climb the young seedlings early in winter and enter the growing points of the plants. They then move to ovaries in the seedhead as the plant matures, mate, and produce eggs which hatch and develop to larvae. The larvae moult several times before they mate and produce galls which develop in place of the plant's seeds.

It has since been confirmed that the nematodes take the bacteria into the plant with them, leading to production of the characteristic slime and eventually to formation of the toxin.

**Fig. 2.**—Life cycle of the nematode.
By mid-1973, following the work of Mr P. Price in South Australia, details of the lifecycle of annual ryegrass toxicity were generally understood although, in spite of five years of isolation and testing, research workers are still not certain how the toxin is produced.

It seems certain that the nematode is not involved in this stage of the disease, and work is in progress to find whether the bacteria produces the toxin, or whether the ryegrass produces the toxin in response to colonisation by the bacteria. This particular work has required the development of techniques to grow plant callusses (loose massess of living plant cells) for infection by bacteria.

Understanding the life cycle increased the scope for controlling the disease. If the seedhead could be removed, possibly the pasture would not be toxic; but ryegrass was a valuable pasture plant and was difficult and expensive to eradicate. If however the nematode could be eliminated the ryegrass could remain because there was no way for the bacteria to enter the ryegrass seedheads. Or, if the bacteria could not react with the seedhead to produce a toxin.

Work with nematicides (chemicals which kill eelworms) to reduce nematode populations began in 1973 and has continued as new chemicals have become available. The technique is expensive but shows economic promise during pasture years immediately following cropping when the risk of toxicity is highest.

There have also been a number of trials with bacteriocides but these have not shown the same potential for controlling the disease.

**Monitoring disease occurrence**

To provide a service to farmers, and to check how far the disease might be spread and what conditions apparently suited it, the Department began a sample testing service for farmers in late 1973. Persons suspecting their paddocks to be toxic were requested to contact their district office or forward samples to South Perth. Within a month of starting the service the laboratory concerned was testing 50 samples a week.

The service was continued in subsequent summers and was supplemented in 1975 by taking samples from all Cooperative Bulk Handling bins, and by conducting a survey of one per cent of all properties in agricultural areas. These surveys confirmed the known distribution of the disease and defined its limits. Annual surveys are still done to assess the effect of control measures in containing the disease.

**Future prospects**

While research workers will undoubtedly continue their efforts to completely understand the cause, nature and effects of annual ryegrass toxicity, results so far might well satisfy farmers’ needs and concern. It seems likely that the disease could spread, but its effects on pastures and livestock are known and controllable.

Farmers are now avoiding losses, as shown by Figure 3 illustrating recent relatively low death rates in ryegrass affected flocks. This has been achieved by good stock management and better weed control which has further increased crop yields.

Good stock management includes careful examination of ryegrass pastures for signs of slime and potential toxicity. This means twice daily observation of stock in risky paddocks, and their immediate removal to a safe area at the first sign of disease symptoms. It means continued observation of stock in ‘safe’ paddocks to ensure that they are safe.

Good management also means care with hay made from possibly suspect paddocks, and care with feed, seed and machinery movement to lower the chance of spreading the disease to new areas.

Better weed control means killing ryegrass in crop years by effective timing of ploughing and herbicide operations, effective use of heavy spring grazing and autumn burning techniques, and possible selection of heavily ryegrass infested paddocks for continuous cropping.

To improve control methods, research workers will undoubtedly continue looking at the toxin, the nematode, the bacteria and tissue effects in animals for many years. They will also continue to look for new management techniques, new grasses, new clovers and new antidotes to make control of ryegrass toxicity disease easier. Meantime however, eight years of intensive field and laboratory work, and continuous extension work, has changed annual ryegrass toxicity disease from an unknown killer to a widely recognised, manageable problem for farmers.

![Fig. 3.—Average death rates in affected flocks, showing the decline in losses as management techniques have become recognised.](image-url)