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RYEGRASS STAGGERS IN SHEEP AND CATTLE

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SUMMARY

Ryegrass staggers has been shown to be caused by a toxin present in or on pasture, but neither the toxin nor its source has been unequivocally identified. This paper discusses various aspects of ryegrass staggers such as the development of symptoms, seasonality, and the effect of pasture type and stocking rate on its occurrence.

INTRODUCTION

Gilruth (1906) described the sporadically occurring neurological disorder in farm animals known as ryegrass staggers, because of the staggering gait of affected animals, coupled with the observation that outbreaks tended to be associated with ryegrass-dominant pastures. It occurs during summer and autumn, and sheep, cattle and horses of all ages and either sex are affected. Outbreaks have been reported in most parts of New Zealand, although they appear more prevalent in areas subject to dry summers. Affected stock cannot be mustered, moved to fresh pasture or, in the case of dairy cows, brought in for milking. It thus places severe limitations on stock management and may upset grazing management, mating and milking programmes. Growth rates, however, appear largely unaffected. Stock losses, although not high, are common, resulting largely through misadventure. Recent work has also shown that plasma testosterone concentrations were markedly reduced in six out of nine rams fed toxic pasture (Peterson *et al.*, 1978). Many suggested causal agencies — *e.g.*, mineral and vitamin deficiencies (Cunningham and Hartley, 1959) and ergot sclerotia (Thornton, 1964) — have been eliminated. On the basis of feeding experiments, Cunningham and Hartley (1959) suggested that the causal factor was a toxin in the grass while more specifically Latch *et al.* (1976) suggested a mycotoxin. Further, di Menna *et al.* (1975) suggested that tremorgen-producing *Penicillium* species, present in soil, are involved. However, neither the causal toxin nor possible fungus or fungi have been unequivocally identified.

The results reported in this paper have been collected over the past three seasons from work aimed at trying to determine the causal factors of ryegrass staggers.

TOXIC PASTURE

Feeding trials have confirmed the earlier work of Cunningham and Hartley (1959) that the causal toxin is either in or on pasture. Pasture was collected from various outbreaks using rotary or reel mowers and then frozen (-10°C). When this material was thawed and fed to unaffected animals, it caused them to develop the disorder. There was little or no apparent loss of toxicity when the frozen material was thawed and either dried or washed vigorously with water and then dried at 25 to 30°C , but oven drying at 100°C appeared to reduce or destroy the toxin.

Unfortunately, neither guinea-pigs, rats nor rabbits appear to be affected by the toxic material, precluding the possibility of determining what fraction of the collected material contains the toxin. Attempts have been made by Dr E. P. White of Ruakura to extract the toxin, but, as yet, this work has met with little success.

In view of the suggestion that soil-borne fungi may be the cause of ryegrass staggers, soil, taken to a depth of 0.2 cm, was obtained from a number of outbreaks, oven dried at 25 to 30°C and given by stomach tube to lambs. However, none of the lambs developed any symptoms despite daily soil intakes of 400 g given over 10 to 14 days.

SYMPTOMS

Affected animals if undisturbed appear normal, but, when disturbed, they develop head and shoulder tremors in mild cases, and inco-ordination and collapse in severe cases. From feeding trials, it has been established that unaffected animals will develop mild symptoms within 7 days and severe symptoms within 14 days of admission to known toxic pasture. However, this varies, probably depending on animal susceptibility and intake of the toxin. Recovery takes place within one to two weeks of being transferred to safe pasture. Animals appear to need a continuous intake of the toxin for the symptoms to develop and be maintained, suggesting that the causal toxin is either a cumulative neurotoxin or one which exerts a cumulative neurological effect. No lesions pathognomic of ryegrass staggers have been found. Mason (1968) described cerebellar lesions involving Purkinje cell axons, but concluded that the lesion was not the specific effect of some toxin on the Purkinje cells but secondary, arising from the effects of the symptoms.

PERIODS OF TOXICITY

It would appear that pastures become toxic during two main periods, early summer and late summer/autumn. However, these periods can merge, as farmers report stock being affected from December to April. Generally, the later outbreaks are more widespread and severe and are usually triggered by light rain following a dry period. Early outbreaks do not appear to be characterized by any weather pattern. From a trial where unaffected animals were introduced on to a toxic paddock at regular intervals it has been found that areas can remain toxic for as long as 38 days. This contrasts with facial eczema in which pastures are usually toxic for periods of a week or less. Outbreaks generally wane when rain stimulates ample fresh growth.

STOCKING RATE AND PASTURE TYPE

The influence of stocking rate and pasture type in the aetiology of ryegrass staggers was highlighted by an outbreak in autumn 1976, on a self-contained farmlet trial, which was designed to assess the effect of pasture renovation and stocking rate on the control of soldier fly (*Inopus rubriceps*). Plots of the original and renovated pasture were each stocked at three rates with either sheep or cattle, making a total of 12 farmlets. The stock were rotationally grazed and, during the outbreak, the number of affected animals on each farmlet recorded. As the outcome was similar for both sheep and cattle, only the sheep results have been presented (Fig. 1). The outbreak started in early March and reached a peak of incidence and severity early in April. The data show a marked difference in the number of animals affected on the two pasture types. At the peak of the outbreak 65% of the sheep on the renovated pasture treatments were severely affected, compared with only 15% on the original pasture treatments.

The renovated pastures contained 74% ryegrass in the green material compared with 36% in the original pasture. This agrees with the general observation that outbreaks are more prevalent on ryegrass-dominant pastures.

Within the renovated pasture treatments, the incidence of staggers increased with an increase in stocking rate. The increase was greater at the beginning of the outbreak than at the peak, when the number affected at the medium and high stocking rates were similar. This was attributable to the development of high grazing pressures at the medium stocking rate as the result of slow pas-

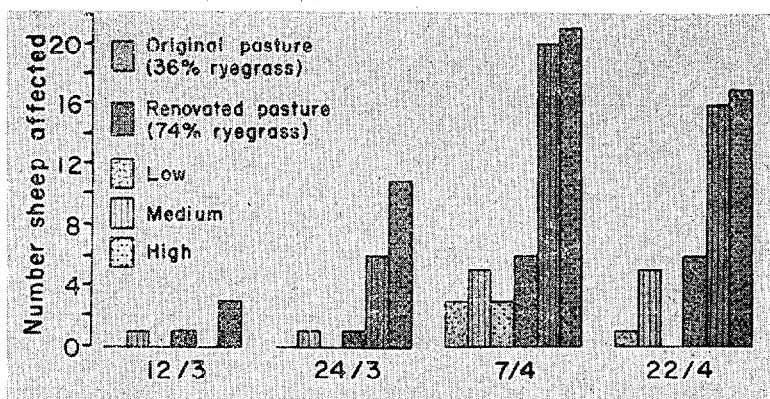


FIG. 1: Effect of stocking rate and pasture type on the incidence of ryegrass staggers.

ture growth due to dry weather. No stocking rate effect was apparent on the original pasture.

Soil content of the faeces for each group of sheep was measured at frequent intervals during the period of observation using the technique of Healy (1968). Soil intakes estimated from these data were generally less than 10 g/head/day and were not correlated with the incidence of staggers. It would therefore seem unlikely that differential ingestion of soil fungi was responsible for the observed difference in staggers.

Observations were also carried out during autumn 1977, and the results confirmed those of the previous year, except that the incidence was greater at the low and medium stocking rates than at the high. This appeared to be because low pasture availability at the high stocking rate reduced herbage intake below some critical level.

The results therefore suggest that animals are at most risk when grazing ryegrass-dominant pastures sufficiently hard to ingest appreciable amounts of dead material but not so hard that intake is severely restricted. They further suggest that ryegrass staggers may prove increasingly troublesome as pasture improvement continues and stocking rates rise.

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