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Lupinosis — a disease still with us

Two sheep of the same age, one effected by chronic lupinosis. In chronic lupinosis a "tail" develops in the flock

By J. G. Allen, P. McR. Wood, K. P. Croker and J. Hamblin

Current research on lupinosis continues to reveal what a complex problem it is, but the improved understanding should help to reduce losses.

Lupinosis is a disease caused by eating toxins produced by a fungus which colonises lupins. This fungus, Phomopsis leptostromiformis, formerly called Phomopsis rossiana, actually infects the green lupin but persists on the stubble.

In Western Australia natural outbreaks of lupinosis have occurred in sheep, cattle, horses and donkeys grazing lupin stubbles, and the disease has been experimentally produced in pigs, poultry and a wide variety of laboratory animals. Sheep are the main species affected in the field.

Research into lupinosis and particularly its prevention, has involved studies on the fungus, the lupin plant and the grazing animal. The Department of Agriculture research team includes a range of specialists working on these aspects.

The fungus

In seeking to control the fungus which causes lupinosis, the investigation of fungicides was a logical step. It was found that application of fungicides to the dead lupin plant did not control the fungus, but that some control was achieved when green lupin plants were sprayed between August and October.

In subsequent trials over two consecutive years, one or two applications in August or September of benlate, at 400 g/ha in a solution of 0·5 per cent summer spraying oil, gave good control of the fungus. Significantly less toxicity developed in the treated lupins as compared with untreated lupins.

However, in the third year of trials control was poor and treated lupins were as toxic as untreated lupins.

Because of this variability, it has been concluded that fungicides are of only limited use, although new ones are being tested in the laboratory as they become available.

One recent breakthrough has been the demonstration that lupinosis can be prevented by cutting the green lupins and conserving them as hay.

In trials by the Department, the lupins were cut after pod formation but before leaf drop. They were then conserved in fodder rolls and bales, which were left in the paddock until being fed to sheep. Sudden drying of the lupins resulted in control of the fungus. Furthermore, it appeared that the lupin hay was less suitable for toxin production by the fungus than normally matured plants.

Lupin hay prepared in this way can therefore be considered a safe feed, equal in feed value to unharvested lupins.

The lupin plant

In 1974 it was found that the Ultra variety of lupin was apparently resistant to infection by Phomopsis.

Ultra has subsequently been compared with other lupin varieties throughout the lupin-growing areas.

On every occasion the level of Phomopsis infection of Ultra has been less than that of other varieties, and Ultra has developed significant toxicity on only one occasion. In that case, Ultra was being grown in small plots, completely surrounded by Unicrop lupins that were heavily infected with Phomopsis.

Ultra has been concluded, therefore, that Ultra is resistant to the fungus and lupinosis is unlikely on its stubbles.

Resistance in Ultra has resulted in an extensive search for Phomopsis resistance in other lupins. No major popular variety is resistant to the fungus but resistance has been found in some minor lines.

Breeding to develop resistant lupins has begun and considerable gains have been made. Some of the breeding material is now twice as resistant to Phomopsis infection as Unicrop.

When resistance is established, further breeding will be required.
Lupin hay prepared as fodder rolls is considered safe feed for sheep to bring the new lines up to commercial grain yields. This will take a number of years.

Research with animals

Selenium and/or vitamin E have been suggested to prevent lupinosis, but recent trials to test this hypothesis have had little success. Oral doses of up to 15 mg of selenium did not protect field-grazed sheep. In field trials 800 IU of vitamin E given monthly, and in laboratory trials, 1 000 IU followed by 700 IU given weekly to sheep, also gave no significant protection. These levels are very high so it can be assumed that neither selenium nor vitamin E will protect against lupinosis.

Successes such as those reported with selenium probably reflected a response to white muscle disease rather than lupinosis. White muscle disease is discussed later in this article.

There is considerable field evidence that in general, lupinosis causes more deaths among weaners than other sheep. This was confirmed in a recent trial in which weaner sheep suffered more liver damage than mature wethers grazing the same lupins at the same time.

However, it must be realised that weaner sheep grazing lupins are susceptible to white muscle disease as well as the recently discovered muscle disease described as "lupinosis-associated-myopathy". The apparent susceptibility of weaner sheep to lupinosis is therefore probably exaggerated.

Another line of research has indicated that eating habits may be responsible for the apparently greater susceptibility of weaners to lupinosis.

In a recent trial at Bakers Hill, mature non-pregnant ewes, mature wethers and weaner wethers were grazed on plots containing only lupin stubble. In this trial the weaners developed lupinosis first, but over the trial period of nine weeks equal mortality rates (40 to 45 per cent) and equal degrees of liver damage occurred in each group. Adult sheep presumably selected less of the toxic plant material than weaners did, but when this was exhausted were forced to eat the toxic components of the stubble.

If sheep do select non-toxic material, provision of alternative feeds may prevent lupinosis. This technique has been recommended for many years and in fact, a recent trial at Bakers Hill showed less liver damage in sheep grazing stubbles of weedy lupin crops, than in sheep grazing stubbles from clean crops.

However, simply providing alternative feed, whether in the same or an adjoining paddock, is not the whole answer. The sheep must still be watched carefully. For example lupinosis outbreaks have occurred in paddocks in which lupins were only a very small proportion of the feed available, and also in a paddock where the only access to lupins was by reaching through a fence into an adjacent paddock.

If the provision of alternative feed is to be successful, sheep must be forced to eat the alternative feed. This can be done by rotationally grazing lupin and non-lupin paddocks. Some farmers have adopted this practice, apparently with success, and the Department is evaluating seven-day rotational grazing of lupin stubbles.

Rates of stocking are important in the development of lupinosis. Trials have repeatedly shown that sheep grazed on lupins at stocking rates above 30 per hectare will suffer more liver damage, and therefore are more likely to develop clinical lupinosis, than sheep grazed at lower rates.

For this reason, farmers who try to make the most use of lupin stubble by heavy grazing before toxicity develops in summer must be very cautious. Occasionally, toxicity is present at harvest and may cause severe outbreaks of lupinosis if combined with heavy stocking.

Persistence of the toxin

Since the early 1960s it has been usual to remove sheep from lupins after summer rainfalls above 8 or 9 mm, and return them after two to three weeks of fine sunny weather.

This practice developed because lupinosis outbreaks in sheep...
grazing bitter blue lupins were most frequent a few days after summer rains. It was presumed that *Phomopsis* only produced significant amounts of toxin during warm, humid weather, and that the toxins broke down within two to three weeks. It has now been shown that once toxicity develops in lupins it may persist for three to four months. Field outbreaks of lupinosis associated with long periods of toxin persistence were prevalent during the past two summers. 

The exact environmental conditions needed for toxin production are still not known, but there is good evidence that moisture and warmth are involved. What is important is that the moisture required need not be rainfall; dew or high humidity are probably enough. Conditions leading to breakdown of the toxin are also unknown. In trials monitoring the toxin, high levels of toxicity have declined to almost nothing within three weeks, then toxicity has suddenly risen again within eight days, without any obvious changes in the weather. 

The most probable reason for the past success of removing sheep after rain was that the W.A. blue lupins grown in the 1960s were far less palatable than the sweet lupins now generally planted. Sheep would eat more of the blue lupins soon after rainfall when the stems would be softer and more palatable. Consequently lupinosis outbreaks would coincide with this period of increased lupin consumption.

In contrast, the softer, more palatable sweet lupins are eaten at all times, and rainfall probably has little effect on consumption. Since toxicity cannot be predicted, and it can persist for long periods, all lupin stubbles must be considered as potentially dangerous. However, this does not mean that stubbles cannot be used. Stock should be inspected regularly and early signs of the disease should be known. In acute lupinosis of sheep the first signs are lethargy and a marked loss in appetite, while in chronic lupinosis a “tail” develops in the flock. If a “tail” does develop, loss of appetite and lethargy also occur but often are not obvious until the accompanying loss of condition is noticed. The detection of stragglers, by regularly moving the grazing flock within the paddock, is an early indication of both acute and chronic lupinosis.

**Copper and lupinosis**

It was previously thought that sheep with high liver levels of copper were more susceptible to lupinosis than those with low liver copper levels. This was concluded prematurely because in outbreaks of lupinosis, the most severely affected sheep frequently had high liver copper levels. It has now been shown that lupinoxin toxin actually causes the liver to store copper, and the more severely affected a sheep the more copper the liver will store.

Copper should therefore not be applied to paddocks which will be cropped to lupins, and all efforts should be made to limit copper access to sheep grazing lupins. Even slightly elevated levels of copper intake, combined with liver damage due to lupinoxin, may lead to copper toxicity when it would otherwise not be expected.

**White muscle disease and lupinosis-associated myopathy**

Lupins are low in selenium and vitamin E. As these deficiencies predispose to the development of white muscle disease, it is essential that all weaner sheep be treated with selenium before being introduced to lupin stubbles, thus avoiding a major cause of death. Recently, another form of muscle disease, lupinosis-associated myopathy, has been found in sheep that develop lupinosis. This condition has been seen mainly in weaners, but has also occurred in adult sheep. In affected sheep, lupinosis-associated myopathy is indistinguishable from white muscle disease, but it is clearly a different condition because it does not respond to, and is not prevented by selenium or vitamin E. When lupinosis-associated myopathy occurs in a flock affected by lupinosis the result is increased deaths. Unfortunately, the only way known to prevent this disease is to prevent lupinosis.

**Lupinosis in cattle**

Almost all lupinosis in cattle has been in late summer and early winter, and almost all deaths have involved late-pregnant or...
recently-calved cows.

In a trial at Esperance three pregnant nine-year old cows, three yearling steers and three yearling heifers were introduced to a paddock of highly toxic lupin stubble at the beginning of March. All animals maintained or gained weight until calving began in April. After calving the cows lost condition and died 16, 28 and 49 days after calving, but in the same period the steers and heifers continued to maintain or gain weight.

After death or slaughter, cows were found to have severely affected livers while the steers and heifers only had mildly damaged livers. None of the young calves were affected.

Furthermore, the damage in the livers of the cows was very similar to that seen in starvation ketosis; that is, the livers were extremely fatty, as were the kidneys and adrenal glands. In fact only a small amount of the damage in these livers could be directly attributed to lupinosis. It is now thought that late-pregnant or recently calved cows with mild, and often subclinical lupinosis, either lose their appetite or some other change occurs in the liver which results in nutritional stress. This causes the cow to rapidly mobilise its fat reserves for energy, and this results in the fatty liver, kidney and adrenal glands. It also results in ketosis, which causes death.

Affected cows have recovered when removed from the lupins, treated for starvation ketosis and provided with good quality feed. If a calf is at foot it should be removed.

Steers and heifers do develop liver damage when they eat toxic lupins, and very occasionally there is enough damage to cause death. However more often such animals grazing lupins appear unaffected until the break of the season. At the break, the cattle start eating recently-germinated, green feed and rapidly develop photosensitisation. Excretion of the photodynamic substance phylloerythrin (a breakdown product of chlorophyll) is impaired in the damaged liver. This photosensitisation can be very severe and result in considerable loss of condition, if not death.

Affected cows may also develop photosensitisation. In such cases the udder is often affected and the cow refuses to feed its calf. The calf may then die from starvation.

When the affected cow does feed its calf, the calf may also develop photosensitisation. It is not clear whether the calf is affected as a result of the toxin or of phylloerythrin in the milk. In any case these affected calves may also die.

To summarise recommendations on the use of lupin stubbles for cattle:
- Late-pregnant or recently-calved cows should not be grazed on lupin stubbles.
- Other classes of cattle can apparently be grazed on lupin stubbles with reasonable safety, provided that they are removed from the lupin stubbles before the break of season.

Ammonia toxicity

Another finding in recent years is that lupin grain or any high protein feed supplied to sheep with liver damage from lupinosis may cause ammonia toxicity.

Ammonia is produced in the body normally from protein. However, because it is toxic, it is rapidly removed from the body, mainly by conversion to urea in the liver.

When the liver is damaged, as occurs in lupinosis, urea formation is impaired and ammonia builds up in the blood. Brain damage may follow.

Lupin grain has a protein content of between 30 and 45 per cent, and therefore when fed to sheep will increase protein metabolism and ammonia production. When fed to sheep recovering from lupinosis toxic levels of ammonia rapidly build up and brain damage results.

Affected sheep tend to wander aimlessly and are often found dead, entangled in fences. This is extremely distressing as what is thought to be the best feed, has killed them.

When a flock develops lupinosis it should be removed from the lupins and attempts should be made to stimulate appetite by providing good quality pasture, hay, or cereal grain.