Mycotoxins in New Zealand livestock production

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INTRODUCTION

Mycotoxins, or fungal toxins, from pasture are a major source of contamination in the diet of grazing livestock in New Zealand. A number of conditions need to be satisfied before a particular mycotoxin may become a serious production problem. These are: the weather conditions for toxin production, the quantity of toxin intake by stock per day relative to what may be a toxic dose, and grazing without having alternative diets such as crop, silage or concentrates. These conditions appear to be met more commonly in New Zealand than elsewhere. As a result, New Zealand has more production problems associated with a range of mycotoxins than elsewhere, and it is unlikely that solutions to some of the mycotoxic diseases (especially facial eczema) will be found by science teams offshore.

The present Contract for the New Zealand Society of Animal Production has been arranged to review some areas of mycotoxic research and their relevance to livestock production in New Zealand. With a limited time on the Conference programme, we cannot hope to review all subjects fully. Neale Towers introduces the whole area, with a general review describing facial eczema, ryegrass staggers, fescue toxicosis, paspalum staggers, zearalenone infertility, and other mycotoxic diseases. One of these, zearalenone infertility, is then reviewed in greater depth by Smith & Morris (2006). Two of the other main diseases have been reviewed by other authors in recent years: facial eczema (Smith & Towers, 2002) and ryegrass staggers (e.g., Fletcher, 1999, 2004).

Two short papers then follow in this Contract. The first is by Cullen *et al*. (2006), and gives an update on the animal genetic approach to facial eczema in dairy cattle, documenting the considerable increase in record collection in the last two years to rank dairy sires for resistance. The second is by Fraser *et al*. (2006), and describes some effects on milk quality from dairy herds where there was inadequate protection of grazing stock from facial eczema damage.

Mycotoxin poisoning in grazing livestock in New Zealand

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ABSTRACT

A number of mycotoxicoses including facial eczema, ryegrass staggers, paspalum staggers and zearalenone infertility affect grazing livestock, causing death and reducing productivity. While production losses associated with clinical disease are well recognised, losses associated with subclinical disease are not recognised and most farmers take few or no precautions against the diseases and consequently accept lower productivity levels as ‘normal’. For most mycotoxicoses there are no antidotes. Long-term research projects, some lasting decades, have led to the development of a number of control measures for particular diseases, especially facial eczema and ryegrass staggers. These measures depend primarily on identifying and avoiding toxic pastures, or reducing their impact. Under the current funding regimes, where funders seek quick returns, it is unlikely that many of these now widely used control methods would have come to fruition.

Keywords: facial eczema; ryegrass staggers; zearalenone; disease; sheep.

MYCOTOXICOSES IN NEW ZEALAND

Amongst the more serious animal health problems faced by New Zealand farmers are the diseases and production losses caused by mycotoxins produced by endophytic fungi living within, or saprophytic fungi living on, our pastures. The diseases with well-established fungus-toxin-animal disease cause and effect relationships include facial eczema (FE), ryegrass staggers, paspalum staggers and zearalenone infertility. Other less well-defined animal health and production problems may also be due to mycotoxins; these include kikuyu poisoning and autumn ill-thrift. These problems are not unique to New Zealand but our reliance on pasture as the predominant or sole feed, in conjunction with intensive grazing practices that inadvertently maximise toxin intakes, mean that these problems are often more severe and frequent in New Zealand than in other countries.

Facial eczema (FE)

Facial eczema is a disease of sheep, cattle and deer, but it can also affect goats, llamas and alpacas. The disease occurs in Australia, South Africa, France, the Azores, Uruguay, Argentina and Brazil, and may also have occurred in the USA and Netherlands. The disease is caused by the ingestion of spores of the saprophytic fungus *Pithomyces chartarum* containing sporidesmin, a potent hepatotoxin which also damages the bladder and mammary gland. *P. chartarum* is found in all temperate climate zones of the world but New Zealand isolates may be unique in that they almost all produce sporidesmin whereas in other countries toxin production ranges from 2-67 percent (Collin *et al.,* 1998a). FE 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 signs of photosensitisation affecting non-pigmented areas of skin exposed to sunlight. 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 before signs of photosensitisation and clinical disease are normally seen and therefore are often not recognised by the farmer as being sub-clinical FE. The clinical signs of FE are typical of a secondary photosensitisation arising from liver damage and occlusion of the bile ducts.

Clinical signs appear only in animals with severe liver damage and occlusion of the major bile ducts. In all but the most severe outbreaks the majority of the animals will show little, or no, outward signs of the disease (i.e., photosensitivity, eczema on exposed non-pigmented skin including face, ears, udder), but analysis of specific blood enzyme levels will reveal that many of the animals have suffered liver damage. Production losses arise from animal deaths, condemnation of jaundiced carcasses, weight loss or reduced weight gain (Smith, 2000), reduced fertility in sheep and lower milk yields in dairy cattle. Reduced ewe fertility in the year of an FE outbreak has been widely reported and is perhaps not unexpected as mating often coincides with periods of high FE risk (Jagusch *et al.,* 1986), but there is also evidence that FE can reduce production of lambs by up to 25 percent over the lifetime of affected ewes (Moore *et al.,* 1983). In dairy cattle the fall in milk volumes following liver damage and the onset of clinical disease is well known but, in addition to these production losses, trial data show that sporidesmin intakes too low to cause detectable liver damage can reduce milk volumes significantly (Towers, unpublished) causing even greater production losses. Production losses in NZ have been variously estimated to range from $10 - $106 million per annum, depending on how severe the disease has been in that year (NZMWBES, 1990; Faull, 1991).

Ryegrass Staggers and Fescue Toxicosis

Ryegrass staggers and fescue toxicosis are the best known of the endophyte-related animal health problems. They are found domestically and internationally, occurring wherever either perennial ryegrass or tall fescue are predominant pasture species (Fletcher, 2004) – although in New Zealand, where modern fescue cultivars are mainly endophyte-free, fescue toxicosis is now rare. 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* 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 herbivorous insect 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. Other grass-endophyte associations cause a number of other well known problems including sleepy grass

syndrome (USA) (Petroski *et al.,* 1992), drunken horse disease (China) (Miles *et al.,* 1996), and less well defined problems associated with livestock consuming grasses native to Australia, New Zealand and South America (Cheeke, 1995; Miles *et al.,* 1995a,b, 1998).

Paspalum Staggers

Paspalum staggers, although once common when many pastures were paspalum-dominant, is an almost forgotten problem of sheep and cattle grazing tropical or subtropical *Paspalum* spp. which have been allowed to flower, and the inflorescence has been infected with *Claviceps paspali* (Lacey, 1991). 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

Fusarium species which are known to produce the oestrogenic compound, zearalenone, are common in pastures throughout New Zealand (di Menna *et al.,* 1987), and relatively high concentrations of zearalenone (0.5-5 mg/kg pasture dry matter) may be found in the autumn months (February-May), coinciding with the sheep mating season (Sprosen *et al.,* 1995). Zearalenone intakes exceeding 1 mg per day adversely affect reproduction in sheep, the effects becoming more severe at higher intakes and/or longer periods of exposure (Smith *et al.,* 1990a, 1992a). Other details are given by Smith & Morris (2006).

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 forty 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 (Sprosen *et al.,* 1995).

OTHER PROBLEMS POSSIBLY CAUSED BY MYCOTOXINS

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

(i) Zearalenone and cattle infertility problems

High concentrations of zearalenone in pasture, and of zearalenol in urine and blood, have been found in herds with poor reproductive performance (Sprosen & Towers, 1995). However, as the samples were collected in January or February, when veterinary examination revealed low pregnancy rates, some months after mating in spring (October, November), the data do not necessarily reflect a cause and effect relationship between high zearalenol levels in the blood and the fertility problems. Indeed, given that pasture zearalenone levels are generally low when cattle are mated, it seems that a direct effect on ovulation rates, similar to that found in sheep, is unlikely unless cattle are exceptionally sensitive to zearalenone.

(ii) Autumn ill-thrift

Many *Fusarium* species found in pasture produce not only zearalenone but also a wide range of trichothecene mycotoxins known to be toxic to animals (Lauren *et al.,* 1992). Autumn ill-thrift occurs during the same season as high zearalenone and *Fusarium* levels are found. The possibility that *Fusarium* produce trichothecenes in sufficient quantity to affect animal health and performance has been investigated using ELISA assays for deoxy-nivalenol (DON) and nivalenol (NIV), two trichothecenes commonly produced by New Zealand isolates. Litherland *et al.* (2004) found that on every occasion (10 out of 70 pasture analyses) when NIV and DON levels were above 0.8 mg/kg dry matter, both calves and lambs grew more slowly than expected from the quality and quantity of the pasture available. However preliminary studies dosing lambs with extracts of *Fusarium* cultures containing DON or NIV suggest that pasture concentrations of these toxins would need to be much higher (i.e. 10-50 mg/kg dry matter) before they would affect the health or growth of ruminant animals (Odriozola, 1996). A cause and effect relationship has not been established and an alternative explanation might be that the high DON and NIV levels are merely markers for the presence of an unidentified, but more potent, toxin.

(iii) Kikuyu grass poisoning

Ill-thrift, illness and death amongst sheep and cattle grazing kikuyu pastures have been reported occasionally for many years. The etiology

of the disease has never been resolved but the striking similarities between kikuyu poisoning of cattle and the clinical and pathology findings after dosing cultures of toxin-producing strains of *Myrothecium verrucaria* and *M. roridum* to cattle and sheep have been noted (di Menna & Mortimer, 1971; Martinovich *et al.,* 1972). However, the toxins have never been isolated from pasture in any quantity and the intermittent occurrence of the disease has hindered research so that a cause and effect relationship has not been established.

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.

(1) Reducing toxin intakes

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.

(i) 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 FE can be readily determined by monitoring the number of the distinctive and easily identified spores in pasture (Chapman & di Menna, 1981). This technique was once widely used by farmers, or more often farmers' wives, but is now a service offered by veterinary practices. But, for most other 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 for detecting sporidesmin, zearalenone, DON, NIV, lolitrem, paxilline, ergopeptine alkaloids and peramine have been developed and adapted to the analysis of pasture, serum and urine samples. The relative speed and simplicity of ELISA make them particularly suitable for screening large numbers of pasture or urine samples (Barker *et al.,* 1993, Briggs *et al.,* 1994; Collin *et al.,* 1998b; Garthwaite, 1997; Gallagher *et al.,* 1985; Hill & Agee, 1994; Lauren *et al.*, 1992; Spiering *et al.,* 2002).

Toxic pastures may also be recognised (suspected) on the basis of weather patterns, in the case of FE 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 (grass minimum >12-13 °C and 4-6 mm rain) and these "danger periods" signal the need to initiate control measures. *Claviceps* infections of paspalum seed heads are readily recognised and either stock can be moved to other pastures or, as the toxins are confined to the seed head, the pastures can be made safe by mowing and removing the seed head material. Endophyte toxins are at highest concentrations when ryegrass is flowering and seeding and following hot dry weather later in summer. To date no clear cut associations between weather patterns and *Fusarium* growth and toxin production have been established. This may be in part due to several different *Fusarium* species co-existing in the pasture, all with different optimal growth conditions, with the pre-dominant species gradually changing, the further south the location.

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 which grow on the dead litter, while the endophyte toxins are in highest concentration in the basal leaf sheath and under low grazing pressure livestock can avoid ingesting this material (Smith *et al*., 1963).

When toxin levels of sporidesmin or zearalenone in pasture are high, feeding conserved fodders or crops (maize, brassicas, chicory, red or white clover, hybrid millets, etc.) can reduce toxin intakes if 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 (Meat New Zealand, 1999a).

(ii) Reducing toxin production

Benzimidazole fungicides have been used successfully to reduce *P. chartarum* spore numbers, to control FE (Oldman & di Menna, 1983). These fungicides inhibit germination of spores present at the time of spraying, and reduce the spore production by subsequent generations of mycelia by 55-65 percent (Parle & di Menna, 1972). If applied while spore numbers are low, fungicides prevent spore numbers rising to toxic levels but management difficulties (deciding when to spray and to graze sprayed pasture) and apparent breakdowns in control led to a loss of confidence and greatly reduced use of the technique. Benzimidazole fungicides, even at rates 10-fold those used for controlling *Pithomyces,* have not

been effective in controlling *Fusarium* in pasture and have no effect on zearalenone production but other more effective fungicides may be found. Applying fungicides to pasture has no effect on the endophytic *Neotyphodium* 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, as endophyte-free perennial ryegrass and tall fescue are more susceptible to environment stress and insect attack, this option has not been particularly successful.

(iii) Modifying the fungal population

A more promising approach has been to generate ryegrass and tall fescue cultivars infected with *Neotyphodium* selected (Tapper & Latch, 1999) or genetically engineered (Panaccione *et al.,* 2005) to produce low or zero levels of lolitrem and ergopeptine alkaloids while continuing to produce toxins (peramine, lolines) protecting against insects (Popay *et al*., 1999). Animals grazing pastures with modified endophytes perform better than those grazing pastures infected with wild-type endophytes (Fletcher, 1999, 2004). The selected endophytes confer many of the advantages of the wild-type endophyte although the novel endophyte:grass associations appear to be less resistant to some insects.

While most New Zealand *P. chartarum* produce sporidesmin, a few isolates produce neither sporidesmin nor any other readily detected toxins (Collin *et al.,* 1996). The observation raised the possibility of biocontrol, based on introducing large numbers of atoxigenic spores into the pasture prior to the FE season so that they became the dominant population. In many other countries non-toxic isolates predominate. Feasibility studies gave very promising results, with an 80% reduction in toxin levels on treated pasture plots (Fitzgerald *et al.,* 1998) and 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, unpublished). However, difficulties in the first attempts to develop a cost-effective inoculum and the apparently low persistence of the atoxigenic strains used (80-90% of isolates recovered from treated pasture were atoxigenic soon after treatment, but only 53% at 4 months and 4% at 15 months) (Fitzgerald *et al.,* 1998) led to the premature abandonment of this research.

(2) Protecting animals against ingested toxins

Although the protection of animals against ingested toxins has been a major focus for research in FE, ryegrass staggers, fescue toxicosis, and zearalenone infertility, the only practical prophylactic method developed to date is for FE, where zinc salts may be used for protection (Smith *et al*., 1977; Towers & Smith, 1978). Dosing with 15-30 mg Zn per kg liveweight per day as either zinc oxide or zinc sulphate before the animals ingest sporidesmin can reduce liver damage and production losses by 60-90%. There is strong evidence that zinc dosing is effective because zinc forms a stable mercaptide with reduced sporidesmin, removing it from the autoxidation cycle (sporidesmin to reduced sporidesmin and back again) that leads to the cascade generation of reactive oxygen radicals causing cell damage and ultimately cell death (Munday, 1984).

Drenching with zinc oxide, or adding zinc sulphate to the water supply, are now the most widely used FE control methods. A major advance has been the development and marketing under the brand name, the Time Capsule®, of slow-release intraruminal boluses which provide protection to sheep for 6 weeks (Munday *et al.,* 1997) or cattle for 4 weeks (Munday *et al.,* 2001). This innovative delivery method required the development of completely new manufacturing technology by HortResearch engineers to melt and extrude the mix of zinc oxide and binders that form the core of the bolus and to dip the boluses into the wax mixtures that provide the coating.

Attempts to block the action of endophyte toxins with 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 non-immunised animals (Fairclough *et al.,* 1984; Smith *et al.,* 1991b). 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. The major outcome from this work was the production of zearalenone-binding antibodies used in the very sensitive and robust immunoassays for zearalenone. The use of Androvax™ (androstenedione immunogens), to increase ovulation rates to counter the decrease caused by zearalenone, had limited success because of adverse interactions in which Androvax™ reduced tupping and conception rates during the first cycle of mating in zearalenone-dosed ewes. As a result,

although more lambs were born, they had a later mean birth date (Meat New Zealand, 1999b).

(3) Breeding for resistance

The wide range of individual responses to a toxin challenge suggests that some animals are inherently more resistant to the toxin, and raises the possibility of breeding for resistance to the disease. However, in developing a selection 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 (h^2 = 0.42-0.45) and cattle (h^2 = 0.40) (Campbell *et al.,* 1981, Morris *et al.,* 1995a, 1998; Cullen *et al*., 2006) and to ryegrass staggers in sheep (h^2 = 0.13, using a 0-or-1 score: Morris *et al*., (1995b); h^2 = 0.43, using multiple scores: Morris, unpublished). A heritable difference in the response to zearalenone dosing in sheep (h^2 = 0.32) (Morris *et al.,* 2005a) and divergence in response to ergot alkaloid toxins in mice have also been reported (Hohenboken & Blodgett, 1997), with a positively-correlated change in resistance to sporidesmin (Hohenboken *et al.,* 2000).

RamGuard, a commercial service to performance-test potential flock sires for FE resistance, has now been in operation for 20 years. Potential flock sires are dosed with sporidesmin and serum gamma-glutamyltransferase (GGT) concentrations are measured to rank the severity of any subsequent liver damage (Towers & Stratton, 1978). Breeders who have used the service since its inception have increased the sporidesmin resistance of their flocks 6-fold or more. The dairy industry, despite the finding in 1989 (Morris *et al.,* 1990, 1991b) of a high heritability for FE resistance in dairy cattle, and the opportunities for rapid dissemination of the genes through artificial insemination, has not yet begun selection for FE resistance, wasting 15 years of potential genetic gain.

Difficulties in isolating sufficient lolitrem B or ergovaline have so far prevented the development of similar toxin-challenge performance tests for resistance to the endophyte toxins but considerable progress has been made in breeding for ryegrass staggers resistance using naturally toxic pasture (Morris *et al.,* 1999; Amyes *et al*., 2002). Interestingly, despite sporidesmin and lolitrem B having very different chemical structures and modes of action, selection for FE resistance has increased resistance to ryegrass staggers in experimental flocks (Morris *et al*., 1995b), and selection for ryegrass staggers resistance has increased resistance to FE. These observations suggest that resistance to these toxins may have multiple origins, one of which is common to both FE and ryegrass staggers resistance.

NEW OPPORTUNITIES

As new analytical and research techniques arise, so do opportunities for further research. These do not necessarily require new hypotheses; rather, in several instances, the most promising opportunities lie in revisiting previously partially researched ideas:

(i) Biocontrol of Pithomyces *and perhaps* Fusarium

Biocontrol of aflatoxin contamination of corn and cotton (Cotty, 1994, Dorner *et al.,* 1999) by the introduction of atoxigenic strains of *Aspergillus* has been successful in the USA. The New Zealand work suggests that biocontrol could successfully reduce FE risk if problems associated with the production of inoculum and survival of the atoxigenic strains could be overcome. There are opportunities here for genetic modification to eliminate toxin production in aggressively growing strains and to extend the work to other toxin producing fungi.

(ii) Mycotoxins and ill-thrift

The strong relationship between high DON and NIV concentrations and the occurrence of autumn ill-thrift (Litherland *et al*., 2004) deserves a concerted investigation involving mycologists, chemists, toxicologists and animal scientists.

(iii) Mycotoxin-binding feed supplements

A number of mycotoxin-binding feed supplements, primarily aluminosilicate minerals (bentonite, zeolite) or yeast cell wall products have been used to reduce the toxic effects of aflatoxin and zearalenone on animals and to reduce the passage of toxins from feeds to milk (Ramos *et al.,* 1996, Huwig *et al.,* 2001). This approach has not been fully explored in New Zealand as the practicality of delivering such binders to grazing sheep and cattle is challenging. However, changes in dairy cow management with increasing use of maize silage and other supplements provide a potential administration route. It is essential that the effectiveness of these binders is tested in ruminants, as *in vitro* binding tests do not replicate conditions in the rumen/abomasal/small intestine pathway of ruminants.

(iv) Genetic markers for animal resistance.

Breeder resistance to submitting potential sires to a toxin challenge to rank them for FE resistance is cited as one reason that selection for FE resistance

is not widespread in the sheep industry and not used at all in the dairy industry. Identifying the genes controlling resistance to FE and developing gene marker assisted selection procedures represents a major opportunity. The same or similar gene markers may operate in sheep, cattle and deer, so that identifying genes in one species could lead to rapid identification of key genes in other species. Phua *et al.* (1999), in a programme now funded by Ovita, identified catalase as a candidate gene having a statistically significant association with FE resistance, but Hohenboken *et al*. (2004) concluded recently that catalase, alternate forms of superoxide dismutase-1 (cytosolic or mitochondrial), glutathione peroxidase-1 and glutathione reductase, played only minor roles in determining genetic differences in FE resistance in sheep. Further DNA studies in sheep are continuing (e.g., Duncan *et al*., 2002, 2005). Where divergent breeding lines have been established, this approach could be applied to other mycotoxicoses such as ryegrass staggers. The opportunities which these lines provide should not be squandered. A major cost in carrying out DNA-marker searches for disease resistance is the cost of scoring/ranking large numbers of animals for resistance. The procedure described by Cullen *et al.* (2006), using data from dairy sire proving schemes following outbreaks of the FE disease, could have application in the sheep industry and to diseases other than FE.

Review of zearalenone studies with sheep in New Zealand

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ABSTRACT

Zearalenone is a naturally occurring mycotoxin from the *Fusarium* fungus which grows on pastures in New Zealand in autumn, and it has been found on farms in some years from Northland to Southland. The toxin may interfere with oestrogen-related functions in sheep during reproduction, reducing ovulation rates and fertility and thus lambing percentages, because its chemical structure is similar to that of reproductive steroids. Forty four per cent of over 6000 New Zealand pasture samples, collected in autumn and tested for zearalenone, were found to have high enough levels for ewe fertility to be either depressed (9% of samples), or 'at risk' (35%). Control of zearalenone toxin production or of *Fusarium* growth on pasture on a large scale is currently not feasible. Attempts to mitigate its effects by immunisation have failed or even exacerbated the problem. Provision of alternative zearalenone-free feed crops is costly and generally uneconomic. Selection of sheep for genetic resistance would seem to be the most beneficial approach. Resistance to zearalenone is inherited in sheep (heritability estimate = 0.32 ± 0.10), and a test could be set up in ram-breeding flocks to select for resistance.

Keywords: zearalenone; sheep; reproduction; genetics; resistance.

INTRODUCTION

Zearalenone is a naturally occurring mycotoxin from the *Fusarium* fungus which grows on moist, dead plant material in many New Zealand pastures in autumn. In survey work, Garthwaite *et al.* (1994) found zearalenone at toxic levels in autumn on at least some pastures throughout New Zealand from Northland to Southland. Nine per cent of over 6000 samples tested had zearalenone at high enough levels for ewe fertility to be depressed, and another 35% were from paddocks where flocks would be 'at risk'. Although it is a mycotoxin, the chemical structure of zearalenone is unrelated to that of the facial eczema-causing toxin, sporidesmin. Instead, its structure and its metabolic breakdown products are similar to that of the reproductive steroid hormones. This enables it to bind to the oestrogen receptors of mammals (Coulombe, 1993), interfering with the signal transduction and control functions of endogenous oestrogens. In adult sheep, the primary effect of zearalenone is to reduce ovulation rate and pregnancy percentage, resulting in decreased lamb production (Smith *et al.*, 1986, 1987a).