Facial eczema: an invisible drain on production

Key points

- NZ pastures have a number of fungi that produce toxins that can markedly reduce productivity. Facial eczema (FE) is the most common.
- While clinical FE is well-known and easily recognised, farmers are often unaware of major lifetime losses due to sub-clinical FE.
- FE outbreaks can be predicted from weather patterns and FE levels can be easily monitored from pasture or dung samples.
- In FE-prone areas, it is advisable to use rams that have been selected for FE-tolerance.
- Zinc boluses, while expensive, are effective in reducing the risk of losses due to FE.

New Zealand pastures contain fungi that can result in large invisible farm productivity losses.

Poisons known as mycotoxins produced by the fungi cause facial eczema (FE), ryegrass staggers and other conditions.

These affect sheep, cattle, deer and other ruminants and are major contributors to summer/autumn ill thrift of young stock, especially in the warmer north.

Facial eczema fungi (*Pithomyces chartarum*) live on dead pasture litter. Sporidesmin toxin is found in the spores of the fungus.

Clinical FE is well-known. It can occur from summer to late autumn when conditions are moist and temperatures warm.

The toxin causes liver damage, which, in turn, causes jaundice. Bile pigments and photosensitive plant pigments are released into the bloodstream.

The animal becomes sunburnt. The face and ears become painfully swollen and may become scabby. Large areas of skin may be lost.

Other parts of the body such as the udders and legs can also become affected. The animals seek shade.

Deaths can occur three to four weeks after consuming high levels of toxins, but a lot of the animals appear to recover. However, some liver damage remains and symptoms can appear later if the animal is stressed. This occurs most commonly in late pregnancy or lactation in ewes.

Sub-clinical FE

Sub-clinical FE is less well-known, but in many seasons it can lead to reduced liveweight gains and lower lambing percentages, without animals showing any of the classical eczema symptoms.

Some farmers believe, incorrectly, they don't have FE until they see clinical symptoms. In fact, by the time a flock has 5 per cent clinicals, then at least 50 per cent of the flock is likely to have the disease at a sub-clinical level.

When the liver is damaged by FE – even mildly – a liver enzyme called GGT is released into the blood. The higher the GGT level, the greater the resulting production loss.

The liver can repair some of the damage but part of the damage will be permanent and cumulative. This is quite obvious if the liver is examined after a post-mortem. The bulk of the liver will be hard, dull and fibrous, while the repair tissue appears as red lobes or outgrowths from the old liver.

Exposure to GGT can reduce lifetime performance. Hoggets that had high levels of GGT (> 350 i.u. GGT/l) have been shown to have lower fertility over the next four seasons.

While farmers often focus on FE damage and production losses arising from major outbreaks, these can also occur as a result of frequent exposure to smaller doses of toxin.

Control measures

FE outbreaks can be predicted from weather patterns (rain and temperature) and are often published in newspapers in high-risk areas.

FE challenge levels can be easily monitored from pasture or dung samples. Sampling and testing can be carried out by vets, or by farmers themselves. Blood samples can be used to determine levels of liver damage.

Pasture tests indicate the challenge animals are being exposed to, but not necessarily what they are eating. Dung samples tell the farmer what the animal has actually ingested – with the downside that the results are after the fact.

FE occurs mainly in fertile ryegrass-dominant pastures, so stock can be offered alternative feeds such as hay, silage, concentrates or forage crops. Be aware, however, that hay too may contain endophytes, so is not without risk.

Grazing just the tops of the ryegrass pastures is one way of reducing the risk of exposure to the toxic spores which are generally found at the base of the sward in the litter. Grazing cooler southerly faces or dryer northerly faces, which are low in ryegrass, will also help. But be mindful that these grazing strategies reduce risk – they do not eliminate it.

Farmers can breed for FE tolerance in their sheep and breeders of FE-tolerant rams advertise widely. Farmers in FE-prone areas are advised to use rams that have been selected for FE-tolerance.

Spraying with fungicides, such as benlate or thiabendazole, reduces spore accumulation. However, this needs to be done early in the season before spore counts rise, and also expensive if the FE challenge turns out to be low.

Zinc dosing is a good preventative, as is water treatment, daily zinc oxide dosing or use of a slow release zinc oxide bolus ("Time Capsule").

The bolus is the most convenient as it lasts a number of weeks, but it is more expensive than the other methods of administration. Zinc toxicity is also a risk if more than three boluses are used in succession. Boluses need to be administered carefully as damage to the throat can be fatal.