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Facial eczema in West Australian sheep

Cover Page Footnote
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FACIAL ECZEMA in West Australian Sheep

By M. R. GARDINER, B.S. (Ch.E.), V.M.D., and M. NAIRN, B.V.Sc, M.D.A.

SINCE 1894 when the disease was first described in New Zealand, the term “facial eczema” has been used to describe a specific photosensitization of sheep in that country.

Its specific cause remained unknown for many years even though there was enough information about the disease to allow it to be included in a wider category of photosensitizing diseases, of which liver damage of some kind is the principal or essential predisposing factor.

The feature common to this class of photosensitisation is damage to the liver cells and ducts which are responsible for the excretion of the blood red cell breakdown product bilirubin. These take the bilirubin from the circulating blood and pass it to the gall bladder, from which it passes to the small intestine.

Chlorophyll from green plants eaten by the animal is broken down to certain metabolic products which are carried by the blood stream and excreted through the same liver pathway as the bilirubin. Both of these products are therefore retained in the blood stream after liver damage of the type described.

The “photodynamic” agents which result from chlorophyll breakdown are in these circumstances carried to the tiny blood vessels of the skin. Here they are activated by ultra-violet radiation of sunlight to substances which are toxic to the vessels and to the overlying skin.

There are also several plant species that are capable of synthesizing photodynamic agents which may act in the same manner, independent of liver damage.

These so-called primary photosensitizers are not, as far as we know now, of any importance in West Australian pasture species. It is rather the chlorophyll breakdown products, produced in sheep with livers damaged by various toxins, which may be expected to cause photosensitization in this State.

In this article, we are concerned with a particular kind of liver damage caused by a specific toxin. It is, in fact the same disease which has been the subject of New Zealand investigation for many years.

Although the signs of disease seen by the farmer are mainly those of skin breakdown, it is important to realise that we are dealing here with a primary liver disorder and that the photosensitizing lesions are actually a secondary manifestation.
THE CAUSE OF FACIAL ECZEMA

The pathology of the disease was first described by New Zealand workers in 1942 and again in 1959 (1, 2) but the agent causing the liver damage was not determined until 1959, when Percival in New Zealand reported the close association of outbreaks of facial eczema with the growth of a fungus (*Pithomyces chartarun*, first known as *Sporodesmium bakeri*) on dead ryegrass (3).

A great deal of work has been done since this important discovery was made, and today much is known about the toxin produced by the fungus and about the factors that influence its growth and toxicity.

Some of these factors are discussed below in connection with West Australian outbreaks of facial eczema.

It is possible that there may be some difference, in that the strains of *P. chartarun* found here would have different growth characteristics to New Zealand strains. Future additional knowledge is certain to contribute to a better understanding of this fungus and of the disease it produces, and of its relative importance as an animal pathogen in this State. Therefore, while present information is useful, it should be regarded as a guide only, and may not eventually have much relevance under the very different conditions existing here.

Facial eczema has been known for several years in the East Gippsland district of Victoria and an account of Victorian experience with the disease has recently been published (4).

New Zealand workers have usually mentioned the occurrence of facial eczema in connection with perennial ryegrass, and it would indeed appear that wilting or dead ryegrass is best suited to the growth requirements of *Pithomyces* and for the production of the liver toxin.

In Victoria, also, facial eczema outbreaks are reported most often in association with ryegrass. However, this is not invariably the case; on one property, for example, no ryegrass at all was grown.

The biological function of this species of fungus is the breakdown of the stems of the dead, or dying, grass. It is, therefore, not surprising that most outbreaks of facial eczema occur in the summer and autumn when the grasses are drying off, and when moisture and temperature conditions are ideal for the growth of the fungus.

The climatic factors most suitable for this host-parasite relationship have been extensively studied in New Zealand. Most outbreaks there occur in February and March, and it is now possible to predict with a fair degree of reliability facial eczema outbreaks in late summer, from weather information acquired in the preceding November or December.

The main factors preconditioning the soil for the rapid growth of the fungus have been carefully analysed over a number of seasons, and the present indications are that subsequent outbreaks of facial eczema in February or March may be expected if soil temperatures average 65° F. in the previous November or December, and if there are no soaking rains during these two months, so that there has been an accumulated deficit in soil moisture equivalent to about 150 points of rain.

The climatic requirements immediately before summer outbreaks were described by Mitchell and his associates (5) as follows: “The commencement of the first toxic period in a season appeared to be preceded either by two or more periods when falls of rain greater than just a few points are accompanied by, or immediately followed by, two or more successive nights with grass minimum temperatures of 54° F. or more, or alternatively by a single prolonged group of high grass-minimum temperatures with rain. Also the rain which was judged to initiate the first toxic period was itself accompanied, or immediately followed by, two or more successive nights when grass-minimum temperatures were 54° F. or higher”.

**CLINICAL SIGNS AND MAJOR LESIONS**

The earliest sign of facial eczema in a flock of sheep is the avoidance of direct sunlight and the seeking of shade.

The affected animal shows its discomfort by shaking the head or stamping the feet.

The reason for these unusual behaviour patterns is soon exhibited in the form of a thickening of the skin of the face, ears, fetlock regions and unwoollen parts of the hind legs, resulting from an increase in fluid in the skin.
As the disease worsens, due to a progressive disturbance in the circulation of the subcutaneous tissues, the superficial layers of skin degenerate and eventually slough off, leaving raw areas covered with dried exudate and scab.

The entire muzzle and/or surface of the ears may become blackened and leathery (Figs. 1, 2 and 3). Jaundice, or a yellow discolouration of the mucous membranes of the eyes and mouth, may accompany the skin lesions.

A proportion of the affected sheep die in most outbreaks, depending on the amount of toxic feed available and ingested.

The liver is invariably damaged, and many sheep in affected flocks, although they fail to develop skin lesions will be found, on post-mortem examinations to have suffered liver changes. The liver has considerable functional reserve and must be severely damaged before its capacity to remove circulating photo-dynamic agents is significantly impaired.

Typical liver lesions show as yellowish-white streaks or focal areas due to an increase in the number of small bile channels and in the connective tissue framework that accompanies them. The liver lobes may be distorted as a result of connective tissue contractures. In long standing cases, the liver may be shrunken and contracted into a rounded toughened mass of tissue (Fig. 4).

It is often very difficult to determine from a casual look at the liver, how long the toxic process has been going on, or the length of time that has elapsed since ingestion of the toxic feed responsible for it.

Sometimes the lag period is quite long, clinical signs being delayed until chlorophyll pigments are ingested and metabolized. An example of this occurred recently when a stud Dorset imported into the State from Victoria in November, 1958, developed signs of facial eczema in the following June, when green feed became available.

FACIAL ECZEMA OUTBREAKS IN WESTERN AUSTRALIA

Although cases of photosensitization of the skin of the unwoolled parts of the head, ears, and body have been reported in sheep from time to time in this State, no specific instances of facial eczema were recorded until 1959.

However, it has not been unusual to see, in histological sections of the liver of sheep studied for other reasons, bile duct changes typical of the facial eczema type, but not associated with the characteristic skin lesions.

It is, therefore, probable that toxic grass is often eaten by sheep in Western Australia, but only occasionally produces enough liver damage to disturb health.
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This probably happens only when favourable climatic conditions are prolonged sufficiently to cause rapid spore growth of the fungus.

In early August, 1959, a ewe with typical facial eczema from the Northam district was found to have severe liver damage identical to "facial eczema" liver. This turned our attention to the possibility of the disease existing in Western Australia.

In June, 1960, confirmation was obtained in some yearling sheep, from the Two People Bay area along the south coast, showing face and ear lesions.

The first outbreak definitely recognized as facial eczema was brought to our notice by Dr. H. W. Bennetts on a property at Cunderdin, in the wheat belt. Fifteen adult ewes out of a mob of 700 were affected in mid-July, 1960, with varying degrees of facial eczema. Several had to be destroyed.

Very marked typical liver lesions were found in the ewes when studied at the Animal Health Laboratory. These had been grazing a paddock containing brome grass, sub clover, oat stubble and Wimmera rye, the latter dominating in some patches.

There was quite a lot of dead grass left over from the previous year (due to understocking in 1959), and there were unusual fogs and light, misty rains in the district during the month before the outbreak.

Pithomyces chartarum was isolated from samples of dead Wimmera rye, and mild liver lesions were produced in rabbits which were fed cultures of it.

The most completely investigated West Australian outbreak of the disease so far occurred at the Wongan Hills Research Station in July, 1961, when 20 to 30 per cent of a flock of 383 full-mouth ewes, and a proportion of their autumn-dropped lambs, were affected. From 5 to 7 per cent of the ewes were very severely affected, and two died.

From late April until July 13, when the first cases were noticed, the flock had grazed a pasture consisting predominantly of brome grass and sub clover, with a little silver grass and wild turnip. There was no ryegrass and little residual grass from the previous season, but in one part of the paddock considerable quantities of old cereal hay had been left to decay.

Numerous spores of the fungus *P. chartarum* were found in samples of face wool from affected sheep, and a heavy concentration of spores occurred in the old cereal hay. This finding, paralleling Victorian experience, indicated that, under the proper conditions, plant material other than ryegrass will support the growth of this organism and may thus be responsible for facial eczema outbreaks.

Wongan Hills has a mean annual rainfall of 14 inches, most of it between May and October. At this station daily maximum and minimum air temperatures, daily minimum terrestrial temperatures, and daily rainfall figures are recorded. These data for May, June and the first half of July are presented in the appendix table.
Applying the New Zealand criteria discussed in the first part of this paper, it may be said that the climatic conditions in May were unfavourable for the growth of the fungus, but that the period between June 12 and June 15 satisfied the postulated requirements. Between these two dates 125 points of rain fell and the ground temperature did not fall below 54° F. A preconditioning period probably occurred in early April when heavy falls of rain and high ground temperatures were recorded.

We think, therefore, that the main fungus growth occurred in the old cereal hay lying in the paddock, about the middle of June, and that the sheep grazing in or near these piles of cereal hay suffered progressive liver damage over the next two or three weeks.

Sporadic outbreaks of facial eczema will no doubt continue to occur in Western Australia. On the basis of current knowledge, the following requirements would probably have to be met:

1. The presence of considerable amounts of dead ryegrass, cereal hay, or other grasses on the paddocks in late summer and early autumn (from understocking, or other local causes).

2. Significant falls of rain in April, when ground temperatures would be high.

3. Additional rainfall of ½ to 1¾ inches of rain in May, June or July, accompanied by several days of minimum ground temperatures higher than 54° F.

Other Causes

Photosensitization can result in Western Australian sheep from causes other than fungus. Phenothiazine drenching of sheep with damaged livers is often responsible, since the drug is not detoxified and is then capable of acting as a photodynamic agent. Phenothiazine should not therefore be used in sheep suspected of having sustained prior liver damage or if cases of facial eczema have appeared in a flock. Chronic lupinosis is often associated with photosensitization when the sheep are subsequently put onto green feed.
ACKNOWLEDGMENTS

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REFERENCES


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