Lupinosis

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Cover Page Footnote
In carrying out investigations into lupinosis, we wish to gratefully acknowledge the assistance received from M. C. Nottle, Biochemist, A.H.N.L., Veterinary Officer A. M. Williams (Geraldton), Agricultural Advisers, G. H. Neil and B. P. Carlin (Moora), and Pasture Improvement Groups and farmers in the Dandaragan and Gingin areas, notably Messrs. G. M. Roberts and F. A. Wedge.
LUPINOSIS is a disease which has caused severe mortalities in West Australian sheep flocks in recent years, and one which—because of the important role which lupins are playing in light land development—must inevitably continue to be a matter of serious concern.

At the outset, it is essential to clear up a popular misunderstanding and to emphasise that lupinosis is NOT lupin poisoning.

It is well known that many varieties of lupins—including the West Australian blue lupin (*Lupinus digitatus*) and the New Zealand blue lupin (*Lupinus angustifolius*)—contain poisonous alkaloids, an excessive intake of which can cause convulsions, madness and other nervous symptoms which often result in death.

Lupin poisoning is an acute condition and the effects are not cumulative. Heavy mortalities may occur when hungry sheep eat large quantities of lupin seeds in a short period of time, but under local conditions of sheep husbandry such mortalities are rare. Not uncommonly, transient intoxication and convulsions may occur when sheep are driven following heavy intakes of lupins, but complete recovery usually takes place if the sheep are rested. Lupin alkaloids are rapidly eliminated in the urine, but troubles can arise when the absorption of the alkaloids takes place more quickly than they can be eliminated from the body.

Lupinosis on the other hand is a condition which appears to be quite unrelated to the poisoning by alkaloids*. It is cumulative in its effect and is characterised by liver damage manifested by dullness, loss of appetite and "yellows" or jaundice. Death may occur within a few days or be delayed for as long as two months, even following a change of diet. In the more chronic cases, wasting is a feature. Many affected animals recover, because of the remarkable regenerative capacity of the liver, but they naturally show a temporary loss in bodily condition. The death rate may be as high as 30 per cent. of the flock. Indirect losses are by no means negligible. The erratic and unpredictable incidence of lupinosis, moreover, makes this problem a very worrying one. Until more is known about the actual cause and mechanism of the disease the development of light lands with the aid of lupins cannot be undertaken with confidence.

**A PROBLEM DISEASE**

The first task in formulating methods of controlling or preventing a disease is to learn all that is to be known concerning the disease itself—its history in other countries, what causes it, the circumstances or environments that favour its occurrence, the varieties of plants most likely to cause the disease, etc., etc.

Here we have encountered many puzzling features. Apparently, the disease was first described in Germany in the latter part of the 19th century when it became a serious problem, but the most extraordinary feature is that in Europe, as in Western Australia, the disease was recognised and became a menace only after lupins had been used widely and with safety as a stock food for more than 20 years.

The reason for this is not yet understood. It seems evident that lupinosis results from some harmful factor which is not constantly present in the plants in dangerous concentrations.

What this factor is, has yet to be determined.

*1* Lupin alkaloids are constantly present, particularly in the seed, yet the occurrence of lupinosis is most erratic and unpredictable.

*2* The alkaloid content of husks from a sample of W.A. blue lupin, which induced lupinosis when fed to experimental sheep, was found to be very low.

*3* Pure alkaloids, prepared from the same species, have been fed to laboratory animals for prolonged periods without inducing liver damage.

*4* We have fed laboratory sheep for prolonged periods (to 8 months) on the lupin seed alone; no liver damage resulted although signs of alkaloid poisoning were noted from time to time.
Fig. 1.—Portion of a “fatal” paddock of New Zealand blue lupins at Moolalabeenie, 1959. There were only 25 acres of lupins in a 900 acre paddock with a good balance of other feed, yet 70 out of 500 sheep died as a result of only one week’s continuous grazing.

It is something which is not confined to any one of the many lupin varieties as several different species have been implicated.

Its presence cannot be related to any known changes in environment as the incidence of lupinosis varies from year to year and from location to location, apparently without rhyme or reason. The German research workers demonstrated that lupins from the same field could be harmless in some years and highly dangerous in others, yet they could not relate these findings to any set of circumstances.

Experience has shown that the increase in lupinosis cannot be ascribed to changes in farm management and practices. Sheep commonly grazed lupin-dominant paddocks with impunity for many years prior to 1951 when the disease first commenced to be a problem here. Conversely, some very serious mortalities have been recorded, in recent years, on paddocks where lupins comprised only a small proportion of the effective grazing.

In some years, notably 1959, losses may be widespread and severe, yet in other years very few deaths are recorded. On some properties in some years, lupins may be grazed with perfect safety. Sometimes only individual paddocks are dangerous. Occasionally, as in the present season, all lupin paddocks on a property may appear to be implicated.

Climatic influences; soil types; fertiliser treatments; the age of lupin establishment; mould growths or disease affecting lupin plants; changes in farm practice notably those favouring lupin dominance in the diet of the animals, are among factors studied without yielding any definite clues to the predisposing causes of lupinosis.

We know that a diet dominated by “dangerous” lupins is more likely to cause lupinosis than a well-balanced diet, and that repeated feeding on such plants will probably lead to cumulative liver damage.

It seems too, that it is the consumption of lupin roughage which causes lupinosis, and not the seeds, as was believed earlier.

* In view of recent evidence that moulds growing on pasture plants may cause liver damage, this aspect is receiving further study.
German research workers showed that the active principle was concentrated in the husks, and they induced lupinosis in experimental sheep by feeding them on husks or by giving them a watery extract derived from the husks.

In Western Australia we have reproduced the disease in laboratory sheep by feeding lupin roughage collected from three properties where heavy losses were experienced in 1959, and we have a stockpile of this material which is being used for experimental and chemical studies to determine the cause and nature of lupinosis.

GREEN LUPINS AS A CAUSE OF LUPINOSIS

Until recently we thought that green plants could be grazed with impunity. In 1958, however, lupinosis was diagnosed in more than one flock, which, presumably because of unusual seasonal conditions, preferentially grazed lupin plants about 18 in. high during the July period. On one property sucker lambs were affected. Cattle have been affected as a result of eating lupin hay cut green, and recently lupinosis was diagnosed in a mob of cattle which had grazed intensively on young green plants for two weeks, due to shortage of other herbage in their paddock. It appears therefore that the green plant, lupin hay, and by inference, lupin silage, cannot be regarded as a completely safe ration.

MANIFESTATIONS OF LUPINOSIS

The condition, lupinosis, is essentially the manifestation of more or less severe liver damage which may result from a lupin diet. In acute cases the liver is heavily infiltrated with fat, being enlarged, bright yellow and very friable. In more chronic or protracted cases there is liver destruction followed by scarring and fibrosis. The liver may be paler than normal, very firm in texture, and may be very small and abnormal in shape ("boxing-glove" liver). Some degree of liver damage may be tolerated, and abnormal livers (of the chronic type) are commonly detected in sheep killed for rations. When the condition is severe or acute, animals may die after only a few days illness and after only one or two week's grazing on "affected" lupins. At the other extreme there may be little definite indications of sickness other than a slight or progressive loss of condition over a protracted period. Deaths may still occur for two months or so after a complete change of diet, although removal from lupin paddocks in many instances results in complete recovery. Sheep with marked liver damage may succumb to stress—in several instances drenching with phenothiazine, for example, has apparently precipitated mortalities in affected flocks.

Common signs of lupinosis are dullness, isolation and loss of appetite—jaundice or "yellows" is frequently evident. Affected
sheep often die with their heads pressed against obstructions, fences, trees etc. In some instances sheep and cattle have shown evidence of photosensitisation, a sunburn of ears, muzzles and other parts of the body unprotected by pigment, wool or hair. This results from a sensitisation of the skin with derivatives of chlorophyll (the green pigment of plants) which cannot be disposed of by the damaged, abnormal liver.

Lupinosis may affect horses as well as sheep and cattle and may be induced in some other species experimentally. Here we have encountered only one case in a horse and few cases in cattle. In sheep, lambs and weaners appear to be particularly susceptible. There is some evidence which suggests that crossbreds are more susceptible than Merinos. It seems that repeated experiences of lupin grazing do not necessarily increase susceptibility, although sheep which are familiar with lupins are more apt to graze them heavily and selectively when first turned into lupin paddocks and liver damage may be cumulative from season to season.

**LUPINS AND LUPINOSIS IN GERMANY**

A comprehensive account of the use of lupins and of the occurrence and investigations of lupinosis was given by Roloff (1883) and by other workers about the same period. No significant information can be found in scientific literature subsequently. These early investigations, however, are very pertinent to the present situation in Western Australia.

In North Germany, lupins were grown extensively, the white, blue and yellow varieties being used for stock feed. The crop was cut after flowering, when half-ripe, or ripe, and stacked as hay. The lupin hay was fed to stock, particularly sheep, maintained in barns, and meadows hay, maize, potatoes, etc., were also fed.

Lupinosis began to be a problem about 1875 and heavy and widespread losses were experienced in the subsequent years. It was observed that hay from certain fields was highly dangerous, whereas similar hay from other fields could be fed with complete safety for prolonged periods in some seasons; in other years the position might be reversed.

The severity and occurrence of the disease and the location of “dangerous” lupins varied considerably from year to year. The variations in toxicity could not be related to any environmental or other factors, except that it seemed that when the crop grew luxuriantly it was more likely to be dangerous.

It was at one time suggested that moulds growing in the hay were responsible for toxicity but this could not be substantiated. Ripe lupins were more dangerous than plants cut at an earlier stage of growth. It was deduced that the husks and seeds were the most dangerous portions of the plant. This was confirmed by an extensive series of feeding trials with sheep. Lupinosis was readily produced in the experimental animals which received husks or watery (alkaline) extracts of husks from lupin hay which had caused lupinosis when fed on the farms. Experiments with “sound” hay had been completely negative whereas the equivalent of as little as $\frac{1}{2}$ to 1 lb of husks from “affected” hay readily induced lupinosis, frequently apparent within a few days of administration. Toxic extracts were further purified and concentrated but the principle responsible for producing lupinosis does not appear to have been isolated and identified up to the present time.

The German observations on the farm indicated that, although the disease was not so severe when other feeds were given in conjunction with the “affected” lupin hay, neither supplementary fodder nor grazing prevented losses.

Recent information obtained from Europe indicates that lupinosis has ceased to become a problem since the use of sweet varieties of lupins became general. It is difficult, however, to accept this as a valid answer.

**LUPINS AND LUPINOSIS IN WESTERN AUSTRALIA**

For more than 30 years the Western Australian blue lupin (*Lupinus digitatus*) has been grown extensively as a forage plant for sheep in the sub-coastal region north of Perth. This naturalised lupin grows to perfection on the “red gum” deep sands at Gingin and Dandaragan, and also in the Geraldton district. During the past decade it has been used widely for
the development of "light lands," in this region and elsewhere where it can be grown successfully with the aid of phosphate, copper and zinc fertilisers. The plant, on present knowledge, appears to be an essential feature in the development of these poor sandy soils for stock raising.

The New Zealand blue lupin (L. angustifolius) can be grown over a much wider range of soil and climatic conditions. During recent years this species, also, has been cultivated fairly widely for forage, notably in the southern districts.

Both species have proved valuable as sheep feed and for the improvement of country with a low initial fertility. Once established, the lupins reseed annually and a good "stand" is assured almost every year in suitable situations and with the help of fertilisers where these are necessary. On the "light lands," particularly, several years of lupin growing and stocking favour pasture establishment.

Animal Husbandry.

For many years the principal use of the Western Australian blue lupin was for fattening sheep. Sheep were grazed on the lupin paddocks after the plants had seeded in November-December. In general sheep were marketed after a few weeks' grazing at the rate of four to five sheep per acre. Under these circumstances the dry roughage, leaves, husks and fine stalks were preferentially consumed in the early stages. Later the sheep fed mainly on the ripe seed picked up from the sandy soil. A "balance" was provided by edible scrub, volunteer annuals (capeweed and grasses) or by cereal crops grown for the purpose. More recently for economic and other reasons the trend has been to maintain flocks on lupin land at a lower rate of stocking.

The green plant is seldom eaten except in the young stage of growth and it is customary to thin out the lupin plants to the required density (to prevent suppression of the under-cover), by controlled grazing after the germinating winter rains. This under-cover may provide valuable grazing for sheep and cattle during the winter and spring months. The amount of non-lupin grazing available depends also on the initial fertility of the soil and on the period over which lupins have been grown.

Under the conditions of management indicated sheep are only rarely affected fatally by poisoning with lupin alkaloids, which are constantly present in both species.

Since 1950, however, lupinosis has become an increasingly serious problem throughout the region where lupins are grazed. Both species are implicated. The death rate from lupinosis may be high and the indirect losses may be considerable, because of problems of management and loss of condition in animals affected to a milder degree. In the early part of 1959, for example, on one property 70 of 500 sheep died as a result of only one week's continuous grazing on L. angustifolius; grazing L. digitatus resulted in the death of 200 of 700 sheep on another property, and on a third location in a flock of 6,000 sheep some 500 died and 1,000 were sold because of loss of condition and other signs of being affected.

CONTROL

On present knowledge, there are no control measures which can be recommended with complete confidence. It is rational to advise providing alternative fodder in order...
to lower the intake of lupins. The destruction of lupin roughage by burning the paddocks has apparently been beneficial in some instances.

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