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Technical Series

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Managing facial eczema

Facial eczema (FE) occurs when grazing animals eat pasture containing large numbers of spores from the fungus *Pithomyces chartarum*. The damage is incremental, and disease occurs both from short-term ingestion of pasture with high spore counts and long-term ingestion of pasture with more moderate spore counts.

Page 2

Lame cows – are we identifying them quickly enough?

Studies have shown that farmers generally detect about one third of cows with impaired mobility. Mobility scoring detects lame cows earlier than farmer observations. Significant delays can occur between the onset of reduced mobility and provision of treatment. These delays increase the economic impact of lameness.

Page 9

Effective control of parasitic nematodes – an issue for dairy farmers?

Nematode parasites have a significant effect on productivity and profitability in dairy cattle. There are a limited number of drench families available and there is significant variability in the effectiveness of drench products currently on the market.

Page 12

Recently published by DairyNZ

DairyNZ researchers publish their findings.

Page 14

BVD – What does it do and what can you do about it?

Bovine viral diarrhoea (BVD) virus has been recognised for a long time but in the last 10 to 20 years understanding of the disease has advanced considerably and better tools to control it have been developed.

Page 15

Focus on international research

Brief summaries of key international science papers recently published.

Page 20

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Managing facial eczema

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The disease

Facial eczema (FE) occurs when grazing animals eat pasture containing large numbers of spores from the fungus *Pithomyces chartarum*. They contain sporidesmin, a toxin which causes inflammation of the liver and bile ducts. The damage is incremental, and disease occurs both from short-term ingestion of pasture with high spore counts and long-term ingestion of pasture with more moderate spore counts. While the cow's liver has some capacity to heal and regenerate, there is often long-term compromise to its function.

One immediate effect of exposure to sporidesmin is a drop in production and there may be transient diarrhoea. The toxin is concentrated in bile where it generates free oxygen radicals (super-oxides) which cause massive cellular damage especially in the bile ducts. This damage blocks the flow of bile allowing the light-reactive substance phytoporphyrin (also known as phylloerythrin) to accumulate in blood and tissue fluids making the animal photosensitive. Phytoporphyrin is produced during microbial fermentation of the green plant pigment chlorophyll in the rumen but is cleared through the bile when liver function is normal[1].

When it accumulates, if lightly-pigmented skin is exposed to sunlight, the resulting deep tissue burns to produce the characteristic skin lesions of FE. This disease process has a time-lag, so skin lesions do not become evident until 10-14 days after spore ingestion. Severe skin lesions do not heal well, and cows may develop "skin horns" on affected areas. Sometimes the toxin causes circulating red blood cells to break down and blood pigments will stain the urine red (red water). Sporidesmin is also excreted through the kidneys and can cause cystitis and frequent urination[2,3].

Only about 10% of affected animals show clinical signs, for every clinical case there will be 10 cows with sub-clinical FE[4]. The extent of subclinical disease can be monitored by measuring levels of gamma glutamyl transferase (GGT) in blood which is closely correlated with the amount of liver damage. Levels of 250 IU/L indicate moderate damage. Sub-clinical FE cows may show clinical signs after cumulative doses of toxin, or they may lose production and body condition, be unable to regain condition after drying off, or suffer liver failure and "spring eczema" (photosensitisation signs) early in the subsequent season[5].

Predicting risk

Spore numbers increase in summer and autumn, January to May are the high risk months. Conditions are favourable for spore production when overnight minimum grass temperatures stay at or above 12°C over four consecutive nights, and humidity is high e.g., with drizzly rain (4-6 mm/48 h), or when soil is kept moist by irrigation. In natural outbreaks spore counts usually show one or two small increases over several weeks, followed by a major rapid rise when the right weather conditions occur. Given that young spores contain more toxin, pastures are more toxic when conditions promote rapid fungal growth. Conditions for spore growth are also conducive to pasture growth. The amount of dead plant material in the base of the pasture increases, providing an ideal environment for spore production. Making animals graze into the pasture base adds to the risk.

The risk of exposure to the toxin is traditionally predicted by counting spores, which look like microscopic hand grenades, in pasture washings. This gives a useful prediction of risk, but there are two caveats. Fungal growth does not occur evenly across a farm, or indeed across a paddock. Counts may be higher in "hot spots" such as sheltered hollows, alongside hedges, and on north-facing slopes of hills. Secondly, the toxin will leach as spores age, a process accelerated by heavy rain, so counts may over-predict risk. While district counts can be used as a general guide, spore counts for individual paddocks are needed to predict risk accurately[2].

The general recommendation is that preventative treatments begin before pasture spore counts rise above 20,000/g pasture. Counts of 40,000/g or more should be considered toxic, and control measures will be required to limit liver damage.

Laboratories and veterinary clinics have established processes to collate pasture spore count information over summer and autumn. This information is readily available from veterinary clinics and websites such as **www.gribblesvets.co.nz** and describes district trends. Local variations can be large however, and farms with a history of FE outbreaks should conduct their own monitoring programmes.

Faecal spore counts are a new approach to FE risk assessment, providing a direct assessment of spore ingestion. The relationship between pasture and faecal counts was investigated in sheep revealing that faecal spore counts of 600,000-1,000,000 /g occurred during moderate challenge conditions[6]. Spores are concentrated in faeces, but in cattle the larger volume of more liquid faeces is likely to produce some dilution. The tentative recommendation for dairy cows is that faecal spore counts exceeding 75,000-100,000 spores/g faeces represent spore intakes for which preventative treatments should be implemented[3]. It is relatively simple to collect faecal samples for analysis from the dairy yard following milking and commercial laboratories offer analyses. Faecal zinc levels can also be analysed as an indication of whether supplementation is sufficient.

Treatments to prevent or reduce liver damage

There is no effective treatment that can cure cows once they are affected, although the liver has a natural capacity to recover its function. The usual approach once disease is apparent is to provide palliative treatments to support liver healing (vitamins and oil supplements) while protecting animals from UV light by providing shade or applying ointments.

Clearly the best approach is to prevent the liver damage by avoiding spore ingestion; but this is problematic where grazed pasture is a key component of the diet and high risk conditions continue for weeks on end. Control methods in the past have generally focused on preventing or limiting the extent of liver damage rather than limiting spore growth. More recently fungicide pasture spraying has become an additional method to try to prevent FE damage.

Pasture spraying

Spraying pastures with a fungicide that both kills the fungus and inhibits spore production is the only direct means to manage spore numbers on pasture during risk periods. Fungicidal sprays provide a secondary benefit by controlling rusts on ryegrass and are also an opportunity to apply broad-leaf herbicides to improve pasture quality.

Carbendazim-based sprays, with nil milk with-holding periods, are marketed by several companies and performance can generally be expected to be similar provided a surfactant is included in the spray to aid spread over the sward and litter, and protect against rain. If heavy rain (more than 25 mm/24 h) occurs within three days of application, pasture should be re-sprayed. One commercial company claims that their surfactant provides protection from rain within three hours of application. Applications should include areas along fence-lines and under trees and hedges, so aerial spraying may require some land-based follow-up to manage these potential hot-spots. Carbendazim sprays are very toxic to aquatic organisms, so care must be taken to avoid contamination of water bodies.

Best protection is achieved when stock graze pasture seven to ten days after spraying, so control by pasture spraying should be done in anticipation of the danger period, i.e. before pasture spore counts exceed 20,000 /g pasture. Ongoing pasture spore counts are recommended in order to monitor conditions.

Depending on location and risk, it may be possible to spray only part of the farm so that safe pasture is available when needed. Farms with a high risk of FE should coordinate paddock rotation with the spray programme so that grazing blocks are treated every 14-21 days while risk remains high. A single application (with surfactant) is reported to reduce spore counts for up to 6 weeks, but field observations suggest that protection may be only three to four weeks if weather conditions favour fungal growth.

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Spore counts should, therefore, be done before grazing to determine if the pasture is still safe. Integration of the spray programme with grazing rotation can be problematical, especially if the risk is ongoing for several months, and alternative safe forages and crops should also be considered as a means to reduce spore intakes.

The effect of pasture spraying where spore counts are already high (>200,000 /g pasture) has not been well researched. In these situations pastures will remain toxic until existing spores have leached out, but further spore production will be reduced 24 hour after spraying. This may provide a management option in emergency situations, but cows will still be exposed to sporidesmin.

Other pasture management approaches

The fungus is saprophytic, growing on dead plant material, so pastures with a lot of debris have higher counts. This has led to the commonly-held view that topping pastures will increase the risk of spore growth. Early efforts to manage FE focussed on pasture management and topping was thought to increase risk for lambs[2]. Topping was also used as a tool to maximise spore counts in investigations of treatments for FE[7]. Currently, some advisors believe that modern dairy grazing practices and pasture types have changed so that topping no longer alters risk, but there is no definitive proof for this view, and further research is required to clarify if topping dairy pastures increases risk.

The fungus seems to associate more with perennial ryegrass pastures. Clover, kikuyu, paspalum and fescue pastures generally harbour lower spore numbers. Alternative summer feed crops (brassicas, chicory, chopped green maize) and stored feeds (pasture silage, hay) do not contain spores and may be useful to reduce exposure during dangerous periods[3].

Zinc treatments

Since the 1970s, zinc dosing has been the predominant prophylactic to protect the liver against damage by sporidesmin, but blood concentrations of zinc need to attain the recommended range of 18-34 µmol/L **(www.gribblesvets.co.nz)**. Zinc is not 100% effective but correct dosing with either zinc oxide or zinc sulphate at the time of challenge can be expected to reduce the proportion of animals showing severe signs by 80-90%[7,8].

Zinc is not stored long-term in the body, so a programme of continuous dosing is required in anticipation of toxin challenge. To achieve recommended blood levels requires zinc supplementation at a rate of 15-20 mg elemental zinc/kg liveweight/day[8].

The actual mechanism of protection is not well understood, but the general view is that zinc inhibits super-oxide production when bile duct cells are exposed to sporidesmin reducing cell damage[8,9]. Recent studies of sheep and calves treated with elemental zinc bolus devices also suggest that high levels of zinc within the gastro-intestinal contents may provide protection, independent of absorption into the animal's bloodstream. Faecal zinc levels above 200 mg/kg (fresh weight) appear to provide protection[10,11].

To be effective, the zinc must be in the animal's system before the challenge occurs; it does not protect or heal the damage once done.

Two zinc salts (sulphate and oxide), as well as elemental zinc, can be used to protect cows from toxin damage:

- Zinc sulphate is used for drinking water treatment – this is a highly soluble white or greenish-white crystal and easy to disperse in drinking water. It makes the water slightly acid and alters the taste, which can be overcome to some extent by adding flavouring agents. Zinc sulphate should not be used to drench cows – it will stimulate the oesophageal groove to close, diverting the solution straight to the abomasum where it causes chemical damage and ulceration
- Zinc oxide is used for drenching because it is less acidic. It can also be mixed with supplementary feed, sprayed directly onto pasture, or administered in a slow-release intra-ruminal bolus (Time Capsule, Agri-feeds Ltd, Mount Maunganui, New Zealand). Zinc oxide drench treatments provide some flexibility as they can be used at lower dose rates "every other day", or at a higher dose rate in an emergency when weather conditions produce a rapid rise in spore counts and cows are need urgent protection ("crisis dosing"). Zinc oxide is an insoluble white powder and does not readily suspend in water. Stabilisers such as seaweed-based mixes should be added to allow smaller drench volumes and to prevent it blocking the drench system
- Elemental zinc is used in a novel slow-release intra-ruminal bolus (Face-Guard, Bomac, Auckland, New Zealand). This product does not achieve the very high serum zinc levels observed when the sulphate or oxide salts are used, but additional protection is thought to be provided by some as yet undiscovered local activity within the rumen or intestines[10,11].

Drenching with zinc oxide or administration of zinc bolus treatments provides more reliable protection than adding zinc sulphate to drinking water. Serum zinc levels in cows were shown to increase by only 9% over pre-treatment levels on farms using zinc sulphate in drinking water and only 25% of cows had levels adequate to provide protection[4]. By comparison, cows drenched with zinc oxide increased their serum zinc levels by 27-35% and were mostly protected.

For further information on the difficulties of providing protection with zinc sulphate treatment in drinking water, refer to the article by Neil Cullen in this edition of the Technical Series (page 7-8).

For further information on dose rates and dosing regimens refer to the DairyNZ Farm Facts 3.6 and 3.7.

Zinc bolus treatments

Zinc bolus treatments offer a useful solution for protecting animals that cannot easily be mustered for regular drenching especially during high challenge conditions. This form of treatment is particularly useful for calves and heifer replacements that do not have high water intakes and often have alternative water sources. The main drawback is that the initial treatment lasts only four to six weeks, and bolus administration may need to be repeated if the FE season is prolonged.

Two types of zinc bolus treatment are available (zinc oxide and elemental zinc) and both are efficacious provided the instructions are followed regarding dose rates relative to liveweight and repeat treatments if challenge continues[10,11,12]. The performance of the two approaches to prevention was compared during a moderate natural challenge in calves weighing on average 187 kg. No clinical FE was seen at all in the trial, and only 1/21 and 3/21 calves treated with zinc oxide and elemental zinc treatments, respectively, developed subclinical disease compared to 17/21 of the untreated control calves[11].

The treatment rate is proportional to the animal's liveweight, and several boluses may be needed. The elemental zinc bolus (Faceguard, Bomac, Auckland New Zealand) is effective for animals up to 250 kg while the zinc oxide bolus (Time Capsule, Agri-feeds Ltd, Mount Maunganui, New Zealand) can be used to treat animals up to 400 kg liveweight. Animals to be treated should be weighed and it is advisable to discuss these treatments and their correct dosage regimens with a veterinarian.

Some care is required when administering bolus treatments as they can cause animals to choke. The specific applicator for the selected bolus treatment must be used. Animals should be held in a way that does not impede swallowing, and the end of the applicator eased carefully over the solid part of the back of the tongue. Once treated, the animal should be observed for the following couple of minutes to ensure the bolus is not regurgitated. If using zinc oxide boluses, care is needed to ensure that the waxy coating is not damaged by the animal's teeth as it is administered or the bolus will break down faster and protection will not be for the full period.

Zinc toxicity

The dose rates used for FE prevention are 20 times higher than normal dietary intake and very close to maximum safety levels[3,13]. Zinc can damage the pancreas, the gland that also produces insulin. Signs of zinc toxicity include loss of appetite, reduced milk production, diarrhoea, and weight loss. While the recommended doses rates for FE management are not enough to produce damage that would alter production, care must be taken when calculating and administering zinc treatments to ensure that cows are not overdosed[17].

Research in sheep has shown that the risk of zinc toxicity increases once liver damage has occurred[14]. Once signs of an FE outbreak are evident, ongoing zinc treatment may be needed if cows continue to have high spore intakes; but particular care is required so as not to overdose in these circumstances, as could occur if "crisis dosing" is the approach taken.

Zinc interactions with copper

Dosing with high levels of zinc inhibits copper uptake from the gut, so prolonged periods of preventative treatment may induce copper deficiency[15,16]. Interestingly this effect is less likely to occur when copper levels are low. Smith et al.[16] treated non-lactating dairy cows with zinc oxide boluses, with and without copper sulphate at 150 mg elemental copper/day. These cows were fed silage with low copper content, so supplementation was predicted to be slightly higher than maintenance needs. Both groups maintained similar serum zinc levels. The absorption of copper was reduced by 50% in the cows receiving zinc. Liver copper levels fell in animals receiving zinc alone, but remained constant where both zinc and copper were supplemented.

Given that long term zinc treatments are likely to reduce liver copper, there will need to be a plan to monitor and remedy this situation. Where copper deficiency is an established problem there may be a case to supplement copper at a low level when dosing with zinc; but in most situations in late lactation a six to eight week period of high zinc intake is unlikely to cause serious copper deficiency. The recommended approach is to not supplement copper while dosing with zinc in order to maximise the latter's prophylactic benefits. Liver copper content should then be measured in biopsy samples in late autumn and an appropriate supplementation programme for winter and spring determined, so that copper levels are restored by late gestation and early lactation when the animal's requirement for copper is greatest[17].

High levels of copper may interfere with the protective effects of zinc treatment. Free copper ions in the liver can catalyse super-oxide formation, working antagonistically to the zinc and increasing the damage from the sporidesmin[9]. Following a severe outbreak of FE in a herd, despite adequate delivery of zinc sulphate through a dispenser system, cows were found to have received 2 g of copper sulphate/day up until zinc dosing started. Copper levels in liver samples from affected cows, taken three months after copper dosing had ceased, indicated that copper levels were still marginally toxic[18]. Promoters of organic copper chelates often state that their products can be used during the FE season because they will not compete with zinc for absorption through the gastrointestinal tract. While this is likely to be true, these products may still increase the level of free copper ions in the liver which may potentiate the effect of sporidesmin[19].

There have been anecdotal reports of FE outbreaks despite zinc dosing where palm kernel expeller (PKE) was fed at rates of up

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to 50% of cows' diets during drought conditions. The copper content of PKE is 20-30 ppm of elemental copper which is two to three times higher than the usual recommendation of total dietary copper content of 10 ppm for dairy cattle where the molybdenum and sulphur content of the ration is low[17]. When PKE is fed for long periods as a high proportion of the diet, copper accumulates in the liver[20]. While definitive diagnoses could not be established in the reported outbreaks, it is possible that the high copper intakes reduced zinc absorption, or that pre-existing high liver copper levels over-rode the anti-oxidant activity of the zinc. Until these mechanisms are better understood, prudence suggests that additional care should be taken where PKE is being fed at more than 30% of the diet, or where there is a high probability that copper levels in the liver are high due to PKE being fed for a prolonged period prior to zinc supplementation. In these situations, management tools other than zinc supplementation should be considered.

Breeding for facial eczema tolerance

The New Zealand sheep industry has successfully implemented breeding programmes for FE tolerance, and this is also a heritable trait in cattle. Based on a series of experimental studies of dairy cattle exposed to sporidesmin, either naturally or experimentally by drenching with a standardised dose, heritability estimates of the indices that reflect the extent of subsequent liver damage (i.e. the liver enzymes GGT and GDH) were 0.34 and 0.30[21]. While these heritability estimates are lower than the 0.45 reported for sheep, considerable progress could also be made to breed dairy cattle with FE tolerance if appropriate selection procedures were developed. Research into breeding FE tolerant cattle is currently being funded by DairyNZ.

Within a herd there can be wide variation in the expression of clinical signs following exposure to the toxin. Some of this variation is because the dose received is influenced by an individual animal's grazing behaviour, but there is also variation between animals in the extent to which they can deal with phytoporphyrin. For example, in one study of lambs dosed with standardised amounts of sporidesmin, a proportion with severely damaged livers did not show skin lesions. This may reflect differences in digestive processes which influence the amount of phytoporphyrin released, differences in absorption through the intestinal wall, or differences in the way the phytoporphyrin is detoxified once absorbed[22,23]. In sheep, a candidate gene involved in maintaining the integrity of bile duct cells has been identified and demonstrated to influence sensitivity to the effects of sporidesmin[22].

Estimation of breeding values for FE tolerance based on daughter responses to natural exposure takes time, and sires are generally old before accurate proofs are available. A DNA marker approach could circumvent this problem, but one bull breeding company meanwhile is taking a similar approach to the sheep industry. Based on a standardised challenge with sporidesmin, several young bulls have been identified as "FE tolerant" and daughters of these bulls can be expected to be 20% more tolerant to FE than the general population (P Beatson, CRVAmbreed, *pers comm*).

While these gains in tolerance provide protection, they will not completely eliminate the problem because it takes some years to increase the frequency of these genes in the population. This means that prevention programmes will still be required, but the combined approach should result in better outcomes.

Concluding statement

Clearly facial eczema management remains problematic and many cows each year continue to be affected by this disease. While there is a range of tools available, none provide a foolproof solution, and many have downsides. In high risk areas, effective management requires an integrated approach combining methods that reduce spore intakes, such as pasture-spraying programmes and providing alternative feed or crops for grazing, with methods that protect cows from toxic damage, such as breeding for FE tolerance and dosing with zinc.

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Zinc protection – using zinc sulphate by water trough treatment

Neil Cullen, AgResearch Ruakura, Hamilton

Two methods have traditionally been used to administer zinc sulphate to dairy cattle to aid control of facial eczema (FE). Zinc sulphate has a lower safety margin than zinc oxide so cannot be safely used as a drench as it can result in lesions of the abomasum and severe pancreatic damage[1]. Adding to the water supply or direct to the water trough are the only available options for administering it.

Zinc is not stored in the body so supply has to be continuous. Administration of zinc sulphate needs to be done in such a way that its concentration in water is relatively constant at a level that depends on many things, including cow liveweight and water consumption levels. Simply putting handfuls of zinc sulphate directly into troughs is unreliable – the first animals drinking may be over-dosed, while those coming later to drink may not get enough.

The first reliable method of zinc sulphate administration to be developed was dispensers (e.g. Peta Zinc Dispenser; PETA Enterprises, Hamilton) which have to be topped up manually each time cows are given access to a new trough. More commonly today, a concentrated solution of zinc sulphate is titrated into the farm water supply (isolated from supply to houses and cowsheds) using an in-line water dispenser such as a Dosatron (Dosatron NZ, Bell-Booth Ltd, Palmerston North).

The concentration of zinc sulphate in drinking water should be increased gradually to allow animals to get used to the taste. This should happen before its anticipated need during the FE season. A range of flavour-masking agents including aniseed, vanilla and apple flavourings, can be used to disguise the taste.

Dispensing systems need regular inspection and maintenance, to ensure that correct dosage rates are administered.

Factors influencing the effectiveness of zinc sulphate treatment for dairy cows

Some of the issues around the supply of zinc sulphate in drinking water supply were highlighted in a three year study, funded by MAF Sustainable Farming Fund and supported by DairyNZ, by Dr Chris Morris of AgResearch.

The variable nature of individual cow water intakes: There are many reasons for this including alternative water sources, weather and individual production levels. Cows derive a lot of their water intake from pasture. The dry matter (DM) content of summer and autumn ryegrass/clover pastures can be low – 16-25%, meaning that at least 75% is water[2]. Heavy rainfall will also markedly reduce cow intakes of trough water.

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In the first year of the study, the daily individual intakes of cows drinking treated water with a masking agent added were measured. Some cows were regularly large drinkers, and some were small drinkers, and these differences did not even out over time. Those cows in the bottom quartile for drinking volumes were calculated to drink 14% (8.4 L) less than the average 60 L/day drunk by cows in the study[3].

Water-trough treatment levels need to be calculated based on the intakes of the lowest-volume drinkers, not the average; otherwise the low volume drinkers will be regularly under-dosed with zinc and under-protected against FE. Perhaps not surprisingly in this trial, cows with higher milk yields (by volume) drank more and so would have been more likely to be protected; but low yielders drank less and could have been under-protected.

Responses to trough treatment on-farm: Subsequent work investigated zinc concentrations in trough water and serum zinc levels in cows on commercial dairy herds using in-line dispenser systems. It is recommended that trough water should contain 60-230 mg elemental zinc/L based on typical daily yield figures for late-lactation cows. In autumn-calving herds, these figures must be adjusted because milk production will be higher leading to greater water intakes. A lower concentration of zinc in the drinking water may be sufficient.

During the on-farm monitoring carried out by AgResearch Ruakura, blood samples were taken from cows in a dozen herds in December before treatment began, and the same cows were re-sampled in February/March[4]. The majority of herds treated with zinc sulphate were under-protected – there was only a minimal increase in blood zinc concentrations in the second sample, compared with pre-Christmas levels. Blood zinc levels in treated animals averaged only 11.4 µmol/L in the first year and 13.1 µmol/L in the second, compared with the recommended range of 18 – 34 µmol/L[5].

The conclusion that cows were under-protected was further demonstrated in another trial where serum levels of gamma-glutamyl transferase (GGT), the liver enzyme that best indicates liver and bile duct damage from FE, were measured in all cows in herds with clinical cases of FE. More than half of the 70 herds (and 15,000 cows) were using zinc sulphate as their method of protection, and in these supposedly-protected herds, more than 30% of cows (and in some cases 90%) had elevated levels of GGT indicating wide-spread subclinical FE had developed[6].

These results suggest that using zinc sulphate in water troughs is not a reliable method of preventing liver damage, especially at times of high exposure to FE spores. One improvement in this management system would be to check zinc concentrations in drinking water regularly to monitor if they achieve the required levels. Test kits (Zn Merckoquant®)[7] are available from Merck Chemicals. These use an easy visual test-strip that changes colour according to the zinc concentration. It provides a reading in the range of 0-50 mg/L, which is less than the required levels. It is recommended when using these strips that an initial sample of trough water is diluted 1:4 before making the reading, and the test result is multiplied by five to determine the actual concentration.

Conclusion

The overall conclusion from this work is that zinc sulphate dosing of drinking water does not provide full protection against FE. Other control measures should be considered when the risk is high. Monitoring zinc concentrations in water troughs, and sampling a subset of cows to test blood zinc status will provide a better assessment of whether cows are protected or not, and may improve the effectiveness of control programmes based on adding zinc sulphate to drinking water.

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Lame cows – are we identifying all of them quickly enough?

Richard Laven, IVABS, Massey University

A major barrier to tackling lameness is farmers' perceptions of the problem. Overseas studies in housed cows[1] where there are many good opportunities to observe cows, have shown that farmers generally identify only one third of cows assessed as lame by mobility or locomotion scoring. Mobility scoring by observing stride length and evenness of weight bearing across the four limbs as the cow walks over a flat solid surface (see Table 1 for an example) is a husbandry tool that can improve lameness detection.

Table 1. An example of a mobility scoring system (DairyCo, UK; http://www.healthyhooves.eu/pdffiles/dairycomobilityscore.pdf)

Score	Signs
0	Even weight bearing and rhythm. Back feet are placed where front feet were
1	Uneven weight bearing or rhythm, or moderately shortened strides. Affected limb not obvious
2	Uneven weight bearing on immediately identifiable limb or obviously shortened strides
3	As for 2, but cannot keep up with the healthy herd

Mobility scoring versus farm observations for lameness detection

Farmers' estimates of lameness prevalence (the number of cows affected at any particular time) were compared with mobility scores on 60 New Zealand farms (33 and 27 in the North and South Islands, respectively). All mobility scoring was undertaken by a single trained observer. North Island farms were scored in October/November and South Island farms in January/February, the periods when lameness is most commonly seen in these regions[2,3].

Based on farmers' estimations, 2.3% of cows were lame (range: 0 - 20%) but mobility scoring identified 8.3% as lame (range: 1 - 36%). Individual estimates of lameness prevalence were correlated to mobility scores (r=0.79), but farmers identified only 28% of cows with reduced mobility. This was not influenced by herd size, and was similar between North and South Islands. While lameness prevalence was much lower than reported overseas (UK: 5.7 and 22.1% for farmer records and scoring, respectively[1]; USA: 7.9 and 24.6% for farmer records and scoring, respectively[4]), it is interesting that New Zealand farmers, similar to their overseas counterparts, identified only about one third of cows with reduced mobility, as lame.

Even though all farms in the survey were pasture-based, allowing regular opportunities to observe cows walking, detection of lameness was poor. Many lame cows will keep up with the herd. Identifying lame cows simply by observing from behind the herd as it comes to the shed does not work. Effective lame cow identification requires active observation and individual assessment. Formal mobility scoring has been shown to be more effective than ad-hoc farm observations in detecting lameness.

Poor lameness detection methods result in delays before treatment

If farm observations consistently underestimate lameness, then treatments may be delayed. To investigate this, cows (n=450) were monitored on a Manawatu farm that, based on farm records for the previous five years, had an average incidence of 15 lameness cases/100 cows/year, which is thought to be average for the region. Cows were mobility scored weekly by an outside observer using the Sprecher system of mobility scoring[5]. This system varies from the system described in Table 1, with animals categorised as "Normal", "Abnormal gait" (arched back when walking, but flat when standing), "Moderately lame" (stands and walks with arched back, short stride on one or more legs) or "Clearly lame" (stands and walks with arched back, favours one or more limbs when walking).

(cont'd p10)

(cont'd from p9)

Farm personnel were not made aware of mobility scores, but were asked to draft any cows considered lame for treatment on a weekly basis. All treatments, other than for footrot, were done by a veterinarian at no cost to the farm.

The delay between each cow's first record of mobility score classified other than "Normal" and her presentation for lameness treatment was calculated. Figure 1 depicts the time-lags for each lameness category. It shows that 25% of cows scored as "Clearly lame" (green line) were treated within a week of receiving that score, and 55% were treated within 21 days; but treatment was delayed more than five weeks for one in five cows in this most severe category. Of cows observed as "Moderately lame" (blue line), 35% had not been treated after 70 days.

This study has shown that there can be lengthy delays between identification of a cow as lame based on mobility scoring and her treatment.

What is the likely benefit of earlier treatment?

Lameness imposes economic cost through lower milk production, increased rate of culling, effects on fertility, and costs of treatment, but data on the economic impact of lameness under New Zealand conditions are sparse. Tranter and Morris[6] estimated that direct production losses could amount to 240 L milk/clinical lameness event. Based on DairyNZ Healthy Hoof cost models, lameness on the study farm costs an estimated $425/lame cow (Table 2).

While the values have not been quantified, earlier identification and treatment of lame cows should reduce the impact of lameness on both welfare and productivity, providing financial benefits. Delays before treatment can increase lesion severity making them more difficult to treat – white line infections go deeper, more sole becomes under-run, and there is increased risk that infection will enter the joint, necessitating claw amputation and culling. Production will be depressed for longer and fertility may be affected.

In the Manawatu study, lame cows took 12 days longer to get pregnant than cows that were not lame[7]. If earlier treatment could halve this delay (i.e. treated cows became pregnant six days earlier), this farm would have had 360 extra cow-days in milk in the following season, a benefit of $47/lame cow (based on $5.50 /kg MS[8]).

The differing time-frames of lameness prevalence between North and South Islands may influence the cost. North Island cows are more likely to be affected during the mating period while in the South Island lameness is more prevalent later in the season so the effect of lameness on herd InCalf rates may be less [2,9].

Regular mobility scoring adds a labour investment of two to four hours for most herds, as it requires a person to score cows during a complete milking. At a cost of around $100 per session, the savings made from earlier detection of lame cows for treatment provides a financial benefit, and the suffering of those cows that are lame but not being treated is reduced.

Figure 1. Delays between observation of an abnormal mobility score (green: "Clearly lame"; blue: "Moderately lame"; yellow: "Abnormal gait"; red: "Normal" and drafting for treatment based on farm staff assessment of lameness.

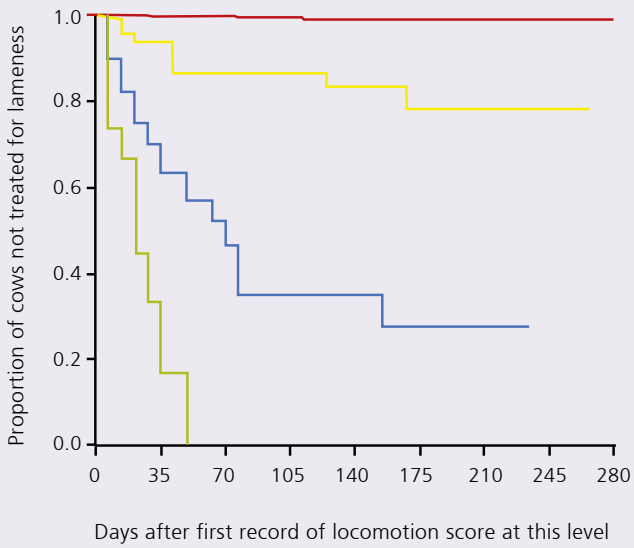

Table 2. Estimated costs of lameness (cost/lame cow) on the Manawatu study farm (based on the DairyNZ Healthy Hoof cost calculator with 1 kg MS valued at $5.50).

Potential lost revenue	Cost / lame cow ($)
Lost milksolids production	183.00
Cost of treatment – farm staff time	12.30
Cost of treatment – veterinary costs	79.00
Wastage – cows culled for lameness	36.00
Wastage – discarded milk	2.00
Impact on reproductive performance – decrease in 6 week InCalf rate	14.00
Impact on reproductive performance – empty rate	100.00
Total/lame cow	425.00
Total for herd (56 lame cows)	23826

In large New Zealand herds, whole herd mobility scoring may not be practicable on a frequent basis, but scoring during strategic periods such as before mating (especially in the North Island) and after Christmas (in the South Island) when the prevalence of lameness generally increases, could identify lameness earlier and minimise its impact.

Conclusion

Farm observations generally detect about one third of cows with impaired mobility. Mobility scoring detected lame cows earlier than farmer observations. Significant delays can occur between the onset of reduced mobility and provision of treatment, even for severely affected cows. These delays increase the economic impact of lameness because lesions become more difficult to treat, while reduced mobility also affects welfare and productivity. Regular and strategic use of formal mobility scoring will benefit both the farm's financial position and improve animal welfare.

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Effective control of parasitic nematodes – an issue for dairy farmers?

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Summary

- Nematode parasites have a significant effect on productivity and profitability in dairy cattle
- A limited number of drench families are available, all of which have been in use for decades
- Anthelmintic resistance is common in New Zealand, and has recently been found in the highly pathogenic species *Ostertagia ostertagi*
- Significant variability occurs in the effectiveness of drench products currently on the market.

Unlike most meat and fibre operations, which rely on the productivity of young stock, the dairy sector relies on milk production from mature animals. This has led to a different level of interest between the sectors regarding the control of internal parasites, as these have a much more pronounced impact on growing animals. However, effective parasite control is necessary to maximise profitability – regardless of sector. The question is: what is 'effective' parasite control?

Impact of parasites

In young stock, parasitism results in lower live-weight[1], which contributes to lower milk production in the first lactation[2], delayed onset of puberty[3] and a reduction in the success of inseminations[4]. Research from Europe in adult cattle suggests that production benefits can be gained by treating adult dairy cows with anthelmintics[5], apparently because treated animals eat more than untreated cows. The validity of extrapolating this conclusion to New Zealand dairy herds remains uncertain, with varied results obtained on different farms (W. Pomroy, presented at the 2011 New Zealand Society for Parasitology Conference).

A significant component of the cost of parasitism to the farmer, as opposed to the livestock, is the purchase of drench, the commonest form of treatment. The question is: are these drenches delivering the best economic return for the investment?

Anthelmintic drenches

While two new classes of anthelmintic have recently been launched for use in sheep, these are not currently available for use in cattle. This means that dairy farmers are restricted to using one of the three drench types which have been in widespread use for decades[6]. A small number of pour-on or injectable products are registered for use in lactating cows in New Zealand, all from the macrocyclic lactone (ML) family.

For non-lactating stock there is a vast array of products available, and these may differ in their performance far more than most farmers realise. There are not only oral, injectable and pour-on variations but also single, dual and triple combinations of the various classes. Very recent New Zealand studies indicate big differences in performance between some of these products, which means that while farmers 'expect' all drenches to be much the same, this is often far from the truth (D.M. Leathwick, unpublished data).

Anthelmintic resistance

On a global scale, drench resistance in cattle is regarded as relatively uncommon, however, it appears that this may be a misconception due to the fact that few studies have been conducted. In general, when people start to investigate the efficacy of drenches in cattle, they find resistance. Resistance has now been confirmed in all the major nematode species that infect cattle, and to all the main classes of drenches which are used to control them[7]. Almost all of these cases have been reported since the turn of the century.

In New Zealand, resistance to the ML class of drenches is very common[8]. To an extent, this has gone unnoticed by farmers because the parasite most often involved (*Cooperia oncophora*) is principally a parasite of young cattle (<15 months old) and is not very pathogenic unless numbers build up to high levels[9]. However, failing to control this parasite can still result in reduced live-weight at 12 months of age, by 4-8 kg (D.M. Leathwick, unpublished data).

In contrast, resistance to other nematode species in New Zealand is still relatively rare, but that is starting to change. The first New Zealand case of ML-resistance in *Ostertagia ostertagi* has just been found in a heifer grazing operation in the lower North Island (D.M. Leathwick, unpublished data). Resistance in this highly pathogenic parasite of cattle[10] will be a very different problem to that involving *C. oncophora*. Dairy farmers need to start paying attention to resistance management to avoid a serious production issue in the future, as has been seen in sheep[11].

Are drenches effective?

A recent study in New Zealand dairy and beef herds indicates quite large differences in performance between different routes of administration, with oral drenching performing significantly better than pour-on and injectable products (D.M. Leathwick, unpublished data). Similar observations from work carried out in the USA were reported at a recent international conference (World Association for the Advancement of Veterinary Parasitology, Buenos Aires 2011).

The varied efficacy of pour-on drenches appears to be due to how much of the drug actually gets across the skin. Indeed, studies have shown that animals ingest significant quantities of drug by licking either themselves or others[12]. What was more of a surprise was the poor performance of injectable products. This work is ongoing and there is much yet to be understood. However, a clear take-home message for farmers is that to kill worms cost-effectively, while also slowing the development of resistance, they should use oral drenches wherever feasible.

Results from a number of studies demonstrate that drenches containing a combination of active families is superior in terms of efficacy against resistant worms and in delaying the development of a resistance problem[13]. This was supported by the results of a national survey of resistance in young sheep[14], which found relatively little resistance to the combination treatment used. However, it must be stressed that using a combination does not guarantee the treatment will be effective, as worms may be present which are resistant to each active family. For that reason, determining resistance status should be an integral part of any parasite-management plan.

Between the variability in drench efficacy and the impact of resistance on productivity, it seems likely that many dairy farmers are spending significant sums of money on drenches which are either a) never going to provide the level of protection required or b) are less than fully effective due to the presence of resistant worms.

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BVD – What does it do and what can you do about it?

By Andrew Weir, Veterinarian, Eltham Vet Services

Bovine viral diarrhoea (BVD) virus has been recognised for a long time[1] but in the last 10 to 20 years understanding of the disease has advanced considerably and better tools to control it have been developed.

Norway and Finland have successfully eradicated BVD, and at the time of writing Sweden and Denmark are nearly there. Most other countries in Western Europe are also working towards eradication.

New Zealand lags behind these countries when it comes to BVD control but DairyNZ has been funding research into BVD in New Zealand which is clarifying the way that BVD behaves here.

BVD effects

The current best estimate is that BVD costs the dairy industry about $135 million per season (based on an $8 payout and about 15% of herds actively infected). This accounts for losses in milk production, delayed calving to conception, abortions, and the reduced survival of persistently infected (PI) animals[2]. Most cases of BVD in dairy herds present either as sick calves, malformed new-born calves (a fatal form of BVD called mucosal disease) or reproductive losses in adults but there are many different ways that BVD can cause problems.

Persistent infection (PI)

The most important aspect of BVD infection occurs in the pregnant animal when it infects the foetus before the immune system develops at between 100 and 125 days of gestation (see diagram). Then it will either kill it (leading to abortion) or trick the foetal immune system into thinking that BVD is part of its own body so the animal will never fight it off.

Foetuses infected during the first 125 days of gestation are born persistently infected (PI) and shed large amounts of virus throughout their lives. They become the main source of infection in the herd allowing the virus to spread and be maintained. Animals that are PI often do not thrive and on average they grow 18% slower, have a 23% higher risk of mastitis, a 22% higher risk of severe illness or sudden death, 23% lower milk production, and a 17% higher mortality rate[3], though the mortality rate can be much higher.

Mucosal disease is a severe, invariably fatal, superinfection of PI animals with an aggressive form of BVD[4]. In the absence of control, 0.5% to 2% of animals in a region are commonly estimated to be PI[5] although there can be a much higher proportion on individual farms after an outbreak.

BVD control depends on keeping PI out of the herd and preventing PI formation by protecting pregnant cows or finding PI early by testing all replacement calves and culling any PI before they can infect pregnant cows and create another generation of PI.

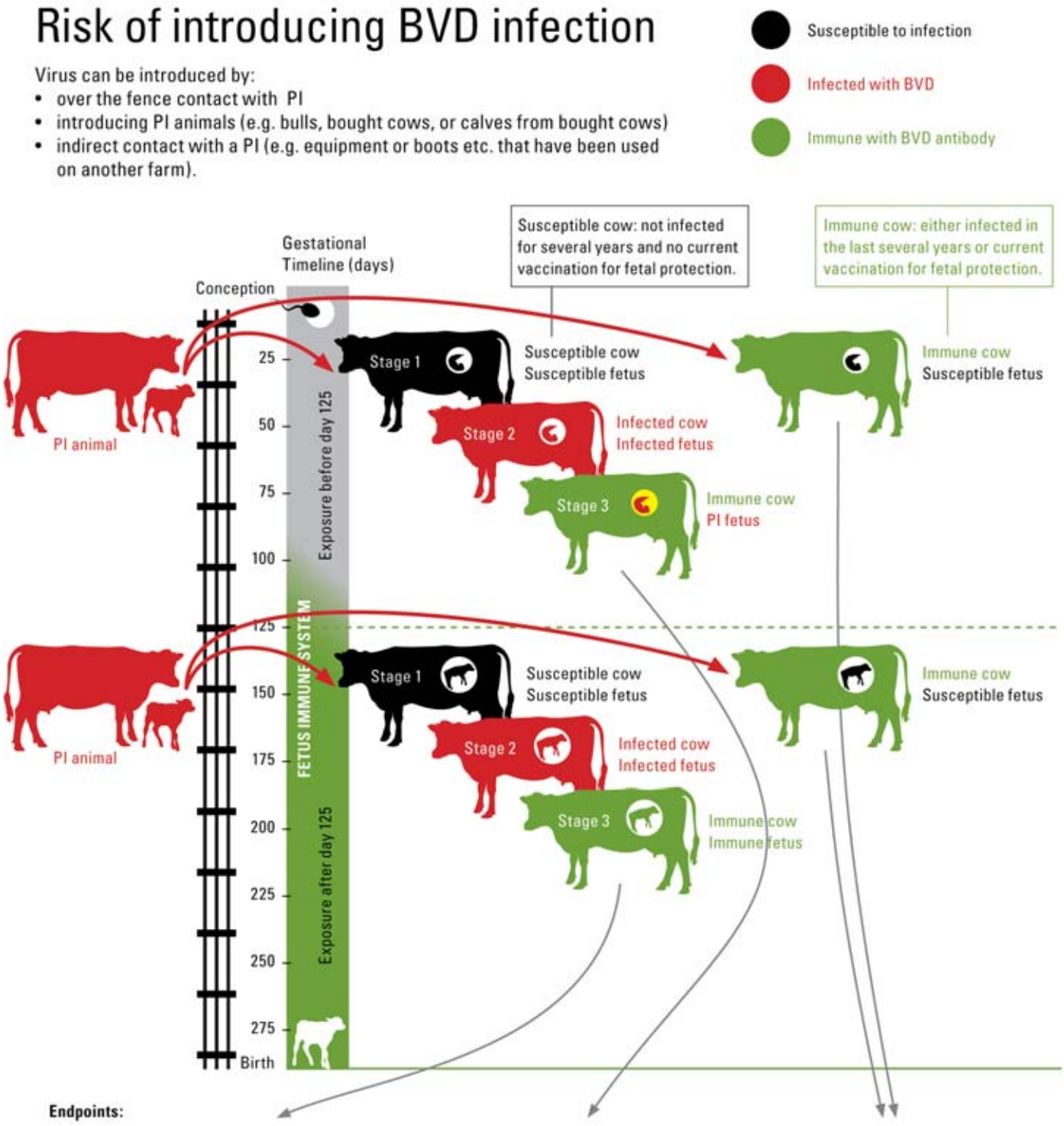
(cont'd p16)

(cont'd from p15)

Risk of introducing BVD infection

Virus can be introduced by:

- over the fence contact with PI
- introducing PI animals (e.g. bulls, bought cows, or calves from bought cows)
- indirect contact with a PI (e.g. equipment or boots etc. that have been used on another farm).



From late gestation infection

IMMUNE COW
IMMUNE CALF (not PI)

Calf is born immune but may be deformed, small and weak, or have poor fertility as a heifer due to fetal BVD infection.

Action: If small and weak, or deformed then cull.

Consider a BVD control programme for the herd.

From early gestation infection

IMMUNE COW
INFECTED CALF (PI)

Calf is constantly a risk to all in-contact animals.
Often poor doing and unhealthy.

Action: Test calves to find these and cull them ASAP.

Consider a BVD control programme for the herd.

Exposure of immune cow at any time

IMMUNE COW
SUSCEPTIBLE CALF (not PI)

Healthy calf.
Will get temporary immunity from colostrum.

Action: Keep away from PIs and consider vaccination.

Diagram provided by: BVD STEERING COMMITTEE

Prepared by MSD Animal Health

Cows

In addition to generating new PI, BVD infection of cows can reduce conception rates by directly affecting ovaries[6], kill the foetus leading to abortion, or cause malformations such as brain and bone defects, and growth retardation[7]. Infection late in pregnancy may be inapparent, may result in the birth of small weak calves, or may compromise future fertility[8]. Infection with BVD virus has also been associated with higher rates of mastitis, ketosis, and other diseases[9-12]. In a New Zealand study, the costs due to other diseases like mastitis doubled for cows that were transiently (acutely) infected during the study (unpublished data).

The largest cost of BVD in the milking herd is through its impact on milk production. Several overseas studies have shown a decline in milk production and/or an increase in somatic cell count due to BVD infection[13-15]. A New Zealand study found herds with current or recent exposure to BVD (high bulk tank antibody) had production that was 5% lower than the other herds in the study[2].

Calves

Calves are particularly susceptible to the effects of BVD. The virus actively suppresses the immune system making other infections, such as scours due to Rotavirus, more likely and more severe[16-19]. Infection has also been associated with a higher risk of calf death and lower weaning weights[20].

A large study of feedlot calves found that calves in the same pen or a pen adjacent to a PI had a 20% lower average daily gain despite vaccination[21]. This may be due to the metabolic cost (energy and protein etc.) of maintaining high levels of antibodies and other aspects of fighting off the continuous challenge as well as an increased death rate and increased rate of respiratory disease.

Although calves in New Zealand are typically reared on pasture after weaning, they spend a lot of time interacting as a group so the risks would be similar. Given that nutrition of young stock is also often limited on pasture, lower growth rates could be expected in New Zealand calves exposed to the virus by the presence of a PI calf.

Bulls

Infection can also have a serious effect on bulls. While PI bulls can have acceptable semen quality, they often have lower fertility even when the semen quality appears to be acceptable[22]. The most important problem with PI bulls is that they are an efficient source of infection for any cows they make contact with. Transient infection of bulls can also affect semen quality[23, 24,] and infected bulls are a potential source of infection[23, 25]. All bulls should be tested for virus to make sure they are not PI, and vaccinated (twice initially) ideally 2 to 3 months before use to minimise the risk that they suffer impaired fertility due to BVD or introduce infection into the herd.

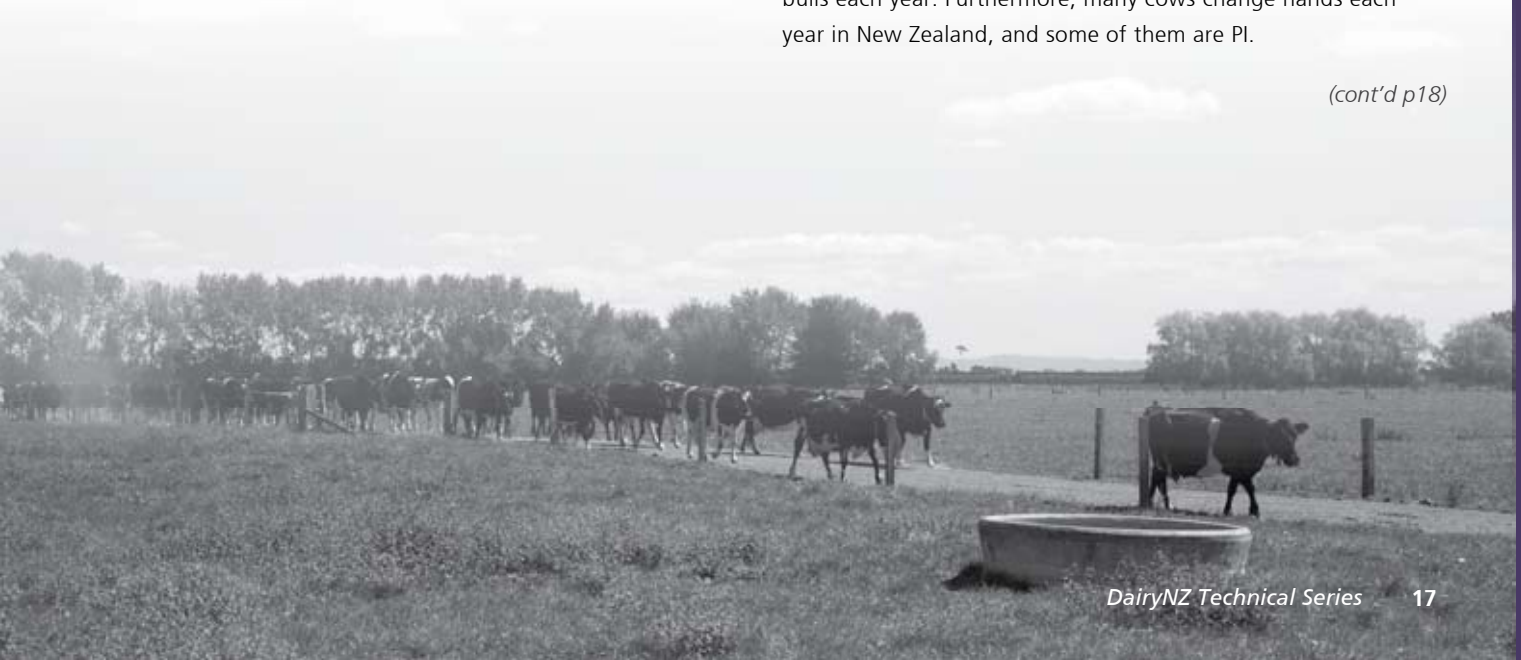
Biosecurity – how BVD spreads and what you can do about it

Persistently infected cattle are the main source of infection and are usually very efficient at infecting in-contact cattle[5]. Indoors the virus can spread over short distances (e.g. 10 m) through the air[26, 27]. It is unclear how important spread through the air is in New Zealand conditions. Although transiently infected cows shed virus for a few days[28], several studies have failed to demonstrate spread of BVD virus from transiently infected animals despite close contact[29, 30]. It is, therefore, unlikely that transiently infected animals contribute much to the spread.

Incoming stock

Movement of PI cattle is the most important route for introducing BVD into a herd. Bulls are the most common class of stock introduced into herds and test results from animal health laboratories in New Zealand indicate that between 0.5% and 1% of bulls tested are PI (Gribbles and LIC pers. comm.). That means most herds will not introduce PI animals in most years, but in the long run, the risk of bringing in at least one PI bull becomes substantial, especially for larger herds that need more bulls each year. Furthermore, many cows change hands each year in New Zealand, and some of them are PI.

(cont'd p18)



(cont'd from p17)

The only way to avoid introducing BVD through bulls or bought cows is to test them. This should be done before they arrive on the farm, if possible. Not only is there the risk of bringing in PI cows, but any pregnant cows could be carrying a PI foetus. New cows should be calved separately (to avoid mismothering) and their calves kept separate until either bobbied or tested for virus. Alternatively, all replacements can be tested in which case calves from bought cows will not slip through untested due to mismothering, which is very common. Non-PI cows that have been vaccinated before mating with a vaccine that provides foetal protection should not generate new PI, so vaccination of bought cows before mating on the farm of origin may be an alternative way to manage this risk.

Calves

The birth of a PI calf is probably the most common way a herd becomes actively infected, and this is also how infection is maintained on a property. This might happen after contact with a PI over the fence, indirect contact with a PI through equipment or shared facilities during the previous season, or by buying a cow carrying a PI foetus. Heifers and cows that are grazed off the farm during the first four months of pregnancy may run an increased risk of infection while away, thus returning with a PI calf.

These risks can be controlled by testing newborn calves every year, and removing any PI calves before they can spread the infection too far. If PI calves remain on the farm after the start of mating, they can create another generation of PI for the next season so early testing of calves is the key to prevent the disease cycling between seasons. If calves are not tested, they should be managed as a risk to the milking herd due to the possibility of PI calves and contact between the two groups prevented as much as possible.

Neighbours stock

One of the most difficult risks to manage is infection "over the fence" from neighbour's stock. In some regions in New Zealand there is at least a one in five chance that a neighbour has PI milking cows, and it may be as high as a one in two chance of PI of any age[31]. While there may be some risk of air-borne spread over a short distance, in most situations it requires nose-to-nose contact with a PI to introduce infection.

Good boundary fencing that prevents stock breaking through, and outriggers or temporary hotwires that prevent direct contact when animals graze along boundary fences should protect against most of this risk. Vaccinating the herd will prevent any contacts creating PI calves, and testing replacement calves each year will mean any breakdown is caught before the worst effects can occur.

Indirect contact

Indirect contact with a PI through equipment can spread infection[32, 33]. Any body fluids from a PI animal or contaminated equipment should be considered a risk for several (seven to be conservative) days[27] so care should be taken that equipment, clothing and boots recently used on another farm are disinfected and take care with shared facilities. Ensure any people like vets, lay scanners, AI technicians, transport operators, and contractors have good hygiene standards. Don't allow people with contaminated boots, leggings or equipment to come onto the farm, especially when cows are in early gestation. The virus is very fragile, however, so basic hygiene measures such as standard disinfectants and a brush will be sufficient to minimise these risks from equipment and clothing. While this may be a small risk compared to the risks associated with direct contact with a PI, it is worth noting.

Bulk tank testing

Regular bulk tank testing is a useful tool for monitoring BVD status. The PCR test detects the presence of virus in any cows contributing to the vat sample (usually a PI), while the bulk tank antibody test gives an indication of the proportion of the herd that has been infected and how recently infection occurred. Individual cows are usually only tested for virus (described as either a virus or antigen ELISA, or as a pooled PCR test) to screen for a PI, since antibody testing only indicates if they are currently immune.

Herds with a bulk tank antibody level below 0.75 are very unlikely to have any PI milking cows so only the antibody test is needed in future, as long as it remains low. However booking both as part of a package can be convenient and speed the investigation if a breakdown occurs. Once a herd has a history of either low antibody or a negative PCR test, a significant rise in antibody or a positive PCR test in future samples indicates a new introduction and only cows that have arrived since the last test could be PI. Annual bulk tank testing, therefore, can detect a breakdown and elicit action to be taken as well as reducing the cost of finding the PI.

Summary

To avoid introducing BVD, incoming stock need to be tested, contact between pregnant cows and other people's stock should be prevented as much as possible, and either vaccinate the herd or test calves before the start of mating each year to cover most of the rest of the risk. The costs and benefits of these options are currently being investigated to determine which are most cost-effective. This is a complex disease and every farm is unique so the farm vet should be involved in planning control measures on a dairy farm.

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Focus on international research

The following is a brief summary of some key science papers recently published

Reader and others (2011) Effect of mobility score on milk yield and activity in dairy cattle. *Journal of Dairy Science 94:5045-5052.*

Mobility score (MS: scale from 0 to 3), milk yield, treatment for lameness, and cow activity were recorded on 312 cows in a largely confined dairy system in the UK, where cows had only limited access to pasture in summer. Cows with MS 2 and 3 produced 0.7 and 1.6 kg less milk/day, respectively, than cows with MS 1. Six to eight weeks before apparently non-lame cows became MS 2 or 3, their daily milk yield decreased by 0.5 kg and 0.9 kg, respectively. Daily milk yield remained 0.42 kg lower for four weeks after cows with MS 2 had recovered. In addition, once cows were lame they remained lame or became lame again despite treatment.

DairyNZ comment: Results indicate that milk production declines before mobility score increases, and remains low after the cow has recovered. Further research is required in pasture systems to determine the ability to use automatic measurements to detect lameness early.

Penagaricano and Kathib (2011) Association of milk protein genes with fertilisation rate and early embryonic development in Holstein dairy cattle. *Journal of Dairy Research 78: Published online: 13 October 2011 DOI:10.1017/S0022029911000744.*

The association between single nucleotide polymorphisms (SNPs) in four casein and two whey protein genes and reproductive traits were evaluated. There were associations among SNPs in genes involved in whey protein synthesis and fertilisation rate and embryo development, but there were no associations among the casein genes and fertility parameters measured. These effects may explain some of the antagonism between milk yield and fertility.

DairyNZ comment: This is a novel experiment providing some possible direction for future genetic selection priorities. Further work is required to determine its relevance in New Zealand dairy cows. Research to discover gene markers for reproductive function is ongoing with DairyNZ.

Falls and Emanuelson (2011) Fatty acid content, vitamins and selenium in bulk tank milk from organic and conventional Swedish dairy herds during the indoor season. *Journal of Dairy Research 78: 287-292.*

The fatty acid composition and vitamin and mineral composition of milk samples collected from 18 organic and 19 non-organic farms were compared in this Swedish study. Organic farms had slightly more conjugated linoleic acid (a noted anti-carcinogen), omega-3 and omega-6 fatty acids, but there were no differences in other important constituents. Survey data from the farms suggest organic farms offered cows a higher forage diet.

DairyNZ comment: Diet alters the fatty acid composition of milk. There was insufficient information collected in the farm survey to ascertain the cause of the milk composition differences, but it is likely that the effect was diet related and not a result of organic management.

Mantysaari and Mantysaari (2010) Predicting early lactation energy balance in primiparous Red Dairy Cattle using milk and body traits. *Acta Agriculturae Scandinavica Section A – Animal Science 60: 79-87.*

Data from 5,922 weekly records from 146 heifers were used to determine the relationship between easily measured parameters and energy balance. Change in body condition score and live-weight in the weeks following calving explained less than 15% of the variation in energy balance. Fat to protein ratio also explained less than 15% of the variation in energy balance and was only useful in the week immediately after calving. A complex multi-variable model combining fat to protein ratio, BCS change, live-weight, and the interaction between BCS change and live-weight was able to predict energy balance with about 40% accuracy.

DairyNZ comment: The study highlights the difficulty in predicting energy balance from easily measured traits. In particular, the results indicate that fat to protein ratio is not a sufficiently accurate predictor of energy balance to be useful in decision making.