MYCOTOXIN POISONING IN GRAZING LIVESTOCK

Dr Neale R. Towers
Toxinology & Food Safety, AgResearch, Ruakura, Hamilton, New Zealand

Abstract
While mycotoxins found as contaminants of grain and pulse crops are well known to cause health and productivity problems in both livestock and humans the role of mycotoxins found in pastures in causing health, welfare and production problems in livestock is less well recognised internationally. However, in New Zealand, and in other countries with primarily a pastoral farming system, several mycotoxicoses cause significant production loses and research has focussed on understanding the causes of the diseases and developing control methods. "Facial eczema" caused sporidesmin - a hepatotoxin found in the spores of the saprophytic fungus *Pithomyces chartarum*, is the best known of these problems and a number of control methods have been developed. These include the use of spore counting to identify toxic pasture, fungicides to reduce spore production, dosing with zinc salts to protect animals against the toxic effect of sporidesmin, and breeding for increased resistance to the toxin. "Ryegrass staggers" is caused by the presence in perennial ryegrass of an endophytic fungus that sproduces a number of mycotoxins, including the tremorgenic neurotoxin Lolitrem B which is responsible for the animal staggers and members of the ergot alkaloid group which can cause vaso construction leading to heat stress and reduced milk yields in cattle. Tall fescue cultivars from the USA often contain a related endophyte that also produce ergot alkaloid toxins causing animal health and production problems particularly heat stress in summer and loss of ears, tails and feet from gangrene in the winter. The control of these diseases is limited to avoiding grazing such pastures when they are toxic and replanting pastures with less toxic species or cultivars. Eliminating the endophytic fungi is not always successful as in some environments the presence of the fungus is essential to provide protection against insect attack or greater resistance to drought stress. Research is aimed primarily at developing cultivars containing selected endophyte strains that produce low levels of the toxins causing animal health problems while still producing high levels of the compounds protecting against insect attack or environmental stress. The third well defined mycotoxiosis recognised in New Zealand is the reduced ewe fertility caused by the presence in pastures in autumn of the oestrogenic compound, zearalenone, produced by several species of *Fusarium*. In culture *Fusarium* species also produce a number of the toxins, including the trichothecenes, and current research is aimed at discovering whether these toxins also occur in pastures in autumn and, whether they are the cause of the poor growth and health of young stock known as "autumn ill-thrift".

Introduction

While mycotoxins found as contaminants of grain and pulse crops are well known to cause health and productivity problems in both livestock and humans the role of mycotoxins found in pastures in causing health, welfare and production problems in grazing livestock is less well recognised internationally. However, in New Zealand, and in other countries with primarily a pastoral farming system, several mycotoxicoses cause significant production loses and research has focussed on understanding the causes of the diseases and developing control methods.

The diseases include, facial eczema, caused by sporidesmin - a hepatotoxin found in the spores of the saprophytic fungus *Pithomyces chartarum;* ryegrass staggers and fescue

toxicosis (known also as summer slump) caused by the presence in perennial ryegrass and in tall fescue of endophytic *Neotyphodium* species; paspalum staggers from grazing paspalum grasses with ergotised seedheads; and zearalenone infertility in ewes grazing pastures with heavy growths of *Fusarium* species capable of producing the estrogenic compound zearalenone.

Several less well defined intoxications, and animal health and production problems which may be due to mycotoxins, have also been reported. These include Bermuda Grass toxicity and autumn ill-thrift in New Zealand.

Facial eczema

Facial eczema is a disease of sheep, cattle and deer, but it can also affect goats, llamas and alpacas. The disease occurs most often in New Zealand but has been reported in Australia, South Africa, France, Uruguay, Argentina and Brazil, and there is evidence it may have also occurred in the USA and Netherlands. The disease is caused by the ingestion of spores of the saprophytic fungus *Pithomyces chartarum* containing sporidesmin, which is a potent hepatotoxin which also causes damage to the bladder and mammary gland. *P. chartarum* is found in all temperate climate zones of the world where it grows on the dead vegetable matter at the base of the pasture. Facial eczema outbreaks occur when weather conditions suitable for rapid growth and spore production (warm, humid weather and light rain) are combined with intensive grazing practices that encourage the ingestion of large numbers of spores.

Facial eczema is named for the visible sizes of photosensitisation affecting exposed non-pigmented uncovered areas of skin. The first sign of intoxication may be a transient diarrhoea and inappetence soon after the animals are first exposed to the toxin. In lactating dairy cows there may be a sudden pronounced fall in milk volumes. These changes occur immediately after exposure to sporidesmin and before signs of photosensitisation and clinical disease are normally seen and therefore are often not recognised by the farmer.

The clinical signs of facial eczema are typical of a secondary photosensitisation arising from liver damage and occlusion of the bile ducts, thus the photosensitisation occurring in animals grazing saponin-containing plants such as *Tribulus terrestris*, *Panicum* grasses., and *Brachiara decumbens* may be mistaken for facial eczema. Therefore it is not sufficient to identify the presence of *P. chartarum* spores in pastures where photosensitisation has occured to diagnose facial eczema. It must be confirmed that the *P. chartarum* produces sporidesmin (many South and North American isolates do not produce toxin) and that the liver pathology and histology is typical of sporidesmin.

Not all animals with sporidesmin-induced liver damage will show clinical signs of the disease. Clinical signs appear only in those animals in which liver damage is widespread, or major bile ducts are blocked so that excretion of bile is severely impaired and bile circulates back into the bloodstream. In all but the most severe outbreaks the majority of the animals will show little, or no, outward sign of the disease but analysis of blood enzyme levels will reveal that many of the animals have suffered liver damage.

Production losses from facial eczema arise not only from animal deaths and the condemnation of jaundiced carcasses but also from loss of body weight, reduced fertility in sheep and lower milk yields in dairy cattle. Reduced ewe fertility in the year of a facial eczema outbreak has been widely reported and is perhaps not unexpected as mating often coincides with the period of highest risk for facial eczema (1), but there is also experimental evidence facial eczema can

reduce lifetime lambs production in affected ewes by up to 25 percent (2). In dairy cattle the reductions in milk yields following liver damage and the onset of clinical disease, is well known, but in addition to these production losses experimental data shows sporidesmin intakes too low to cause detectable liver damage can reduce milk volumes significantly (Towers unpub) causing even greater production losses.

Production losses in NZ have been variously estimated to range from $10 - $106 million, depending on how severe the disease has been in that year.

Ryegrass Staggers and Fescue Toxicosis

These two diseases are closely related as they are both caused by the presence of endophytic fungi which grow intercellularly within the host grasses in a mutually beneficial association. Ryegrass staggers is caused by the presence of *Neotyphodium lolii* (formerly *Acremonium lolii*) in perennial ryegrass (*Lolium perenne*) and fescue toxicosis by the presence of *N. coenophialum* in tall fescue (*Festuca arundinacea*). *N. lolii* produces at least three major groups of toxins: tremorgenic indole diterpenoids such as paxilline and lolitrem B; ergopeptine alkaloids such as ergovaline; and the insect herbivory repellent compound peramine. *N. coenophialum* does not produce significant amounts of the tremorgenic toxins but does produce the ergot alkaloids, peramine and high levels of lolines. Ryegrass staggers and fescue toxicosis are the best known of the endophyte related animal health and problems, occurring wherever either perennial ryegrass or tall fescue are predominant pasture species. However there are other well known problems including sleepy grass syndrome (USA) (3), drunken horse disease (China) (4) as well as less well defined problems associated with livestock grazing grasses native to Australia, New Zealand and South America infected with endophyte *Neotyphodium* or closely related (5; 6; 7; 8).

Ryegrass staggers is the most obvious symptom of the endophyte toxins found in ryegrass. The effects of lolitrem B on nerves and muscles lead to loss of appetite and to unco-ordinated movement. Affected stock may drown in troughs or dams. Others may fall and be unable to get up. Occasionally stock may die of extreme thirst. Even where the effects are not severe, the difficulty of moving stock makes farm management difficult. Horses and deer are very sensitive to ryegrass staggers, but sheep and cattle can also be seriously affected. The effects of ryegrass staggers are most seriously in summer dry areas, as toxin concentrations are highest at this time while poor pasture growth encourages animals to graze pasture hard increasing the intake of the basal sheath region of the plant which contains the highest toxin concentrations.

Less spectacular, but perhaps more serious economically, is the reduction of growth rates and production caused by endophyte toxins, even when no symptoms of ryegrass staggers appear. The effects are not consistent and are difficult to predict, but grazing ryegrass with endophyte has reduced milk production by up to 2 litres per cow per day. Weight gains by lambs grazing high-endophyte ryegrass in spring have been reduced almost 40% compared with lambs on non-endophyte ryegrass. Sheep grazing high-endophyte ryegrass also often have more dags [faecal contamination of wool]. Additionally, because the endophyte in ryegrass also produces ergopeptine alkaloids similar to those in tall fescue, animals grazing ryegrass pastures in ryegrass with high ambient temperatures may also suffer heat stress problems similar to those encountered in fescue toxicosis. (9).

Tall fescue cultivars infected with *N. coenophialum* are planted very extensively in South Western USA, and consequently problems caused by the presence of the ergot alkaloid toxins

are well known, and estimated to cause cattle production losses in excess of $600 million annually (10). Three distinct disorders (summer slump or summer toxicosis, fescue foot and fat necrosis) have been described in cattle suffering from fescue toxicosis. The latter two disorders are minor components of the syndrome, with fescue foot being similar to one form of ergotism (lameness and sloughing of the hooves). Summer toxicosis occurs when temperatures exceed 25 °C and is characterised by rough hair, fever, increased respiration rate, and reduced feed intake, weight gain, circulating prolactin levels, milk production and reproduction (agalactia and fecundity). Many of these symptoms occur in affected sheep and horses, especially the reproductive effects (11). Fescue toxicosis, fescue foot, (gangrene of the extremities – also associated with ergotism) and the heat stress associated with grazing endophyte-infected ryegrass are a result of the vaso-constrictive activity of the ergot alkaloids. Vaso-constriction of peripheral blood vessels, leading to reduced blood flow to the skin, reduces heat loss when ambient temperatures are high leading to heat stress, while in winter cooling of the extremities aggravates the blood flow problem leading to anoxia, tissue death and the onset of gangrene.

Paspalum Staggers

Paspalum staggers is a problem of sheep and cattle grazing tropical or subtropical *Paspalum* spp. which have been allowed to flower, and the inflorescence has been infected with sclerotia of *Claviceps paspali*, although cases have been reported in livestock grazing other grasses infected by *C. paspali* or *C. purpurea* – see Lacey (12). Paspalum staggers is caused by indole diterpenoid tremorgens, known as paspalinine and paspalitrems A-C, that are structurally related to paxilline and the lolitrems and, as might be expected, the symptoms of paspalum staggers are very similar to those of ryegrass staggers.

Zearalenone Infertility

A number of *Fusarium* species capable to producing the oestrogenic compound, zearalenone, are common in pasture microflora (13) and analysis of pasture samples from throughout New Zealand revealed that relatively high (0.5-5 mg/kg dry matter) concentrations of zearalenone are common through the autumn months (February-May) coinciding with the sheep mating season. (14).

Dosing studies have shown that zearalenone intakes greater than 1 mg per day adversely affect the reproduction cycle in sheep, with the effects becoming more severe at higher intakes and/or longer periods of exposure (15; 16).

Ingestion of zearalenone contaminated pastures can be readily detected by analysing urine for the presence of the metabolites, α- and β-zearalenol, which increase in concentration in proportion to the amount of toxin ingested. Analysis of urine samples from throughout New Zealand suggest that more than 30 percent of sheep flocks are exposed to sufficient zearalenone to reduce ovulation rates, and thence lamb birth rates, causing significant production losses in affected flocks (14).

Zearalenone producing *Fusarium* are found throughout the world and it is to be expected that zearalenone contamination of pastures will occur in other countries with similar climate and pasture management to that of New Zealand. This possibility is currently being researched in both Australia and Argentina.

Other Mycotoxin Problems

There are a number of other animal health and production problems which may be caused by mycotoxins. But before they are accepted as being mycotoxicoses confirmation is required that not only are toxin-producing fungi present in pasture but also that sufficient toxin is being produced and consumed by the animals to cause an intoxication. Furthermore it should be demonstrated that administration of the toxin causes the same symptoms and pathology as are observed in field outbreaks of the disease.

(i) Zearalenone and cattle infertility problems.

In New Zealand cattle also graze zearalenone-contaminated pastures and the analysis of blood, urine and pasture samples from herds reported to have fertility problems showed that the pasture zearalenone, urine and blood zearalenol concentrations in these samples was higher than in similar samples from herds with acceptable reproductive performance (17). However, as these samples were collected in January or February, some months after mating in spring (September, October), when veterinary examination revealed the low pregnancy rate results, the data do not necessarily reflect a cause and effect relationship between the high zearalenol levels in the blood and the fertility problems. Indeed given that pasture zearalenone levels are generally low when cattle are mated (September, October), it seems that a direct effect on ovulation rates, similar to that found in sheep, is unlikely unless cattle are exceptionally sensitive to the effects of zearalenone. Furthermore, any effect is not simply a matter of exposure versus non-exposure to the toxin as analysis of blood samples from low and high fertility herds show that both contain zearalenol residues in their blood throughout the year. Although the low fertility herds generally had higher concentrations this was only statistically significant during the October-December and January-February sample collection periods. Thus a cause and effect role for zearalenone (and/or other *Fusarium* mycotoxins) in affecting ovulation, conception or maintenance of the pregnancy after conception, has not yet been proven.

(ii) Autumn ill-thrift

Many of the *Fusarium* species found in pasture are capable of producing not only zearalenone but the trichothecene mycotoxins (18) and at the time zearalenone levels are high in autumn many young animals suffer an “ill thrift” and fail to grow. The possibility that the *Fusarium* are producing trichothecenes in the pasture is sufficient quantity to affect animal health and performance is being investigated, and to this end we have developed ELISA assays for the deoxy-nivalenol (DON) and nivalenol (NIV) trichothecene groups. However preliminary studies in which extracts of *Fusarium* culture material containing DON or NIV were dosed to sheep suggest that pasture concentrations of these toxins would need to 10-fold higher (i.e. 10-50 mg/kg DM) than those found for zearalenone before they would affect the health or growth of ruminant animals (19) and to date we have not detected such high concentrations. An alternative explanation might be that the poor health and growth reflects the effects of endophyte toxins present in ryegrass-dominant pastures.

(iii) Bermuda Grass (*Cynodon dactylum*) tremors

Outbreaks of tremors in cattle grazing Bermuda grass have occasionally been reported, and the similarly of clinical symptoms has led to suggestions that tremorgenic mycotoxins might be involved. However the sporadic occurrence of the disease has made it difficult to investigate and the origin of the problem is unresolved. It is possible that more than one cause of intoxication is involved. *Cynodon* spp. can be infected with *C. purpurea* and grazing ergotised seed heads could lead to tremors. In addition *Cynodon* species have been reported to contain toxic levels of cyanogenic glycosides (21; 22).

Preventing Mycotoxicoses

In general there are three strategies for preventing mycotoxicoses:

1. preventing or reducing toxin intakes,
2. protecting the animal against ingested toxins and
3. breeding more resistant animals

and examples of the practical use of all these strategies exist.

Reducing toxin intakes can be achieved by identifying and avoiding toxic pasture and by reducing toxin production, either by the use of fungicides or modifying the fungal population.

Reducing toxin intakes

(1) Avoiding toxic pasture.

As New Zealand isolates of *P. chartarum* are all sporidesmin producers and, as the toxin is found primarily in the spores, the risk of facial eczema can be readily determined by monitoring the number of the distinctive and easily identified spores in the pasture (23). This technique is widely used by veterinarians and by farmers. However, in those countries where not all *P. chartarum* isolates produce sporidesmin (24) spore counting may overestimate the risk of facial eczema. But for most toxin producing fungi there is no correlation between spore numbers or mycelial mass and toxin concentrations and identifying toxic pastures must depend on the direct analysis for the toxin concerned. ELISA or HPLC methods have been developed for detecting sporidesmin, zearalenone, deoxynivalinol, nivalenol, lolitrem, paxilline, ergopeptine alkaloids, peramine and adapted to the analysis of pastures, serum and urine samples. The relative speed and simplicity of ELISA make them particularly suitable for screening large numbers of pasture or urine samples (25; 26; 27; 28; Garthwaite unpub.).

Toxic pastures may also be recognised (suspected) on the basis of weather patterns in the case of facial eczema or by visual signs in the case of *Claviceps* infestation of grass seed heads (eg paspalum staggers). *P. chartarum* growth and sporulation is associated with high humidity, high overnight temperatures and 4-6 mm rain and in New Zealand these conditions are widely recognised prompting the adoption of control measures, such a moving stock to pastures with histories of low spore counts, and initiating fungicide spraying or zinc drenching. *Claviceps* infections of grass seedheads are readily recognised and either stock can be moved to other pastures or, as the toxins are confined to the seedhead, the pastures can be made safe by mowing and removing the seedhead material.

Reducing grazing pressure (number of animals per hectare) can also reduce toxin intakes as the highest toxin levels are generally in those parts of the pasture closest to the ground. *P. chartarum* and the *Fusarium* spp. are both saprophytes growing on the dead litter, while the endophyte toxins are in highest concentration in the basal leaf sheath and under low grazing pressure livestock avoid ingesting this material.

During periods when toxin levels in pasture are high feeding conserved fodders or crops can reduce toxin intakes, so long as access to the toxic pasture is restricted. Moving stock from ryegrass or tall fescue pastures to swards dominated by other grasses can rapidly alleviate ryegrass staggers or fescue toxicosis (29; 30). Feeding crops (maize, brassicas, chicory, red or white clover, hybrid millets etc) is, in many instances, the only option for preventing the ingestion of zearalenone.

(ii) Reducing toxin production.
Benzimidazole fungicides (eg Benlate®, Topsin® M-4A Carbendazin®, Bavistin® DF) have been used successfully to reduce *P. chartarum* spore numbers to control facial eczema. These fungicides act by inhibiting germination of spores present at the time of spraying, reducing the spore production by subsequent generations of mycelia by 55-65 percent. In most seasons, if the fungicide is applied while spore numbers are low, this is sufficient to prevent spore numbers rising to toxic levels. Fungicides, at economic rates, have not been effective in controlling *Fusarium* species in pasture, having no effect on zearalenone production even at application rates 10-fold those used for controlling *Pithomyces*. Application of fungicide to pasture has no effect on the endophytic *Neotyphodium* species causing ryegrass staggers or fescue toxicosis but, in conjunction with specific storage conditions, fungicides have been used to remove endophyte fungi from stored seeds to enable the sowing of endophyte-free, and therefore toxin-free, pastures.

However, in most instances, the options of using endophyte-free perennial ryegrass or tall fescue as a way avoiding ryegrass staggers or fescue toxicosis has not proved successful as the presence of the endophyte, and its metabolites and toxins, provide growth and survival advantages to the host plant. Endophyte infected grasses show better survival under climatic stress such as drought and are more resistant to insect or nematode attack. Pastures sown with endophyte-free seed quickly revert to endophyte-infected pastures as seed in the soil germinate and endophyte-free plants die.

(iii) Modifying the fungal population.
A more promising approach has been to generate ryegrass and tall fescue cultivars infected with *Neotyphodium* endophytes selected as producing no, or only low levels, toxins adversely affecting grazing livestock (lolitrem B; ergopeptine alkaloids) while continuing to produce toxins providing protection against insects (peramine, lolines) (31; 32). Two such selected endophytes (ARI in perennial ryegrass; MAX-Q in tall fescue) are now being widely tested in commercial grass cultivars which will be commercially available in New Zealand and the USA in 2001 (9). Animals grazing pastures with modified endophytes perform better than those grazing pastures infected with wild-type endophytes. The presence of the selected endophytes confers many of the advantages of the wild-type endophyte although the novel endophyte:grass associations appear to be less resistant to some insects.

Biocontrol of facial eczema may also be possible. Although in New Zealand almost all spore-producing *P. chartarum* isolates produce sporidesmin a few isolates produce neither sporidesmin nor any other readily detected toxins (33). The possibility of developing a biocontrol method depending on introducing large numbers of these atoxigenic spores into the pasture prior to the facial eczema season so that they become the dominant population is under investigation. Very good control has been obtained in trials, with an 80 percent reduction in toxin levels on treated pasture plots (34). In a subsequent trial there was no detectable liver damage in lambs grazing a biocontrol treated plot whereas the adjacent, untreated control pasture, was highly toxic and all lambs grazing on this plot suffered severe liver damage (Collin unpub data). However, difficulties have been experienced in developing an inoculum that provides protection at a cost per hectare that is financially viable. An additional problem observed in our trials has been the low survival and persistence of the atoxigenic strains introduced into the pastures. Soon after application of the inoculum 80-90 percent of isolates were atoxigenic, but 4 months later the percentage of atoxigenic isolates was 53 percent and 15 months later only 4 percent (34). However long term survival

of the atoxigenic isolates may not be a problem in those countries which naturally have a high proportion of atoxigenic strains resident in their pastures (24).

Protecting animals against ingested toxins

Although the protection of animals against ingested toxins has been a major focus for research in facial eczema, ryegrass staggers, fescue toxicosis, and zearalenone infertility the only practical method for protecting livestock against ingested toxins developed to date is the use of zinc salts to protect against facial eczema. The administration of very high doses (15-30 mg Zn per kg liveweight per day) of zinc as either zinc oxide or zinc sulphate at, or before, the time the animals ingest sporidesmin can reduce any sporidesmin-induced liver damage and production losses by 60-90 percent. There is strong evidence zinc dosing is effective because zinc forms a stable mercaptide with reduced sporidesmin, removing it from the autoxidation cycle [sporidesmin-reduced sporidesmin] that leads to the cascade generation of reactive oxygen radicals that cause cell damage and ultimately cell death (35).

Drenching with slurries of zinc oxide, or adding zinc sulphate to the water supply, are now the most widely used facial eczema control methods. As the best protection is provided by daily drenching, and this is not practical for large herds or flocks, slow release intraruminal boluses which provide protection to sheep for 6 weeks (36) or cattle for 4 weeks have been developed and sold commercially under the brand name The Time Capsule™.

Attempts to block the action of the tremorgenic indole diterpenoids and the ergopeptine alkaloid toxins produced by endophytic *Neotyphodiums* by administering various pharmaceutical agents have not so far produced practical on-farm control methods. Similarly attempts to immunise animals against sporidesmin or zearalenone have also failed. Despite high titres of toxin-binding antibodies circulating in the blood, immunised animals were more susceptible to the toxin challenge than un-immunised animals (37; 38; Towers unpub.). This is in contrast to the protection Australian researchers have demonstrated for phomopsin, a mycotoxin found on lupin stubble (39). Although immunisation appears to be a possible control method the 1:2 stoichiometry of antibody:toxin binding means it is unlikely that sufficient circulating antibody could be produced to bind the 1-10 mg of toxin ingested daily by livestock grazing pastures contaminated by sporidesmin, zearalenone, or the endophyte toxins.

However the use of Androvax™, a proprietary anti-androstenedione vaccine [AgVax Developments Ltd., Upper Hutt, New Zealand] to increase ovulation rates to counter the depression in ovulation rates caused by zearalenone has met with limited success. Although ovulation and conception rates in Androvax treated ewes dosed with zearalenone were higher than those in ewes dosed with zearalenone alone and similar to control ewes at the end of the mating period, conception to the first cycle of mating was lowered and lambing was delayed (29).

Breeding for resistance

The very wide range of individual responses to a toxin challenge suggests some animals are inherently more resistant to the toxin the others. This raises the possibility of breeding animals with a greater resistance to the disease although, in developing a breeding programme, it is important to ensure that the selected trait is not negatively linked to other desirable production traits. A heritable resistance to sporidesmin has been identified in both sheep and cattle (40; 41), and a service performance testing rams for facial eczema resistance has been available since 1985. The performance test depends on dosing rams, selected as

potential flock sires, with sporidesmin and using the analysis of serum and gamma-glutamyl transferase (GGT) concentrations to detect and quantitate the severity of any subsequent liver damage. Resistant animals show no increase in serum GGT levels. Breeders who have used the sporidesmin service since its inception have increased the resistance of their flocks to sporidesmin 4-5 fold.

Despite the high heritability of facial eczema resistance in dairy cattle, and the opportunities for rapid dissemination of the genes through artificial insemination, the dairy industry in New Zealand has not adopted selection for facial eczema resistance as a control measure for this disease.

Similarly there is a heritable resistance to ryegrass staggers and while the difficulties in isolating sufficient lolitrem B from ryegrass seeds prevents the development of a performance test for resistance similar to that used for selecting facial eczema resistant sires, some progress has been made in breeding for ryegrass staggers resistance using data from natural outbreak of the disease (42). Interestingly, despite sporidesmin and lolitrem B having very different chemical structures and modes of action, selection for facial eczema resistance has increased resistance to ryegrass staggers in experimental flocks, and selection for ryegrass staggers resistance has increased resistance to facial eczema. These observations suggest through the observed resistance to these toxins may have multiple origins, one of which is common to both facial eczema and ryegrass staggers resistance. In the absence of a practical performance test selection of ryegrass staggers has been adopted by only one or two individual sheep breeders.

It appears that there have been no similar studies of the heritability of resistance to the ergopeptine alkaloids in ryegrass and tall fescue, but a study in mice (43) has demonstrated a selection response in mice suggesting that selection for resistance may also be feasible in livestock.

Summary
A number of mycotoxicoses including facial eczema, ryegrass staggers, paspalum staggers, fescue toxicosis and zearalenone infertility affect grazing livestock causing death and reducing productivity. In most instances there are no antidotes and for most mycotoxicoses control measures depend primarily on identifying and avoiding toxic pastures. However research has lead to the development of a number of control measures for particular diseases, especially facial eczema.

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