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A Review of Facial Eczema

(Pithomycotoxicosis)

Report of the Dairy Australia Facial Eczema Working Group

The fungus → FE spores → Sporidesmin in the gut → Liver damage → Photo-sensitisation

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- Andrew Debenham, Debenham Australia
- Neil Cullen, AgResearch, New Zealand
- Chris Mulvaney, AgriNetworks Ltd., New Zealand
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- Hugh Archibald, Phibro Animal Health
- Peter Norwood, Daly's Feed and Fertiliser
- Hans Van Wees, dairy farmer, Maffra

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Contents

Executive Summary

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Facial eczema (FE) can have significant impacts on dairy cattle productivity, health and welfare. Although the name suggests it is a skin disease, in most outbreaks most animals in a herd show little or no visible skin lesions, but have suffered liver damage. It is these animals that give the major economic impact.

Production losses in dairy cattle arise from animal deaths, weight losses or reduced weight gain, reduced milk yield and reproductive performance.

While outbreaks of FE has been reported across Victoria in the past, it has been seen mainly in east Gippsland. However, the period January to April 2011 saw outbreaks of FE across Victoria, with unconfirmed cases also reported in areas of coastal NSW, Western Victoria, Northern Victoria, South Australia and Tasmania.

The prevalence of FE in New Zealand, Gippsland and other southern dairying regions of Australia may increase in the years to come as the effects of global warming may provide suitable conditions for more widespread outbreaks of FE over longer periods.

FE is caused by ingestion of spores of the fungus Pithomyces chartarum, which lives mainly on ryegrasses. Under favourable conditions the fungus can rapidly multiply in pastures. The spores of the fungus release a potent mycotoxin known as sporidesmin in the gastrointestinal tract, which causes damage to the liver (particularly the bile ducts), bladder and mammary gland.

In most cases of clinical FE, the presenting sign is photosensitisation arising from liver damage and bile duct blockage, which tends to occur around two weeks after exposure to sporidesmin. An initial transient diarrhoea and sudden milk production drop is often not recognised by the farmer as being associated with FE. Skin lesions may progress further and eventually lead to large sheets of skin peeling off, especially in Holstein-Friesian cows. Haemoglobinuria and jaundice occur occasionally and often as a prelude to a severe outbreak of FE. However, they are not seen in many herd outbreaks. Cows that die in the short term generally do so from complications of photosensitivity rather than liver failure. Some cows recover, only to go down and die around the time of calving.

Certain weather conditions are favourable to germination or sporulation of P. chartarum. Pastures tend to become toxic in late summer and autumn when periods of rain or high humidity occur in combination with high night-time minimum temperatures. However, to accurately predict FE danger periods and take action to prevent liver damage before it occurs, use of a pasture spore monitoring program is essential.

There are several strategies that can be used to help reduce the risk of cows ingesting large numbers of toxic spores, which tend to concentrate at the base of the pasture sward. These include careful grazing management, use of alternative pasture species, crops and supplements such as hay, silage and grain/concentrates.

Zinc is protective for FE, as it forms a complex with sporidesmin, inhibiting its ability to cause cell damage. FE prevention using zinc can be effective if it maintains the cow's blood serum zinc level at 20-35 µmol/L.

This is difficult to achieve using zinc sulphate via drinking water as daily water intakes can vary for many reasons. Feeding zinc oxide in grain/concentrates (in mash or pelleted form) in the bail at milking can be very effective for FE prevention on Australian dairy farms. However, the amount of zinc oxide included in each tonne of grain/concentrate for 'prevention dosing' must be carefully calculated to achieve the required dose of 20 mg elemental zinc/kg liveweight/day and minimise the risk of zinc toxicity. The farmer, stockfeed company, vet and any nutrition advisers involved need to share responsibility for implementation of each zinc supplementation program to ensure it is effective and safe, using a HAACP approach.

A controlled release, intra-ruminal zinc bolus is an effective and very safe option for zinc administration, and is particularly useful in young stock. Two intra-ruminal zinc bolus products are marketed in New Zealand. However, at the time of publication, controlled release, intra-ruminal zinc bolus products were not approved for use in Australia.

Breeding for FE tolerance holds promise as being a very useful long-term approach to managing FE. FE tolerant sires are now commercially available to dairy farmers in New Zealand. The next step for the dairy industry is to use the DNA of these FE tolerant dairy sires to develop a reliable DNA marker test. Joint development of genomic tools by New Zealand and Australian dairy industry partners should be considered.

Section One

Introduction

Facial eczema (FE) is a disease of sheep, cattle and deer, but can affect other grazing animals. FE can have significant impacts on animal productivity, health and welfare and is a common seasonal problem in dairy cattle in New Zealand.

The condition is caused by ingestion of spores of the fungus Pithomyces chartarum, which lives mainly on ryegrasses. Under favourable conditions the fungus can rapidly multiply in pastures. The spores of the fungus release a potent mycotoxin known as sporidesmin in the gastrointestinal tract, which causes damage to the liver, bladder and mammary gland.

Facial eczema is named for the visible signs of photosensitisation that affect non-pigmented areas of skin exposed to sunlight and result in severe skin irritation, dermatitis and flystrike (see Figure 1). However, in most outbreaks of the disease the majority of animals show little or no visible skin lesions, but have suffered liver damage. FE is therefore not well named.

Production losses in dairy cattle arise from animal deaths, weight losses or reduced weight gain, reduced milk yield and reproductive performance (Smith, 2000). Using conservative assumptions, FE has been estimated to cost the New Zealand dairy industry between $10 and $100 million per year depending on the severity of the disease year to year (Faull, 1991). There are no estimates published on the cost of FE to the Australian dairy industry.

While FE outbreaks have been reported across Victoria in years past, they have been seen mainly in east Gippsland. However, as Figure 2 shows, there were confirmed, probable and suspect FE outbreaks in dairy cattle across most areas of Gippsland between January and April 2011. Suspected cases were also reported in areas of coastal NSW, Western Victoria, Northern Victoria, South Australia and Tasmania.

This caused considerable concern among dairy farmers, veterinarians, nutrition advisers, extension providers and farm consultants in Gippsland and also in other southern dairying regions. Uncertainty around climate variability in future seasons and its implications for the prevalence of FE has compounded these concerns, as has confusion about the factors that increase the risk of an outbreak and how to safely and effectively control and prevent FE.

In response to the situation, Dairy Australia formed a Facial Eczema Working Group with two specific objectives:

- Review FE in dairy cattle, covering the disease, risk factors, and control and prevention strategies in the Australian context, and produce a report to serve as the basis for extension information.
- Generate a number of recommendations to industry in relation to Australian FE research and development, field surveillance programs, and control and prevention strategies.

Facial Eczema Working Group members:

- Steve Little (Capacity+ Ag Consulting)
- Jakob Malmo (Maffra Veterinary Centre)
- Andrew Debenham (Debenham Australia)
- Neil Cullen (AgResearch, NZ)
- Chris Mulvaney (AgriNetworks Ltd, NZ)
- Ian Sawyer (Feedworks)
- Hugh Archibald (Phibro Animal Health)
- Peter Norwood (Daly's Feed and Fertiliser)
- Hans Van Wees, dairy farmer, Maffra

This report summarises the working group's findings.

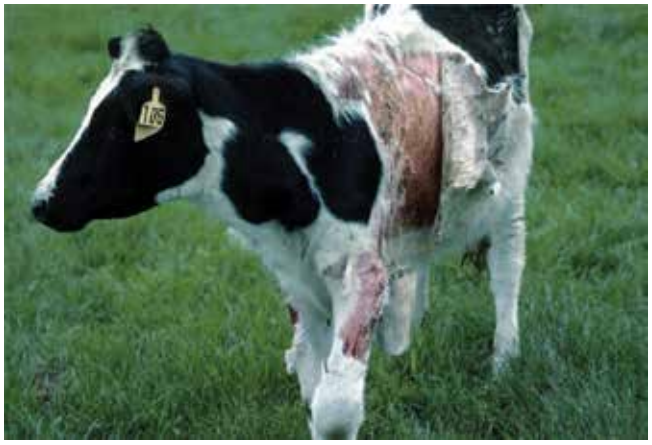

Figure 1. Typical FE skin lesions

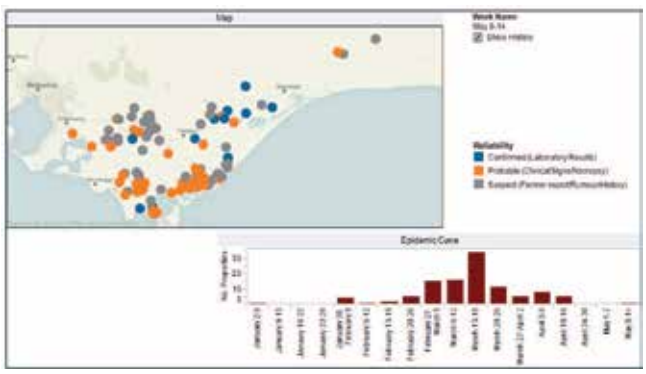

Figure 2. Time series – Gippsland FE outbreaks, 2011. Source: DPIV (2011)

Section Two

A brief history of FE

Facial eczema (FE) has been known in New Zealand since the 1890s. A severe and widespread outbreak in the autumn of 1938 and calls for answers from farmers led to action that established a research station at Ruakura under the management of the Department of Agriculture and its new Animal Research Division.

A review paper by di Menna et al.(2009) documents the research that eventually identified the causative agent in 1958 as a toxin (sporidesmin) produced from the spores of a fungus which was (re-)named Pithomyces chartarum.

Despite a subsequent proposal to name the disease pithomycotoxicosis, the name 'FE' had already become embedded in the farming vocabulary and the scientific literature.

FE in Victoria was first reported in sheep in east Gippsland in 1956. An outbreak in 1959 resulted in 10,000 sheep deaths, with 40% of the properties in the area experiencing losses of five to 1,200 sheep. This led to the Rosedale Facial Eczema Research Station being established in 1960. A 1961/62 survey found that the fungus was widespread in Victoria.

Outbreaks in 1960 and 1961-63 saw reports of 2,000 sheep and 100 cattle deaths, and 1,000 sheep and 100 cattle deaths respectively (see Figure 3).

The first year dairy cattle were reported as involved in an FE outbreak was 1974. This was also the first time it was seen in an irrigation area (Macalister Irrigation District). Some FE outbreaks with up to 3% deaths were also reported in northern Victoria (Shepparton, Benalla, Wangaratta).

FE outbreaks also occurred in Gippsland in 1976, 1981, 1999 and 2011. In the 2011 outbreak, DPI Victoria recorded disease on 116 properties (all animal species), of which 86 were dairy farms in the following shires: Wellington (Macalister Irrigation District) 31 farms, Baw Baw (Willowgrove) 25 farms, South Gippsland 23 farms; Cardinia 4 farms. DPI Victoria has robust animal data for 50 of these 86 farms: of a total population of 16,281 animals (all ages), there were 3,180 cases (19.53%) and 169 deaths (1.04%).

It is likely that in all these FE outbreaks, the cases of clinical disease and deaths have been grossly under-reported.

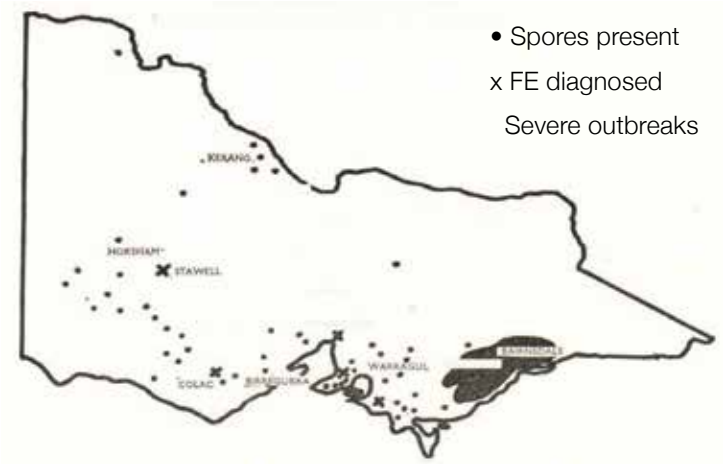

Figure 3. Victorian Regional Incidence 1960-63
Source: DPIV (2011)

Section Three

Could we see more FE in future?

The prevalence of facial eczema (FE) may be affected by the effects of global warming.

In New Zealand, a study reported by di Menna et al (2009) has shown that if higher temperatures do not reduce rainfall, the areas where FE outbreaks occur may expand to include higher altitudes and more southerly areas, and the FE risk period may be extended further into autumn. Figure 4 shows the expansion in FE outbreak areas likely in New Zealand under 3°C climate warming.

In Gippsland and other southern dairying regions of Australia, global warming is expected to bring an earlier start to summer and higher nighttime minimum temperatures over summer-autumn. When combined with wet weather, this may provide suitable conditions for germination and sporulation of Pithomyces chartarum over longer periods and more widespread outbreaks of FE.

In addition to the effects of global warming, a question to consider is whether the genetic susceptibility of cows to FE is increasing. A recent New Zealand study of genetic trends for Holstein-Friesian and Jersey size with reliable Breeding Values (BVs) suggested that the susceptibility to FE has been increasing gradually over the past 25 years. Further work is required to understand this unfavourable trend (pers. comm. N Cullen, AgResearch, 2013).

Figure 4. Areas in New Zealand currently liable to FE outbreaks (left) and areas predicted to be liable to FE under 3°C climate warming (right).
Source: di Menna et al (2009)

Section Four

The fungus and FE spores

P. chartarum was originally believed to have a tropical habitat. However, since it was associated with facial eczema (FE), P. chartarum has been found across the world, including temperate climate zones. P. chartarum is usually saprophytic and is found on a wide range of decaying plant matter and in soil and air. As shown in Figure 5, it is characterised by hand-grenade shaped spores.

FE tends to be mainly associated with perennial ryegrass because of its ability to produce large quantities of dead litter, with the ongoing senescence and death of leaf and leaf-sheath material. Clover, kikuyu, paspalum and tall fescue pastures are safer than ryegrass-dominant pastures.

Certain weather conditions are favourable to germination or sporulation. While ryegrass pastures can provide adequate substrate for fungal germination all year round (a low inoculum of P. chartarum spores overwinter in FE prone areas), the greatest development of the fungus is from mid-summer with the death of the pasture. Late haymaking, topping or mowing of pastures may increase the substrate for fungal growth and sporulation, increasing pasture toxicity potential. The extent of defoliation during grazing largely determines intake of dead litter and spores – very close grazing will increase the uptake of spores.

Toxigenic strains of P. chartarum form the toxin sporidesmin in their mycelium and this toxin is concentrated in the spores during sporulation. As Figure 6 shows, while the spores may be distributed throughout the whole sward, the greatest concentrations, and hence the most toxic part of the pasture, tend to be at the base of the sward. Spores are highly concentrated in litter at the base of the sward, with numbers decreasing markedly with the increase in height above the base of the sward.

It has been suggested that New Zealand isolates of P. chartarum may be unique in that they almost all produce sporidesmin whereas in other countries toxin production ranges from 2% to 67%. Collin et al (1998) examined the sporidesmin producing capabilities of 391 P. chartarum isolates and found that 86% of those from New Zealand, 67% from Australia, 28% from Uruguay and 2% from Brazil produced sporidesmin. Variation in the ability to produce sporidesmin was found not only between countries, but also between samples taken from the same country.

Exactly how long sporidesmin levels in spores persist after sporulation is uncertain.

Key points

- FE tends to be mainly associated with perennial ryegrass because of its ability to produce large quantities of dead litter, which supports germination of P. chartarum
- While germination can occur all year round, sporulation requires special weather conditions
- The toxin sporidesmin is concentrated in the spores during sporulation
- The greatest concentrations of spores tend to be at the base of the pasture sward

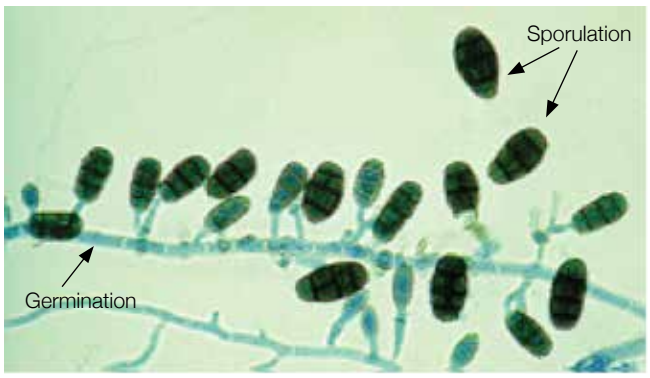

Figure 5. P. chartarum mycelia and hand-grenade shaped spores
Source: C Mulvaney, AgriNetworks, NZ (2011)

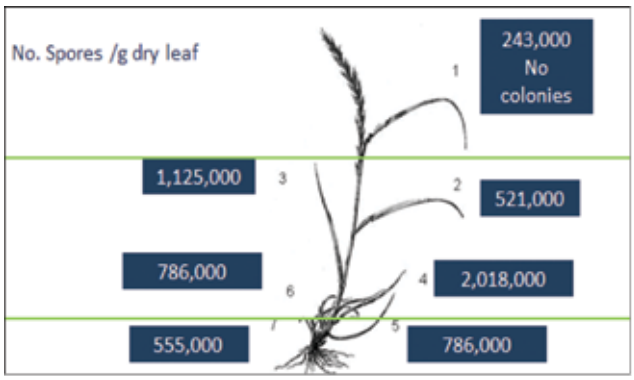

Figure 6. P. chartarum spore distribution in pasture sward.
Source: Brook (1963)

Section Five

The disease

Sporidesmin is released from ingested spores in the upper digestive tract, absorbed into the portal bloodstream and taken to the liver where it generates oxygen free radicals which damage cell membranes. Concentration of sporidesmin in the bile ducts lead to severe necrosis of their mucosal surfaces, resulting in rapid reduction of biliary secretion, bile duct thickening and eventual blockage (see Figures 7 and 8). Cholangiohepatitis, ductular hyperplasia and fibrosis are also seen histologically.

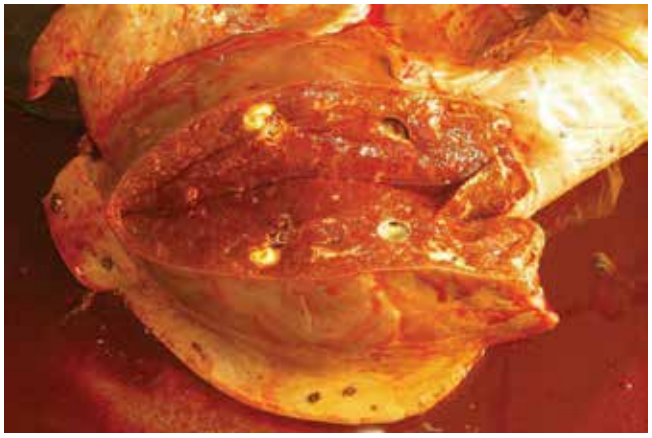

Figure 7. Cut surface of liver showing thickened bile ducts. Source: J Malmo, Maffra Veterinary Centre (2011)

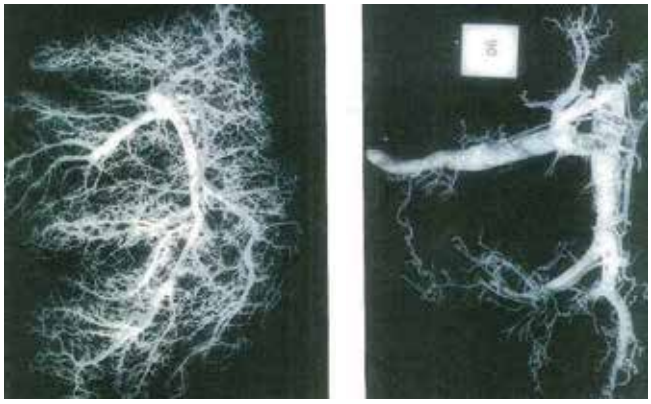

Figure 8. Biliary duct system of normal liver (left) and damaged liver (right). Source: C Mulvaney, AgriNetworks, NZ (2011)

Figure 9. Fibrosed left liver lobe.
Source: J Malmo, Maffra Veterinary Centre (2011)

As shown in Figure 9, the left lobe of the liver eventually becomes fibrosed and shrunken, and the liver takes on the shape of a boxing glove.

The resulting pericholangitis leads to obstructive jaundice. Phylloerythrin, a normal breakdown product of chlorophyll in the rumen, is normally absorbed into the portal bloodstream and excreted by the liver in the bile. With the bile ducts blocked, it accumulates and spills over into the bloodstream.

The reaction between circulating phylloerythrin and sunlight results in tissue damage (photosensitisation), and this is most severe in lightly coloured and non-pigmented skin such as on the face, ears, lips, vulva and udders.

GGT as a diagnostic aid

Blood serum concentrations of the enzyme gamma glutamyl transferase (GGT) give the best indication of the severity of liver damage, or more specifically, bile duct damage. Although GGT is synthesised by many tissues, blood serum GGT originates mainly from the liver. Blood serum GGT activity principally increases in cholestatic disease, although hepatocellular disease in which cholestasis is a secondary feature (e.g. hepatic lipidosis) also causes increased GGT activity. While other things such as liver fluke can damage liver tissue, facial eczema (FE) causes much higher GGT levels.

Serum GGT concentrations may become elevated 2-3 weeks after exposure to sporidesmin. The increase in serum GGT activity is proportional to the degree of hepatobiliary damage. GGT levels drop gradually over a period of several weeks after exposure to the toxin ceases, but often remain elevated for several months. It can be difficult to assess the severity of liver damage based on GGT activity as the period of exposure to sporidesmin is not always known.

As a general rule, in cattle within one month of exposure:

- Blood serum GGT concentrations between 30 and 70 IU/L are not clinically significant.
- Blood serum GGT between 70 and 300 IU/L indicates mild liver damage;
- Serum GGT between 300 to 700 IU/L indicates moderate damage; and
- Serum GGT higher than 700 IU/L indicates severe liver damage.

(Source: Parkinson, Vermunt and Malmo, 2010)

Progression of clinical signs

The first signs of ingestion of the sporidesmin toxin may be an initial transient diarrhoea and sudden milk production drop, which farmers often do not recognise as being associated with FE.

Some research work on dairy cattle in the 1970s and 1980s at Ruakura, New Zealand, to estimate the effects on milk production showed that a single exposure to sporidesmin caused an immediate drop in milk yield with

recovery to pre-dosing levels in about 5 days. If this dose was followed by another in close proximity, the milk yield losses were greater and persisted much longer. The cows treated with zinc at the time of this second dosing also experienced a loss in milk yield. There was a large degree of variation in the milk yield response of cows to this second dose, indicating some degree of genetic variation. High GGT levels were not detected until about 10 days after the second dose. The decrease in milk yield cannot be due to liver damage as glutamate dehydrogenase (GDH – an early indicator of liver damage) does not elevate for several days after exposure and elevated GGTs are observed only about 10 days after exposure. It appears that sporidesmin has some direct effect on the mammary gland to account for the immediate milk yield losses.

This research also demonstrated the effect of 'potentiation', where subsequent doses of sporidesmin cause more severe damage and is similar to the situation in the industry where spore counts are elevated for long periods, and seemingly non-toxic pastures can induce FE (cumulative effect).

In most cases of clinical FE the presenting sign is photosensitisation, arising from liver damage and bile duct blockage, which tends to occur around two weeks after exposure to sporidesmin. Exposed hairless and non-pigmented skin such as on the face, ears, lips, vulva, udder and teats, and inside the hind legs becomes reddened, raised and oedematous (Figure 10). The nictating membrane (third eyelid) commonly appears sunburnt (Figure 11). Cows may be restless, seek shade and lick or rub affected areas and if photosensitisation is acute, collapse showing extreme pain (Figure 12).

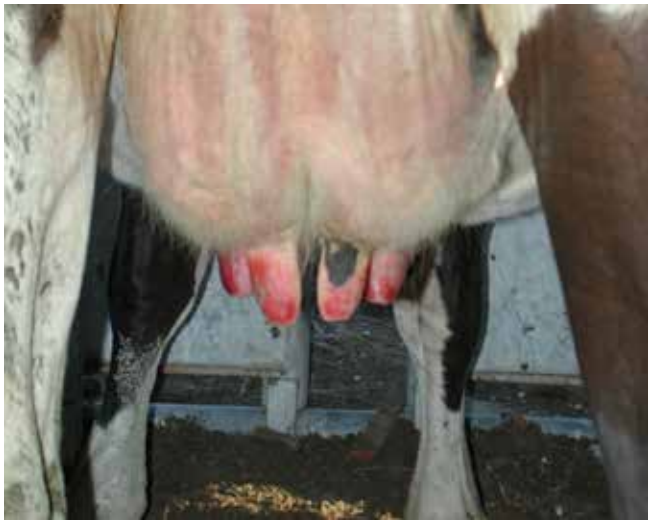

Figure 10. Acute photosensitisation of teats, which is extremely painful

Skin lesions may progress further and eventually lead to large sheets of skin peeling off, especially in Holstein-Friesian cows (Figure 13).

In addition to dermatitis, some cows may also suffer photodynamic coronitis. This eventually grows out with the horn of the hoof, sometimes resulting in painful horizontal fissures of the hoof wall.

Figure 11. Burning of the nictating membrane (third eyelid)

Figure 12. Cow with acute photosensitisation collapsed

Figure 13. Cow with large sheets of skin peeling off

In severe cases, cystitis (inflammation of the bladder) may also occur, with affected cows exhibiting prolonged and frequent urination. Haemoglobinuria and jaundice may also occur, associated with an acute haemolytic crisis, often as a prelude to a severe outbreak of FE (Figure 14). However, they are not seen in many herd outbreaks.

Figure 14. Haemoglobinuria
Source: Figures 10-14: J Malmo, Maffra Veterinary Centre (2011)

As already stated, most animals affected by FE do not show obvious signs, but will have some degree of liver damage and their overall well-being and milk production will be affected. For every cow in a herd with skin lesions, up to 10 or more cows may be affected sub-clinically. A New Zealand study of 565,000 dairy cows (Faull, 1991) showed that only 6% of all animals had skin lesions, while 38% had liver damage.

In affected animals a drop in milk production may be temporary, but in severe cases affected cows may dry off completely. Cows that die in the short term generally do so from complications of photosensitivity rather than liver failure. A small number of affected animals may apparently recover, only to go down and die around the time of calving, the damaged liver being unable to handle the additional stress around the time of calving.

Post mortem findings

Since the biliary tract rather than the hepatocytes is the principal target for sporidesmin, it is common for affected animals to show clinical signs of cholestasis (i.e. photosensitivity) without other signs of liver failure.

At post-mortem examination there may be macroscopic evidence of oedema and fibrosis of the biliary tract. Cholangiohepatitis, duct hyperplasia and fibrosis may be seen histologically.

Chronic cholangitis eventually leads to left lobe atrophy in the livers of ruminants. Therefore, a bovine liver in which the right lobe is enlarged while the left one is pale, thin, tough and small, should always signal the possibility of chronic sporidesmin toxicity.

Differential diagnosis for photosensitisation

Primary cases of photosensitisation are caused by the ingestion of a photodynamic agent from a range of plants including St John's wort – in these cases there is no liver damage. Cattle induced to calve prematurely with a long-acting corticosteroid may develop a photosensitive dermatitis on the teats. Photosensitisation, with an absence of liver damage, is occasionally seen in cattle grazing lush green oats or millet.

Secondary or hepatitis photosensitisation is associated with a buildup of phylloerythrin in the peripheral circulation – this occurs as a result of an impaired hepatobiliary system. The most common cause of hepatogenous liver damage in Australia and New Zealand is FE, but it can also be a result of liver damage arising from a range of toxins including blue green algae, and a range of specific plant toxins (including Lantana and pyrrolizidine alkaloid-induced hepatic disease). Acute bovine liver disease (ABLD) must also be considered.

Prognosis and treatment

Mortality may not be very high, especially when FE-affected animals are given shelter and adequate care. Cows have a remarkable ability to regenerate liver compared to sheep.

It is generally recognized, however, that animals severely affected by FE may have problems in the following spring. These may be metabolic problems related to calving or further photosensitisation. In these cases liver regeneration, although sufficient to handle normal daily requirements, may be insufficient to cope with the additional demands of spring (e.g. added burden of the fast-growing fetus and greater absorption of phylloerythrin).

There appears to be a significant negative association between GGT concentrations after an FE challenge and cow survival. It has been found that survival of animals to the end of the second lactation in exposed herds is about 10% lower in heifers with blood serum GGT >200 IU/L than in those with lower GGT values. In general, young stock tend to recover better than older animals.

There is no specific treatment for FE, and any therapy considered is only symptomatic and palliative. Ideally, affected animals should be kept indoors in darkened buildings during daylight hours and allowed to graze at night. At a minimum they must be given access to shelter. This will prevent the development of further skin lesions and allow the existing lesions to heal while the liver regenerates. Areas where skin is peeling should be dressed with sun-blocking ointments. It may be necessary to dry off severely affected lactating dairy cattle.

Zinc does not prevent FE if given after the sporidesmin challenge, nor does it have a therapeutic effect when given orally to animals that are displaying clinical signs of FE. Zinc cannot reverse liver damage, but can help reduce the risk of further liver damage due to sporidesmin.

Key points

- FE is not a skin disease. The skin condition seen with FE is the result of the liver damage caused by the sporidesmin toxin.
- It is very common to have a significant FE problem without animals showing photosensitisation. It is the 80% of cows without skin lesions, with liver damage, that give the major economic impact.
- FE doesn't just occur on white skin. Even black cattle get FE (Every animal has a liver).
- Zinc can only help prevent FE. It cannot reverse liver damage already done by sporidesmin.

Section Six

Predicting and identifying periods of pasture toxicity

There are two methods farmers can use to attempt to predict and identify periods of pasture toxicity:

- Monitoring weather conditions
- Monitoring spore counts

Monitoring weather conditions

Before the cause of facial eczema (FE) was known it was recognized that pastures tend to become toxic in late summer and autumn when periods of rain or high humidity occur in combination with high nighttime minimum temperatures. Despite recent advances in short and long term weather forecasting, use of weather data to accurately predict FE danger periods has not been very successful in New Zealand or Australia. This is illustrated in Figure 15.

P. chartarum sporulates at the base of pasture under a complex set of conditions – it requires warm conditions and high humidity levels, and the length of time for which humidity is 100% depends not only on rainfall and irrigation, but also on other factors including day length, cloud cover, soil moisture and wind (Brook, 1963). However, current understanding of the associations between these factors does not enable the level of sporidesmin challenge to be accurately predicted using weather data.

We also now know that:

- Germination is all-year round, but sporulation is very seasonal
- There is no fixed beginning or end to the yearly FE danger period and it may extend more than 100 days
- Onset of cold weather does not spell the end of a FE danger period – fungus won't germinate but spores are still there and may persist until May/June
- Hills are worse than flats (not better as commonly believed)
- Rain does not wash spores off pasture

Monitoring spore counts

Spores may be counted either in pasture or in animals' faeces. Pasture spore counting, the older of the two methods, is predictive, in that it tells you what the cows are likely to ingest if allowed to graze the pasture. Faecal spore counting, on the other hand, is reactive in that it provides a more accurate indication of what the animals are actually ingesting and therefore the actual risk of FE.

As shown in Table 1, faecal spore counts tend to be much higher than pasture spore counts. However, this depends on grazing pressure, making faecal spore counts more difficult to interpret.

	Pasture spore count 	Faecal spore count 
High	80,000 – 100,000/g	600,000 – >1,000,000/g
Moderate	20,000 – 80,000/g	100,000 – 600,000/g
Low	0 – 20,000/g	0 – 100,000/g

Table 1. Comparison of pasture and faecal spore counts.
Source: C Mulvaney, AgriNetworks, NZ (2011)

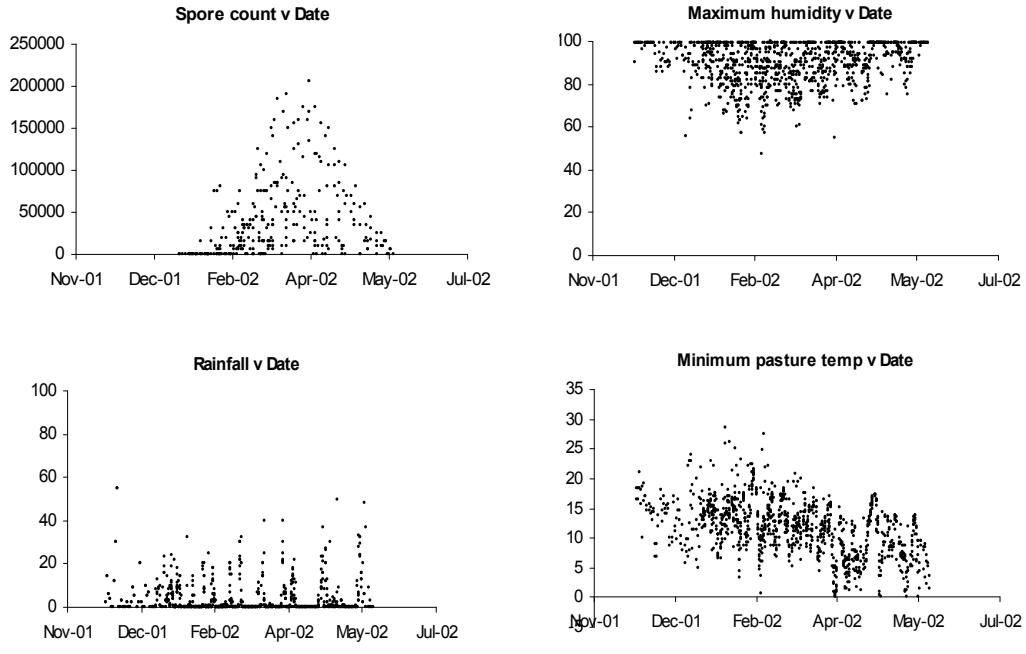

Figure 15. New Zealand study of a farm's spore counts versus local weather conditions.
Source: C Mulvaney, AgriNetworks, NZ (2011)

Relying entirely on prevailing spore counts as shown in Table 1 to assess FE danger level is risky. It does not account for the cumulative effect of ingestion of low amounts of sporidesmin toxin over long periods. Grazing a pasture with a spore count of 10,000 for ten days has the same toxic effect on an animal as grazing a pasture with a spore count of 100,000 for one day.

Although commercial test kits for faecal spore counting are available in NZ, insufficient research work has been done on faecal spore counting to support interpretation (i.e. Low/Moderate/High danger levels).

The FE Working Group believes that use of both pasture spore counting and faecal spore counting in Australia is likely to cause confusion, and believes that industry efforts are best focused on pasture spore counting as the preferred method.

The most favoured method of pasture spore counting is the 'wash method' (after Thornton and Sinclair, 1960). The steps in this method are:

1. Collect pasture sample (it is important to cut pasture to within 1 cm of ground) (See Appendix A, page 29, for method)
2. Mix 60 g of sample grass with 600 mL of water and shake vigorously for 3 minutes
3. Count the spores in the 5 chambers of a Neubauer haemocytometer slide under a good binocular microscope and apply the appropriate multiplication factor for the depth of the slide to give the number of spores per gram of pasture

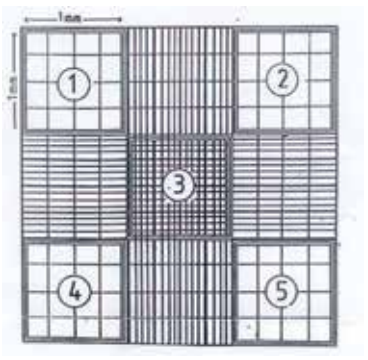

Figure 16. Spore counting using a Neubauer slide under a binocular microscope

As shown in Figure 16, equipment required for pasture spore counting are a Neubauer slide – 0.1mm or 0.2 mm deep – and a good quality binocular microscope, with a moving stage and 100 x magnification.

There is little point monitoring pasture spore counts unless you use them in farm management decisions. Early intervention is critical, when pasture spore counts are trending upwards of 20,000 spores/gram and weather conditions are favourable for sporulation.

As explained by Parkinson, Vermunt and Malmo (2010), when considering whether pastures are safe or unsafe, the following factors need to be considered:

- The amount of dead and dying leaf in the pasture sward
- The grazing intensity and level of pasture being consumed (animals grazing down to the base are at most risk)
- Prior exposure of animals to toxic spores (makes them more susceptible)
- The length of time for which the high spore level is present and consumed

Experimental evidence suggests that previous exposure to small spore rises makes animals more susceptible to further doses. Therefore, even long-term ingestion of low levels of spores may lead to FE.

The examples in Figures 17 and 18 illustrate the extreme differences between farms in the same area and between paddocks on the same farm, over a summer-autumn period.

District warning systems (like that in Figure 19) are therefore of dubious value on their own, and have not been very effective in New Zealand. However, if they are used to trigger spore counting on local farms then they are useful. The VetEnt SporeMap program aims to look at trends in the area, using a set of sentinel farms, on which spore counts are done in 3-4 paddocks every fortnight. If spore counts exceed 20,000/gram on these farms, this serves as a trigger to get other farms in the local area to do their own spore counts.

In response to a recommendation of the FE Working Group, Dairy Australia and the regional development program, GippsDairy, now provide Gippsland dairy farmers with early warning of high risk periods for FE in summer/autumn through a pasture spore monitoring program (www.dairyaustralia.com.au/facialezcema)

Key points

- When local pasture spore counts are trending upwards of 20,000 spores/gram and weather conditions look favourable for sporulation, each farm should monitor its own pasture spore counts week to week and implement FE control and prevention strategies
- Think about germination and sporulation
- Monitor and manage pasture height and quality carefully

Consider every spore as toxic

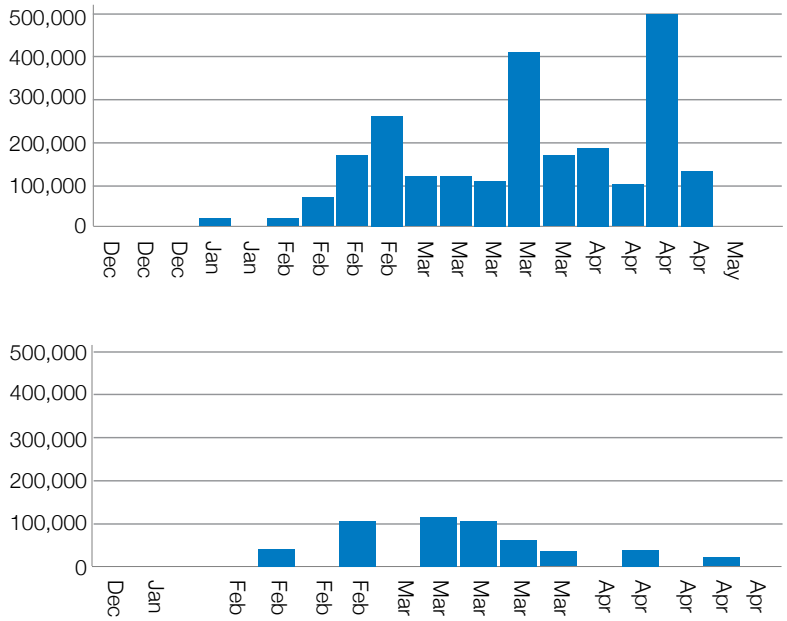

Figure 17. Pasture spore counts – same area, different farms. Source: C Mulvaney, AgriNetworks, NZ (2011)

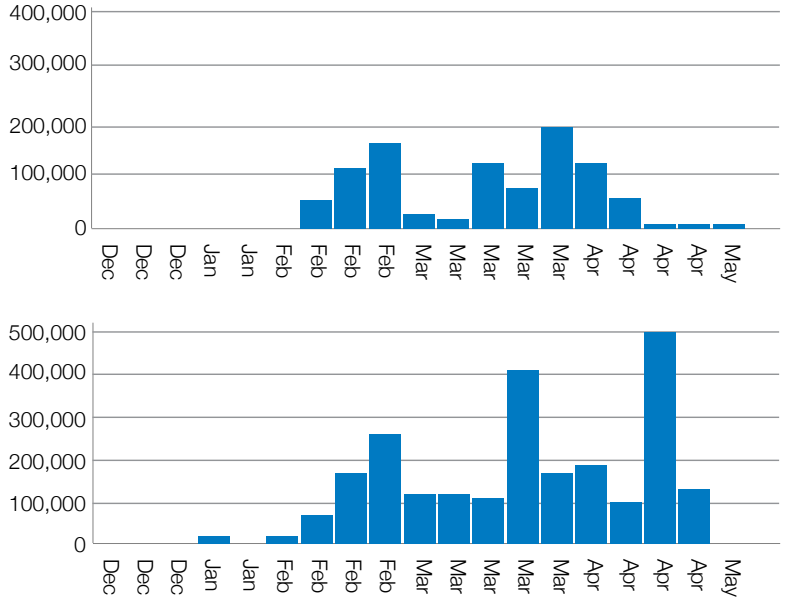

Figure 18. Pasture spore counts – same farm, different paddocks. Source: C Mulvaney, AgriNetworks, NZ (2011)

Figure 19. District FE warning sign in New Zealand

Section Seven

Control and prevention strategies

There are a number of strategies available for control and prevention of facial eczema (FE). These are:

- Avoid the toxin
- Suppress the toxin
- Protect the animal if toxin is ingested with zinc
- Breed for FE tolerance

Avoid the toxin

The toxicity of pastures can be reduced or minimised by reducing the amount of dead and dying leaf which P. chartarum requires for growth and sporulation. This may be achieved through spring/summer by carefully managing pre- and post grazing heights (Figure 20), increasing summer survival of grasses, and controlling pasture pests and diseases. Topping pastures may not help reduce the toxicity of pastures if there is already plenty of dead and dying leaf present for fungal growth and sporulation. (Figure 21).

Figure 20. Carefully managing pre- and post grazing heights helps reduce the toxicity of pasture

Figure 21. Topping may not help reduce the toxicity of pastures

Spore intakes can also be reduced by keeping pasture residuals high, moving cows onto a safer ryegrass pasture area (as determined by spore counts), onto a safer pasture species include clovers, kikuyu, paspalum, tall fescue, and chicory, or by feeding a low FE risk forage crop or supplement such as hay, silage, or grain/concentrate.

Suppress the toxin

In New Zealand, commercial fungicides are available to reduce the toxicity of pastures.

Fungicides will be largely ineffective for pastures with spore counts >200,000/gram, and such pastures remain dangerous to stock. They should only be applied when spore counts are <20,000/gram and pasture is green and growing, as fungicides act systemically.

Respraying must be carried out if more than 25 mm of rain has fallen in a 24-hour period within 3 days of spraying. Pastures should not be grazed within 5 days of spraying, except in emergency. Properly sprayed pastures can remain safe for 6 weeks, but it is advised to monitor spore numbers from the fourth week following application to check for the pastures' safety.

Recently, the carbendazim fungicide (Mycotak®) has been introduced onto the New Zealand market as an effective preventative product for FE. This product is promoted for use in combination with a surfactant (Mycowet®), which takes the fungicide to the base of the plant where it is absorbed, then travels up and throughout the plant. Note – At the time of publication, Mycotak® was not approved for use in Australia.

Protect the animal if toxin is ingested with zinc

In the early 1970s a Waikato farmer, Mrs Gladys Reid, promoted the idea that zinc deficiency was widespread on New Zealand farms and was associated with a number of metabolic problems. This led to research work at the Ruakura Animal Research Station which showed that zinc had a preventative effect on FE in cattle and sheep at approximately 25 times their daily requirement. In 1999, Smith and Embling concluded that the potential economic and welfare gains from the preventative use of zinc in FE control outweighed the risk of zinc toxicity.

Zinc works by forming a complex with sporidesmin, which inhibits sporidesmin's ability to form oxygen free radicals and cause cell damage. Zinc also inhibits intestinal absorption of copper which catalyses the reaction (Munday, 1984 and 1985, Henderson et al 1995).

Zinc supplementation can be effective for FE control and prevention if well managed, as the data in Figure 22 demonstrate.

The desired dietary intake of elemental zinc required when 'prevention dosing' to maintain protective blood serum zinc levels (between 20-35 µmol/L) is 20 mg/kg liveweight/day. As previously discussed, zinc administration should commence 2-3 weeks before pastures become toxic.

'Prevention dosing' should be distinguished from 'crisis dosing' (treating previously non-treated animals with higher levels of zinc oxide during danger periods only, i.e. 25 to 28 mg elemental zinc/kg liveweight/day). Crisis dosing is less effective than prevention dosing, and will only be discussed further in this report with regard to young stock and dry cows.

Options for zinc administration include:

- Zinc sulphate via drinking water
- Zinc oxide by oral drench
- Zinc oxide in feed
- Zinc oxide by intra-ruminal bolus

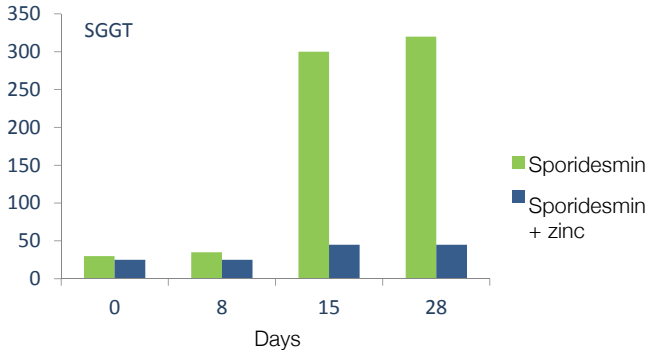

Figure 22. Serum GGT in sheep fed sporidesmin with and without zinc Source: Towers & Smith (1978)

Zinc sulphate is only used for drinking water (first option). Zinc oxide is used for the other three options as it has a greater safety margin than zinc sulphate.

Note - The concentration of elemental zinc and the level of impurities (including lead, cadmium and other heavy metals) varies between different zinc sulphate and zinc oxide products. Always check the certificate of analysis provided by each supplier for its specifications.

Zinc sulphate ($ZnSO_4$) via drinking water

This method, commonly used in New Zealand, involves the proportioning of concentrated zinc sulphate solutions using a Dosatron™ in-line dispenser into the water reticulation system (Figure 23). An alternative but more labour intensive option is the use of floating in-trough dispensers (such as the Peta™ floating in-trough dispenser). Direct addition of zinc sulphate to the water trough without use of a dispenser is not recommended.

Two forms of zinc sulphate are available:

- $ZnSO_4$ monohydrate (36% Zinc), a fine, dry white powder or crystal which is difficult to dissolve.
- $ZnSO_4$ heptahydrate (22% Zinc), a coarse, greenish crystal which is easy to dissolve but can go hard in storage.

Figure 23. Dosatron™ in-line dispenser

Zinc sulphate needs to be gradually introduced over 5 days or more so that the animals become accustomed to the taste of the medicated water. Sweet flavours may also be useful to improve animal acceptance (Figure 24). It is important that no alternative sources of water are available as cows will discriminate against the zinc-treated drinking water.

It can be difficult to deliver the desired dietary intake of elemental zinc of 20 mg/kg liveweight/day to achieve and maintain protective blood serum zinc levels between 20-35 µmol/L using zinc sulphate via drinking water, as a number of factors affect zinc sulphate intake via water troughs, including:

- Zn concentration in the trough
- Daily water intake from the trough, as influenced by:
  - Weather (temp., humidity, rainfall)
  - Alternative water sources (rain; no streams, dams)
  - Animal-to-animal variation
  - Daily milk yield; liveweight
  - Pasture Dry Matter %
  - Whether a masking agent such as a sweet flavour is used

An AgResearch trial in New Zealand involving 8 cows illustrates how variable individual cow intakes of zinc sulphate treated drinking water can be day to day, and the implications this can have for blood serum zinc levels. On a wet day (26 mm) during this trial, water intake from troughs fell to a third (as shown in Figure 25), and the blood serum zinc concentration fell to 70% of dry-day value (as shown in Figure 26).

Figure 24. Flavours make zinc treated water more palatable

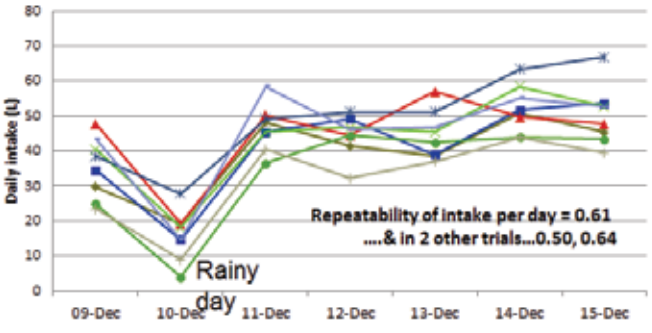

Figure 25. Daily drinking water intakes of eight cows in AgResearch trial. Source: N Cullen, AgResearch, NZ (2011)

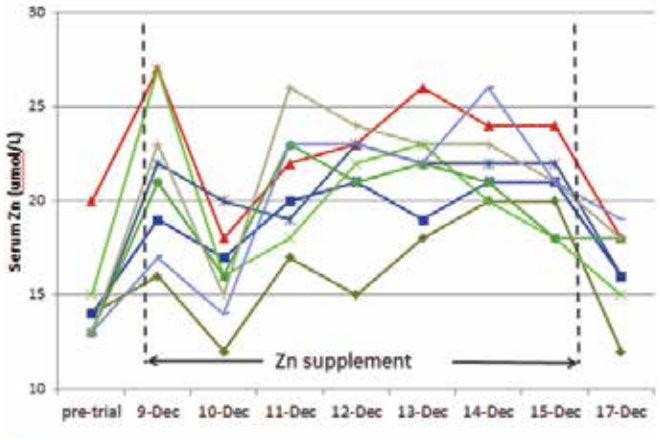

Figure 26. Serum zinc levels of eight cows in AgResearch trial Source: N Cullen, AgResearch, NZ (2011)

Use of zinc sulphate in drinking water to control and prevent FE will not provide protection in a crisis period of high exposure to sporidesmin. It should only be used for low challenge periods.

Farmers need to monitor pasture spore counts and be prepared to change to a more effective system using zinc oxide if pasture spore counts indicate an increased FE risk.

Zinc oxide (ZnO) by oral drench

This approach is commonly used in New Zealand, where most farms do not have the option of supplementing grain/concentrates with zinc and feeding in the dairy bail. Zinc oxide is mixed in the ratio 1:2.5 w/v with water to form a slurry with reasonable stability which will flow through suitable drenching guns.

Zinc oxide can be administered by oral drench to animals daily or every second day. (Higher dose rates will be required if dosing every second day than if dosing daily). Drenching can be made easier using a power doser (Figure 27).

It can be difficult to dose individual animals in a herd accurately for liveweight. Cows should be weighed, classified as small, medium or large, and different dose rates used for each. This is illustrated in a VetEnt case study (2011). See Figure 28.

Figure 27. Power doser

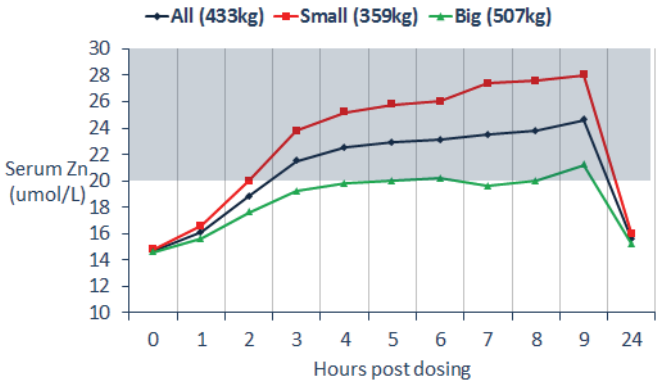

Figure 28. Blood serum zinc levels of small and big cows on a VetEnt case study farm. Source: C Mulvaney, AgriNetworks, NZ (2011)

Risks associated with source of zinc oxide used

As previously explained, the concentration of elemental zinc and the level of impurities (including lead, cadmium and other heavy metals) varies between different zinc oxide (and zinc sulphate) products. This is because zinc oxide is used for a wide variety of applications, including industrial, pharmaceutical, fertiliser and animal feeds.

Always check the certificate of analysis provided by each supplier for its specifications. Only use zinc oxide products suitable for use in animal feeds, containing no more than 200 mg/kg (0.02%) lead and 20 mg/kg (0.002%) cadmium.

Zinc oxide (ZnO) in feed

On most Australian dairy farms, pasture is supplemented with grain/concentrate (in mash or pelleted form), fed in the bail at milking. This provides the opportunity to use the grain/concentrate component of the milker diet as a vehicle to supply cows with zinc oxide and maintain their blood serum zinc level between 20-35 µmol/L. Zinc oxide can also be delivered in molasses.

As stated earlier, a dose of 20 mg elemental zinc/kg liveweight/day is required. The amount of zinc oxide included in each tonne of grain/concentrate must be carefully calculated to achieve this dose, taking into account the amount of grain/concentrate fed per cow per day and the zinc content of the zinc oxide product being used. Too little, and it will not provide adequate protection against high exposure to sporidesmin toxicity, too much and there is the risk of zinc toxicity. See Appendix B (page 30) for a look-up chart on FE prevention dosing rates with zinc oxide in mash/pelleted feed.

Problems supplying zinc oxide via grain/concentrate (mash or pelleted feed) most often occur when:

- the incorrect zinc oxide inclusion rate per tonne of grain/concentrate is used for the daily feeding rate and average herd liveweight
- the herd's average liveweight has been under or over estimated, or the liveweights of animals vary widely
- the zinc oxide settles out of the grain/concentrate before or during feeding

- cows do not receive and consume the intended quantity of zinc-treated feed in the dairy bail
- the feeding rate of the grain/concentrate is increased or decreased by the farmer without an appropriate adjustment in zinc oxide inclusion rate per tonne of grain/concentrate

Blood testing can confirm that a protective blood serum zinc levels (between 20-35 umol/L) is being consistently achieved. See appendix C (page 31) for details.

While there is no research to support any maximum safe zinc supplementation period, be it 60, 80 or 100 days, experience in New Zealand over forty years provides confidence that when administered correctly, prevention dosing with zinc is safe up to 100 days. Beyond 100 days, monitoring blood serum zinc levels in conjunction with a vet becomes more important.

The FE Working Group recommends that a risk management approach be taken, in which:

- only zinc oxide products suitable for use in animal feeds are used ie. those with very low levels of heavy metals and other impurities
- the zinc oxide additive used in all mash feeds is in a pelleted form (rather than powdered form)
- the farmer, stockfeed company, vet and any nutrition advisers involved share responsibility for the zinc supplementation program implemented, and confirm all details in writing

Dosing young stock and dry cows

Prevention dosing of young stock or dry cows with zinc sulphate via drinking water or with zinc oxide in feed may be impractical in many grazing situations. However, 'crisis dosing' previously untreated animals may be necessary during danger periods. This is best achieved using zinc oxide (ZnO) by oral drench (accepting that 'crisis dosing' is less effective than long term prevention dosing).

Drenching 1 to 2 times per week may be adequate for young stock and dry cows.

- Sprinkle 10kg zinc oxide powder into 25 litres water, leave to wet and then stir until lump-free. This produces about 27 litres of drench
- Administer 10ml/100kg liveweight x no. of days between drenches

(Source: Dairy NZ Farm fact sheet '3-7 Facial Eczema - Zinc treatment, recipes and dose rates', 2010)

Zinc oxide (ZnO) intra-ruminal bolus

A controlled release, intra-ruminal zinc bolus is an effective and very safe option for zinc administration. It is particularly useful for treating young stock.

An intra-ruminal zinc bolus consists of a cylindrical core of zinc oxide plus a binding and a releasing agent with a water-impermeable coating except at one end from which the zinc oxide is eroded leaving no final residue.

At the time of publication, controlled release, intra-ruminal zinc bolus products were not approved for use in Australia. However, two intra-ruminal zinc bolus products are marketed in New Zealand: the Time Capsule™ (Agrifeeds) and Face-Guard™ (Bomac). The Time Capsule™ contains 83% zinc oxide and releases 20 mg of zinc oxide/kg liveweight/day over a 4-5 week period (Figure 29). Face-Guard™ is 88% zinc oxide and suitable for young cattle of 90-200kg liveweight and lasts 6 weeks (Figure 30). Repeat administration is necessary for continued protection. Various capsule sizes are available for different weight categories of cattle.

Intra-ruminal boluses provide the opportunity to use a combination of zinc supplementation methods across summer – autumn and reduce the risk of zinc toxicity, e.g. use one or more boluses over the peak FE danger period, and zinc via water or feed in the lower risk period(s).

Figure 29. The Time Capsule™

Figure 30. Face-Guard™

Risks associated with zinc administration

The two risks which need to be managed when controlling and preventing FE with zinc:

If under-dose with zinc: ineffective FE control and prevention

Under-dosing with zinc during a sporidesmin challenge will mean there is insufficient zinc to form inactive complexes with the sporidesmin molecules, allowing more sporidesmin toxin to be absorbed by the animal.

If over-dose with zinc: zinc toxicity

As previously mentioned, the amount of zinc required to prevent FE is close to the level that is likely to cause toxic effects. However, provided correct procedures are followed and animals receive 20 mg elemental zinc/kg liveweight/day, the risk of zinc toxicity is minimal.

Clinical signs of zinc toxicity are dose-dependent, varying from acute gastroenteritis to anorexia and reduced milk production. You may initially see a sudden milk drop plus with some weight loss, rough coats and lack of appetite, and some deaths. Haemolytic anaemia and haemoglobinuria also occur in some animals.

Pathological changes may occur in various organs, and lesions in the kidney and abomasum appear to make the most significant contribution to the deterioration in health of affected animals. However, the pancreas is the only organ that is consistently affected. The pancreatic atrophy of zinc toxicity, on its own, is usually mild in its clinical manifestation, but the abomasitis and haemolytic crises (when they occur) have a more dramatic effect.

In the live animal, zinc toxicity can be diagnosed by measuring blood serum zinc concentrations, bleeding the same 10 cows each time (Target: 20-35 µmol/L). Serum amylase is also a useful test, particularly in chronic cases; levels are usually low due to the reduced amount of functional pancreas tissue. At post-mortem examination the lesions associated with zinc toxicity are abomasal ulceration, pancreatic atrophy and fibrosis.

The FE Working Group has developed a Hazard and Critical Control Point (HACCP) plan for managing risks of zinc toxicity and heavy metal contamination (see Appendix D, page 32). Separate HAACP plans are provided for supplementing with zinc oxide via:

- a commercial mash or pelleted feed (pages 32-33)
- a home-mix mash feed using the farmer's own mixer or additive dispenser (pages 34-35)

Breed for FE tolerance

Individual animals within a herd or flock show a wide range of tolerance levels to FE (as shown by their GGT level in the face of a natural or artificial sporidesmin challenge). There a relatively high genetic component of FE tolerance (heritabilities of the order or higher than those used for milk production traits). Breeding for FE tolerance therefore holds promise as being a very useful long-term approach to managing FE.

Copper supplementation

Prolonged administration of zinc has been associated with hypocupraemia and lower liver levels of copper (and also selenium) in all classes of cattle, although it has not been shown to induce copper (or selenium) deficiency. It has been suggested that reduced availability of copper is one of the mechanisms by which zinc may exert its preventative effect against FE, and that copper supplementation should not be given during the FE season.

Copper supplementation is often given at the same time as prevention dosing of zinc for FE, or given at high levels prior to zinc supplementation to counteract the apparent negative effect of such supplementation on the animals' copper status. Recently it has been found that previous excess copper intake may significantly reduce the efficacy of zinc in preventing FE. Copper intake should therefore be assessed prior to the start of zinc supplementation.

A number of factors have been suggested by Collin et al (1998) as to why some animals are more sporidesmin tolerant than others:

- Differences in how animals absorb sporidesmin from their digestive tract, metabolise and excrete it
- Differences in the resistance of animals' biliary duct linings and other tissues to sporidesmin
- Differences in the ability of animals' tissues to repair damage caused by sporidesmin

It may also be possible that some animals graze at lower levels in the pasture sward where spores are at higher levels, and differ in terms of their rumen microbes and how they metabolise sporidesmin.

An animal's level of tolerance to FE can be assessed by assessing its GGT level in response to an artificial challenge with a dose of sporidesmin or a natural FE challenge when grazing.

As a result of recent work by AgResearch, FE tolerant Holstein-Friesian and Jersey sires are now being marketed in New Zealand for use by dairy farmers who are prepared to discount some breeding worth for a gain in FE tolerance. The next step for the dairy industry is to use the DNA of these FE tolerant dairy sires to develop a reliable DNA marker test.

It is important to remember that when FE tolerant sires are introduced into a herd, FE control and prevention strategies must be continued for several years, until a high level of FE tolerance across the herd has been attained.

Other strategies

Research has also been done into two other strategies to control and prevent FE:

- Biological control using a competitive exclusion approach with atoxigenic isolates of P. chartarum
- Immunisation against sporidesmin

However, these strategies have as yet proved unsuccessful in cattle and are as yet unavailable.

Section Eight

Conclusions

Facial eczema (FE) can have significant impacts on dairy cattle productivity, health and welfare, as seen in outbreaks across Gippsland in January to April 2011. The prevalence of FE in New Zealand, Gippsland and other southern dairying regions of Australia may increase because the effects of global warming may provide suitable conditions for more widespread outbreaks of FE over longer periods.

Despite its name, FE is not a skin disease. The skin condition seen with FE is the result of the liver damage caused by the sporidesmin toxin produced from the spores of the fungus Pithomyces chartarum. Dairy farmers and advisers need to be aware that the major economic impact of FE is from the impacts of sub-clinical liver damage.

FE tends to be mainly associated with perennial ryegrass because of its ability to produce large quantities of dead litter, which supports germination of P. chartarum. Certain weather conditions are favourable to germination or sporulation of P. chartarum. However, to accurately predict FE danger periods and take action to prevent liver damage before it occurs, use of a pasture spore monitoring program is essential. Early intervention is critical. FE control and prevention strategies should be implemented when pasture spore counts are trending upwards of 20,000 spores/gram and weather conditions are favourable for sporulation.

Zinc supplementation can be effective for FE control and prevention if it provides 20 mg elemental zinc/kg liveweight/day, as necessary to maintain protective blood serum zinc levels (between 20-35 µmol/L).

Of the options available in Australia for zinc administration (which do not include intra-ruminal zinc bolus products as at the time of publication none were approved for use in Australia), the use of zinc oxide in grain/concentrate fed as a mash or pelleted feed in the bail at milking is the most common one for milking cows on Australian dairy farms.

A more pro-active approach is required to assess FE risk levels and use FE control and prevention strategies effectively and safely. Avoid ineffective FE control and prevention as a result of under-dosing with zinc and zinc toxicity from over-dosing. Two key elements of a pro-active approach are:

- A regional pasture spore monitoring program, using a set of ‘sentinel farms’, which provides local farms with weekly spore count data and triggers farms to commence their own spore counting when the FE risk level rises
- Shared responsibility for implementation of each zinc supplementation program by the farmer, stockfeed company, vet and any nutrition advisers involved, using a HACCP approach to manage the risk of zinc toxicity

Breeding for FE tolerance holds promise as being a very useful long-term approach to managing FE. Joint development of genomic tools by New Zealand and Australian dairy industry partners should therefore be considered.

Section Nine

Appendices

Appendix A

Method for collecting pasture samples for spore counting

To ensure maximum reliability of pasture spore counts, a consistent approach to collecting pasture samples is required. Samples must be cut at the same height above the ground from the same paddocks each time.

Paddock selection

1. To get a reasonable indication of the spore count trends on a farm, select 2 paddocks that represent the different types of land on the farm e.g. flats, hills and steep hills.
2. Pick and mark out a sample line between two points diagonally across the whole paddock. The sample line should avoid parts of paddocks sheltered by trees and hedges. This line is to be used each time a sample is collected.

Collection of pasture sample

1. Label paper bags
   a. Farm name and date
   b. Paddock name
   c. Type of land, e.g. flats, steep hill, north facing, south facing
2. Collect sample
   a. Using scissors or shears cut a handful of pasture every 10-15 paces along the sample line. Avoid pasture close to urine and dung spots and ensure no dirt is collected
   b. Cut 1 cm above ground level
   c. Fill 2/3 of the bag with pasture
3. Despatch sample
   a. Put paper bag into an overnight courier bag addressed to the laboratory
   b. Take into the local post office as soon as possible (before closing on the day of sampling)

Appendix B

Look-up chart: Facial Eczema prevention dosing with zinc oxide in mash/pelleted feed

Daily intake of medicated feed, kg/cow/day	Zinc oxide inclusion rate (kg/tonne feed) (assuming 80% elemental zinc) to give 20 mg elemental zinc/kg liveweight/day						
	450 kg animal	500 kg animal	550 kg animal	600 kg animal	650 kg animal	700 kg animal	750 kg animal
1.0	11.25	12.5	13.75	15	16.25	17.5	18.75
2.0	5.625	6.25	6.88	7.5	8.13	8.75	9.38
3.0	3.75	4.17	4.59	5	5.42	5.84	6.25
4.0	2.81	3.13	3.44	3.75	4.07	4.38	4.69
5.0	2.25	2.5	2.75	3	3.25	3.5	3.75
6.0	1.88	2.09	2.30	2.5	2.71	2.92	3.13
7.0	1.61	1.79	1.97	2.15	2.32	2.5	2.68
8.0	1.41	1.57	1.72	1.88	2.03	2.19	2.35
9.0	1.25	1.39	1.53	1.67	1.81	1.95	2.09
10.0	1.125	1.25	1.38	1.5	1.63	1.75	1.88

Note - If a feed additive is used with contains a lower % elemental zinc, the inclusion rate necessary to give 20 mg elemental zinc/kg liveweight/day will differ accordingly.

Appendix C

Monitoring blood serum zinc levels of animals

Blood testing can confirm that a protective blood serum Zinc level (between 20-35 umol/L) is being consistently achieved through zinc supplementation.

Blood testing is recommended 3 to 4 weeks after commencing zinc oxide supplementation, and may be worth repeating if:

- this test indicates any problems with zinc supplementation
- animals are to be supplemented with zinc beyond 100 days

Farmers should arrange for their vet to blood test 10 cows. 8 mls of blood should be collected in a plain tube from 5 smaller cows and 5 larger cows in the herd. The ID number of each cow tested should be recorded.

Blood tubes should then be despatched to a veterinary laboratory for blood serum zinc analysis. Farmers should expect to pay approximately $600 to $650 (including lab. testing fees and vet's time and materials, but not travel costs).

Results should be available within 4-5 days and need to be interpreted carefully by the farmer and their vet.

- If most of the 10 cows tested are found to have blood serum zinc levels above or below the target range (20-35 umol/L), this indicates consistent over-dosing or under-dosing.
- If cows' blood serum zinc levels are found to be very inconsistent (ie. some are above while others are below the target range), this indicates a more complex problem such as inconsistent mixing of the zinc oxide additive in grain/concentrate, and / or inadequate control over the allocation and consumption of the grain/concentrate per cow at milking.

Following consultation with their vet regarding the blood test results, farmers may require advice from a stockfeed company or nutrition adviser if a problem is identified.

As discussed on pages 23-24, problems supplying zinc oxide via grain/concentrate (mash or pelleted feed) most often occur when:

- the incorrect zinc oxide inclusion rate per tonne of grain/concentrate is used for the daily feeding rate and average herd liveweight
- the herd's average liveweight is under or over estimated, or the liveweights of animals within the herd vary widely
- the zinc oxide settles out of the grain/concentrate before or during feeding
- cows do not receive and consume the intended quantity of zinc-treated feed in the dairy bail
- the feeding rate of the grain/concentrate is increased or decreased by the farmer without an appropriate adjustment in zinc oxide inclusion rate per tonne of grain/concentrate

Problem investigation should therefore include:

- Confirmation that the daily elemental zinc dosage has been correctly calculated (20 mg elemental zinc / kg liveweight / day), based on:
  - Animal liveweights
  - Zinc oxide inclusion rate currently being used in milker feed (kg / tonne feed)
  - Quantity of grain/concentrate currently being fed per cow per day
- Confirmation that the amount of grain/concentrate being consumed by cows is as required:
  - Quantity of grain/concentrate being dropped per bail in the dairy
  - Quantity of grain/concentrate being consumed by each cow

Collection of representative feed samples and submission to a feed laboratory for zinc analysis may also be necessary.

Appendix D

Facial Eczema HACCP plan Hazard Analysis – zinc toxicity and heavy metal contamination

Zinc oxide supplementation of commercial stockfeed (mash or pelleted feed)

Process step	Hazard	Source	Hazard rating	Preventative measure
1. Farmer decides to start zinc oxide supplementation in feed	Farmer starts zinc oxide supplementation in feed before necessary (i.e. when the FE risk level is still low)	Farmer	Low	**Farmer** starts monitoring pasture spore counts weekly on own farm when triggered by spore count alert from local sentinel farm **Farmer** starts zinc oxide supplementation in feed when own pasture spore counts trend upwards of 20,000 spores/gram and weather conditions favour fungal sporulation
2. Farmer orders first custom load of mash/pellets from stockfeed company with zinc oxide at FE prevention level	Incorrect zinc oxide inclusion rate/tonne feed is calculated for daily mash/pellet feeding rate per cow to be used: • If too low, poor protection from toxin • If too high, risk of zinc toxicity	Farmer and stockfeed company	Medium	**Farmer** advises stockfeed company in writing of: • daily mash/pellet feeding rate (kg/cow/day) to be used while load is fed • average herd liveweight and range • date intends to start feeding **Stockfeed company** sales staff are trained to confirm with farmer his intended daily mash/pellet feeding rate per cow and average herd liveweight and calculate appropriate zinc oxide inclusion rate/tonne feed to deliver 20 mg elemental zinc/kg liveweight/day as recommended for prevention dosing
3. Stockfeed company manufactures first custom load of mash/pellets according to farmer's order	Zinc oxide additive used is unsuitable for use in animal feeds, containing unacceptable levels of lead, cadmium and other heavy metals	Stockfeed company	Low	**Stockfeed company** only sells a custom load of mash/pellets with zinc oxide at FE prevention dose if it: • only uses a zinc oxide additive in a distinctive, well labelled bag with a certificate of analysis provided by the supplier which confirms it is suitable for use in animal feeds, containing no more than 200 mg/kg (0.02%) lead and 20 mg/kg (0.002%) cadmium • is FeedSafe accredited • has a suitable mixer which is regularly maintained and is checked to ensure its coefficient of variation is greater than 0.8 • uses signed batch sheets • keeps retention samples of every batch of feed on all mash feeds • only uses a pelleted form of zinc oxide additive if to be included in a mash feed • provides a certificate of conformance (i.e. fit for purpose statement) for the feed **Stockfeed company** tests one in every 20 production batches to confirm elemental zinc level is to intended specification
	Incorrect zinc oxide inclusion rate/tonne feed is used	Stockfeed company	Low (if FeedSafe accredited and pellets) Medium to High (if not FeedSafe accredited and/or mash feed)	
	Zinc oxide is not uniformly dispersed in feed during mixing and/or settles out after mixing as conveyed into outloading bins and delivery truck	Stockfeed company		
	Zinc sulphate used instead of zinc oxide	Stockfeed company	Low	
4. Stockfeed company delivers first custom load of mash/pellets to farm and augers/blows into designated farm silo	Zinc oxide separates out so is no longer uniformly dispersed in feed	Stockfeed company	Medium (if mash feed) Low (if pellets)	**Farmer** only orders a pelleted feed or a mash feed which includes a pelleted zinc oxide additive **Stockfeed company**: • ensures blower pipe is directed away from the walls of the silo • does not use high pressure blower trucks to deliver mash feeds supplemented with extra zinc oxide • records intended farm silo on feed order form and delivery docket **Farmer** clearly labels all farm silos
	Mash/pellets delivered into wrong farm silo	Stockfeed company and farmer	Low	

Process step	Hazard	Source	Hazard rating	Preventative measure
5. Farmer feeds first custom load of mash/pellets to cows in dairy bails	If herringbone dairy: • Has open trough rather than individual bails • Feeding system does not drop equal quantity of feed in each bail • Has mismatch between number of cows and number of bails	Farmer	High	**Farmer**: • monitors effectiveness of program in conjunction with vet, checking at least 10 cows' blood serum zinc levels to confirm desired zinc level of 20-35 µmol/L is being consistently achieved • does not use zinc oxide supplementation if cannot provide individual bail for each cow • switches off individual cow feeding system • regularly checks bail feeding system to ensure correct quantity of feed is consistently dropped in all bails • regularly cleans out bails if excess residual feed • regularly checks expected versus actual usage of mash/pellets • orders a small load so can re-order feed with a different zinc oxide inclusion rate sooner if likely to want to change daily mash/pellet feeding rate (kg/cow/day) **Stockfeed company** provides farmer with a delivery docket with every load of feed which clearly states: • zinc oxide inclusion rate/tonne feed • reco. daily mash/pellet feeding rate (kg/cow/day) and includes a bold warning that varying the daily cow feeding rate of that load of feed from that specified may result in risk of toxicity (if increased) or reduced protection from toxin (if decreased)
	All dairies: • Quantity of feed dropped per bail is incorrect • Feed is not eaten by all cows and builds up in bails • Feeding cows individually at different rates versus at a flat rate	Farmer	High	
	Herd has a wide range in liveweights	Farmer	Medium	
	Farmer changes daily mash/pellet feeding rate (kg/cow/day) before custom load of feed is consumed	Farmer	High	
	Early clinical signs of zinc toxicity in cows are not recognised and vet is not consulted to investigate	Farmer	Medium	
6. Farmer orders, receives and uses additional custom loads of mash/ pellets	Farmer orders repeat loads at previous zinc oxide inclusion rate (kg/ tonne feed) despite changing daily grain/pellet feeding rate (kg/cow/day)	Farmer	Medium	**Stockfeed company** provides farmer with a delivery docket with every load of feed which clearly states: • zinc oxide inclusion rate/tonne feed • reco. daily mash/pellet feeding rate (kg/cow/day) • that the daily mash/pellet feeding rate (kg/cow/ day) must not be changed **Farmer** takes preventative measures listed for Steps 2, 4 and 5 **Farmer** continues to monitor pasture spore counts regularly on own farm and stops using zinc oxide supplementation in mash /pellets when own pasture spore counts trend downwards of 20,000 spores/gram and weather conditions no longer favour fungal sporulation **Farmer** consults vet if wishes to extend zinc administration via feed or other means for more than 100 days' duration.
	Farmer continues to order custom load of grain mix/ pellets from stockfeed company with zinc oxide at FE prevention level preventative level, despite reduced FE risk level	Farmer	High	
	Early clinical signs of zinc toxicity in cows are not recognised and vet is not consulted to investigate	Farmer	Medium	
	Other hazards as per Steps 2 to 5	Stockfeed company and farmer	Low to High	
7. Farmer decides to stop zinc oxide supplementation in feed	Farmer continues zinc oxide supplementation in feed longer than is necessary (i.e. after FE risk level has fallen to low)	Farmer	Low	**Farmer** continues to monitor pasture spore counts regularly on own farm and stops using zinc oxide supplementation in mash /pellets when own pasture spore counts trend downwards of 20,000 spores/gram and weather conditions no longer favour fungal sporulation

Zinc oxide supplementation of home mix mash feed via mixer or additive dispenser

Process step	Hazard	Source	Ranking	Preventative measure
1. Farmer decides to start zinc oxide supplementation in feed	Farmer starts zinc oxide supplementation in feed before necessary (i.e. when the FE risk level is still low)	Farmer	Low	**Farmer** starts monitoring pasture spore counts weekly on own farm when triggered by spore count alert from local sentinel farm Farmer starts zinc oxide supplementation in feed when own pasture spore counts trend upwards of 20,000 spores/gram and weather conditions favour fungal sporulation
2. Farmer plans first custom batch of home-mix mash feed with zinc oxide at FE prevention level	Incorrect zinc oxide inclusion rate/tonne feed is calculated for daily mash/pellet feeding rate per cow to be used: • If too low, poor protection from toxin • If too high, risk of zinc toxicity	Farmer	High	**Farmer** determines: • daily mash/pellet feeding rate (kg/cow/day) to be used while batch is fed • average herd liveweight and range **Farmer** calculates appropriate zinc oxide inclusion rate/tonne feed to deliver 20 mg elemental zinc/kg liveweight/day as recommended for prevention dosing
3. Farmer manufactures first custom batch of mash feed according to own requirements	Zinc oxide additive used is unsuitable for use in animal feeds, containing unacceptable levels of lead, cadmium and other heavy metals	Farmer	Low to Medium	**Farmer** only manufactures own custom batch of feed with zinc oxide at FE prevention dose if he/she: • only uses a zinc oxide additive in a distinctive, well labelled bag with a certificate of analysis provided by the supplier which confirms it is suitable for use in animal feeds, containing no more than 200 mg/kg (0.02%) lead and 20 mg/kg (0.002%) cadmium • has a suitable mixer which is regularly maintained and checked to ensure its coefficient of variation is greater than 0.8 OR has an additive dispenser which is correctly calibrated • uses signed batch sheets • keeps retention samples of every batch of feed
	Incorrect zinc oxide inclusion rate/tonne feed is used	Farmer	Medium	
	Zinc oxide is not uniformly dispersed in feed during mixing	Farmer	Medium to High	
	Zinc sulphate used instead of zinc oxide	Farmer	Low	
4. Farmer augers first custom batch of mash feed into designated silo	Zinc oxide separates out as conveyed into silo so is no longer uniformly dispersed in feed	Farmer	Medium	**Farmer** uses a suitable auger and positions a designated silo nearby **Farmer** clearly labels all farm silos
	Mash feed delivered into wrong farm silo	Farmer	Low	

Process step	Hazard	Source	Ranking	Preventative measure
5. Farmer feeds first custom batch of mash feed to cows in dairy bails	If herringbone dairy: • Has open trough rather than individual bails • Feeding system does not drop equal quantity of feed in each bail • Has mismatch between number of cows and number of bails	Farmer	High	**Farmer:** • monitors effectiveness of program in conjunction with vet, checking at least 10 cows' blood serum zinc levels to confirm desired zinc level of 20-35 µmol/L is being consistently achieved • does not use zinc oxide supplementation if cannot provide individual bail for each cow • switches off individual cow feeding system • regularly checks bail feeding system to ensure correct quantity of feed is consistently dropped in all bails • regularly cleans out bails if excess residual feed • regularly checks expected versus actual usage of mash feed • makes a small batch so can make feed with a different zinc oxide inclusion rate sooner if likely to want to change daily mash feeding rate (kg/cow/day) **Farmer** understands that varying the daily cow feeding rate of that batch of feed from that specified may result in risk of toxicity(if increased) or reduced protection from toxin (if decreased)
	All dairies: • Quantity of feed dropped per bail is incorrect • Feed is not eaten by all cows and builds up in bails • Feeding cows individually at different rates versus at a flat rate	Farmer	High	
	Herd has a wide range in liveweights	Farmer	Low	
	Farmer changes daily mash feeding rate (kg/cow/day) before custom batch of feed is consumed	Farmer	High	
	Early clinical signs of zinc toxicity in cows are not recognised and vet is not consulted to investigate	Farmer	Medium	
6. Farmer manufactures and uses additional custom batches of mash feed	Farmer makes repeat batches at previous zinc inclusion rate/tonne feed despite changing daily mash feeding rate (kg/cow/day)	Farmer	Medium	**Farmer** takes preventative measures listed for Steps 2 to 5 **Farmer** continues to monitor pasture spore counts regularly on own farm and stops using zinc oxide supplementation in mash feed when own pasture spore counts trend downwards of 20,000 spores/gram and weather conditions no longer favour fungal sporulation **Farmer** consults vet if wishes to extend zinc administration via feed or other means for more than 100 days' duration.
	Farmer continues to make custom batches of mash feed with zinc oxide at FE prevention level preventative level, despite reduced FE risk level	Farmer	High	
	Early clinical signs of zinc toxicity in cows are not recognised and vet is not consulted to investigate	Farmer	Medium	
	Other hazards as per Steps 2 to 5	Farmer	Low to High	
7. Farmer decides to stop zinc oxide supplementation in feed	Farmer continues zinc oxide supplementation in feed longer than is necessary (i.e. after FE risk level has fallen to low)	Farmer	Low	Farmer continues to monitor pasture spore counts regularly on own farm and stops using zinc oxide supplementation in mash feed when own pasture spore counts trend downwards of 20,000 spores/gram and weather conditions no longer favour fungal sporulation

Section Ten

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