Lupinosis

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In spite of many years of intensive investigation lupinosis remains a remarkably complex and baffling problem. Current research is throwing new light on the disease and it is now much better understood—although far from beaten.

Lupinosis research is co-ordinated by the Department of Agriculture's Lupinosis Committee, on which are represented all groups working on the disease. These include veterinary surgeons and pathologists, chemists, plant pathologists and animal husbandry specialists.

Chairman of the Committee is Dr M. R. Gardiner, Chief of the Department's Animal Division and a veteran of lupinosis research.

In this article Dr Gardiner summarises the current state of knowledge of lupinosis and reviews recent research on the disease.

A following article is devoted to Phomopsis rossiana, the fungal cause of lupinosis.

Lupinosis has been an important disease of sheep, cattle and horses in lupin growing districts of Western Australia for many years. The greatly increased area of sweet lupins planted in the last few years for seed production has produced large areas of lupin stubbles for summer grazing, so a solution of the problem has become far more urgent than ever before.

An intensive research programme has been in progress in Western Australia for the past 15 years and a tremendous amount of information has accumulated, some of which can now be used in lupinosis control programmes.

The disease has been a significant cause of sheep deaths and illthrift, and an occasional cause of cattle deaths and illthrift during the summer months for the past 25 years, especially on the coastal sandplains extending northwards from Perth.

The development of lupinosis in stock was, for years, unpredictable and rather mysterious and many theories on its cause were developed.

However, in 1963, it was conclusively shown that dry lupin stubble often became unsafe for sheep a few days after summer rainfall and almost always became toxic to some degree after wetting. This association of rainfall with toxicity suggested the growth of a microorganism on the dead plant, an event which requires both warmth and moisture in the right amounts and for the right periods of time.

A vigorous search for such a micro-organism, and in particular a fungus, led to the isolation of more than 25 species of fungi from toxic lupin roughage by Mr Attila Bokor*.

Of these species, about five were found always to be present. Each of these five species of fungi were inoculated onto clean lupins and incubated for a few days under appropriate moisture and temperature conditions and then the infected lupin roughage was fed to sheep. Only one of the five fungi consistently induced toxicity and when this lupin roughage was fed to sheep, typical lupinosis was produced.

This fungus has since been identified as Phomopsis rossiana although it was first placed in the Cytospora group.

Two other discoveries at about this time had important effects on

* Formerly of the Animal Health Laboratory, now Plant Pathologist, Department of Agriculture, Albany.
the research programme. One was the discovery that the mouse could be used experimentally to detect the presence of toxin produced by the fungus on dead lupins and the second was the discovery by chemists at the Animal Health Laboratory that the toxin could be concentrated in almost pure form by a series of solvent extractions, enabling it to be injected into mice (and sheep) to produce the typical pathological changes of lupinosis.

However, biological tests such as that based on the degree of damage caused by toxic material in the mouse liver are not fully satisfactory and a reliable chemical test for toxicity is needed.

During the last five or six years, research on lupinosis has concentrated on the mycological aspects of the problem and on a deeper understanding of the ways in which the toxin affects the cells of the liver, the principal target organ in all animal species so far studied.

**The clinical disease**

Affected sheep characteristically show a variable loss of appetite, ranging from partial to complete, a few hours or days after consuming the toxic lupin roughage.

With very toxic material one small intake of lupins may be sufficient to cause almost immediate and complete loss in appetite and many of these sheep are likely to die during the following weeks with signs of acute lupinosis and without eating again.

Sheep eating lupin roughage of lesser toxicity develop a partial loss of appetite. This may be soon after starting to eat the lupins or may be delayed for as long as two or three weeks.

Some sheep may show a fluctuating appetite, although with an accompanying loss of weight and condition.

Many, perhaps most, sheep grazing affected lupins develop some degree of liver damage without showing marked signs of lupinosis. These sheep, however, may suffer loss of production.

In most forms of lupinosis, jaundice (yellows) occurs to some extent. In severe disease, the mucous membranes of the eyes and mouth may be intensely yellow while in the milder forms, they may have a "muddy" appearance.

Affected animals look dejected and depressed and fail to keep up with the rest of the flock. They may wander off in a disoriented manner and soon go down, or get caught up in fences or other obstacles, and die. If not too badly affected, sheep that will still eat recover, in many cases, if they are taken off the toxic lupins and given access to non-lupin pasture.

**Pathology of lupinosis**

If acutely affected sheep are opened up soon after death, the liver is seen to be greatly swollen, of a bright yellow or orange colour, and intensely greasy when cut with the knife. These signs are due to massive accumulation of fat in the liver cells.

The gall bladder is also enlarged, sometimes from five to six times its normal size, and is filled with a dark green bile. The subcutaneous tissue, body fat, and other body tissues are tinged with various shades of yellow to orange, and the kidneys are usually darkened.

The rumen may contain a little watery ingesta or be impacted with a mass of dry feed. Areas of fat necrosis in the omentum and elsewhere are common. As with sheep, the liver is the main target organ in cattle and horses and resembles the liver of sheep with acute lupinosis.

Lupinosis of longer duration and lower intensity is apparently much more common than acute lupinosis and in these cases the liver has a coppery or tan colouration and is smaller than normal. It often feels hard and has a granular appearance due to the proliferation of fibrous tissue in its substance.

In a sheep severely affected with chronic lupinosis the liver is misshapen and distorted and the disease will in fact have become cirrhosis.

**Lupinosis in other species**

In the laboratory animal, the pig or the chicken, few if any of these clinical and pathological signs of lupinosis are seen. However, the main organ affected is again the liver. Only by microscopic study can the disease be identified as lupinosis.

These species have been invaluable in our present understanding of the disease as they have revealed the fundamental effect of the toxin on the liver cells. There is apparently an added factor in the ruminants and the horse which gives these species a more dramatic response to the toxin.

**Field aspects of lupinosis in sheep**

Sheep and cattle in Western Australia are affected mainly by eating the upper and smaller stems of the standing plant.

Department of Agriculture trials carried out some years ago showed that under field conditions this part of the plant becomes much more toxic than other parts, although the leaf and pod material may develop some toxicity. However, we have not yet found toxic seeds under natural conditions although Mr P. Wood of the Plant Pathology Branch, has found natural seed infection by the *Phomopsis* fungus.

There is as yet little understanding of conditions that prompt grazing animals to eat toxic roughage. In some stands, due to local conditions, all of the lupins may be toxic and if dominant in the pasture mixture may leave the animals little choice of feed. In many lupin pastures, however, some parts of the paddock may be more toxic than others and there may even be parts with little or no toxicity, again due to local conditions conducive or otherwise to the growth of the fungus.

It would appear, therefore, that whether an animal will be seriously poisoned or not will depend on chance encounters with the more toxic parts of the stand and on the availability of other pasture species.

All varieties of lupin, both bitter and sweet, support the growth of *Phomopsis*, although there may be some varieties that are more resistant to infection. This possibility is being investigated.

Some observers have reported that fat sheep are more quickly affected and more susceptible to lupinosis than lean animals. This is probably because, when appetite is impaired, body fat is mobilised for energy, passing to the liver and charged with excessive quantities.
Jaundice (yellowwing) of the gums (above) and eye (below) can be an external indication of liver damage caused by lupinosis. In ewes it can sometimes also be seen in the vulva. Jaundice is not always present in cases of lupinosis and varies from slight to severe levels. The lupinosis toxin damages liver cells and where these contain much copper the lupinosis pigment and fibrosis/cirrhosis are greatly exacerbated. There are also similarities in the pathology of lupinosis and copper poisoning.

However, it has been difficult to demonstrate a definite relationship between copper intake and lupinosis, and there is no general agreement among pathologists on this aspect of the disease.

During the summer of 1974-75 a high incidence of white muscle disease has been observed among young sheep grazing lupin stubbles. This is assumed to be a result of inadequate selenium intake in these animals, as the selenium content of lupins is known to be low.

Although there is no indication of a relationship between lupinosis and white muscle disease or selenium deficiency, it does seem likely that, especially during the 1974-75 summer, lupinosis may have been blamed for deaths which were in fact due mainly to white muscle disease.

Sick sheep with typical appearance of the terminal stages of lupinosis.

Sheep affected with parasitism also appear less resistant to the damaging effects of the disease.

Early investigations suggested a relationship between lupinosis and high liver copper levels of sheep grazing lupins. Sheep with high liver levels of copper as a result of exposure to copper topdressed paddocks or high copper pastures appeared more susceptible to the subacute and chronic forms of lupinosis than those with lower liver copper levels. The lupinosis toxin damages liver cells and where these contain much copper the lupinosis pigment and fibrosis/cirrhosis are greatly exacerbated. There are also similarities in the pathology of lupinosis and copper poisoning.

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Mycological aspects of lupinosis

Following the elucidation of the fungal cause of lupinosis, a Departmental research team which included mycologists Dr Alan Brown and Mr Peter Wood, of the Plant Pathology Branch, assisted by chemist Mr David Petterson and research officer Mr Murray Nottle, of the Animal Division, has investigated the life cycle of the fungus *Phomopsis rossiana* and the conditions which favour the development of the toxin.

Mycological aspects of lupinosis are discussed in the following article (Page 31).

The toxin

In recent years Mr Petterson has succeeded in concentrating the lupinosis toxin and in removing most of the impurities. The resulting concentrated substance is immensely toxic and extremely small amounts can induce the typical changes of lupinosis in animals.

If the chemical structure of the toxin can be unravelled, a chemical antidote is a possibility in the treatment or prevention of lupinosis.

The fundamental toxic effect has been shown to be an interference with cell division in the liver and the toxin has been classified among...
Sheep affected by acute lupinosis. The subcutaneous tissue, body fat and other body tissues are tinged with various shades of yellow to orange. The liver is bright yellow and the gall bladder is greatly enlarged.

Fibrosis of the liver of sheep affected with chronic lupinosis. The liver is shrunken and hard and has a granular appearance.

Swollen, yellow, friable liver and great enlarged gall bladder of acute lupinosis, compared with normal liver. The yellow colouration and friable texture are partly due to massive deposition of fat in the liver.

the poisons known as mitotic inhibitors.

In some species, such as the pig, the small laboratory animals and poultry, damage to individual liver cells undergoing division is the only real change seen but in ruminants and the horse, cell injury is much more extensive.

A single dose of toxin has been shown to produce cell division damage lasting for eight weeks or more, so some part of it apparently becomes permanently attached to constituents of the cell.

Appetite does not seem to be impaired in the non-ruminant animal and the continuance of adequate nutrition of these animals after exposure to the toxin may explain to some degree why they do not show clinical effects unless very large doses are given.

There is evidence that the lupinosis toxin profoundly depresses rumen function, possibly by acting on the micro-organisms essential for digestion of food in sheep and cattle.

**Prevention**

The prevention of lupinosis so far remains empirical despite the great advances in understanding the disease.

Weather forecasting can be of great value, as can actual meteorological observations on the farm. Lupin roughage can take up large quantities of water and hold it long enough even in hot weather to allow the fungus to produce toxin.
Although precise moisture and temperature criteria are not known, it is suggested that if lupins become thoroughly wet either by rain or by heavy dews, and retain this moisture for a day or more with the assistance of much cloud cover and high atmospheric humidity, conditions for the development of toxicity can be anticipated. A close watch should then be kept on sheep and cattle grazing lupin pastures.

Signs of inappetence (not eating) especially should be noted. Moving stock to alternative non-lupin grazing, if such is available, is of course an obvious necessity.

A number of cases of lupinosis were reported during the 1974-75 summer among sheep grazing lupin stands which were apparently not wet by rain after maturity. In at least some of these cases it seems that little if any dew would have been deposited on the lupins, as the summer was unusually dry.

In the absence of detailed understanding of the environmental conditions necessary for the *Phomopsis* fungus to grow actively and to produce toxin, this situation is difficult to explain and further information on these outbreaks is being gathered.

However, in some circumstances it is probable that short periods of high moisture which would escape normal detection may be sufficient to bring about production of enough toxin to cause a mild degree of liver damage.

Stands of mature lupins may therefore have developed some toxicity at times when rain or heavy dew have not been apparent.

Toxicity may diminish, especially, we believe, if certain other microorganisms proliferate on the lupin stubble. Sheep may be cautiously reintroduced to such paddocks two or three weeks after the primary rainfall or other moisture precipitation.

However, the continuation of very hot, very dry weather after the primary event will almost certainly allow toxicity to persist.

Sheep that have been previously exposed to high copper environments, and overfat sheep, should not, if possible, be allowed to graze lupins subjected to the climatic factors mentioned above.

**Stands of mature lupins may be expected to become toxic after periods of rain or heavy dew**