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# IMPROVING FACIAL ECZEMA TOLERANCE IN NEW ZEALAND DAIRY CATTLE

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## SUMMARY

After implementing genomic selection for milk and production traits in dairy cattle, a New Zealand breeding company (CRV Ambreed) has been encouraged to expose a selection of their young bulls to a challenge with the toxin responsible for causing the facial eczema (FE) disease. Resistance to FE is heritable in dairy cattle, and breeding values (BVs) for it are freely available. For young bulls, BVs are based on parent-average predictions, but there is now also the option of phenotyping. This process can identify the more tolerant bulls which can be used by those farmers who suffer major production and cow losses on their farms as a result of this disease. In susceptible animals, FE causes severe liver damage, and may also cause severe and painful skin damage resulting from photosensitisation. This paper describes a protocol which, by removing sources of chlorophyll from the diet, allows animals to develop a sufficient range of liver damage to rank bulls for FE tolerance but does not cause animals to present with photosensitisation.

## INTRODUCTION

Facial Eczema (FE) is caused by the toxic spores of the fungus *Pithomyces chartarum.* The toxin (sporidesmin) is produced by spores which are released during warm, humid weather over mid-summer until late-autumn (December – May). It occurs particularly in ryegrass/white clover pastures in almost all the North Island and the upper South Island of New Zealand (NZ). While FE is of major concern in NZ, the same disease is reported elsewhere in regions of similar latitude (both North and South) and particularly in coastal southern regions of Australia. Di Menna *et al*. (2009) have reviewed FE research in NZ since 1939.

Damage to the liver and bile ducts was identified in early research as the most important pathology, although sporidesmin can affect many organs. As a result of bile duct blockage, a breakdown product of chlorophyll, phylloerythrin, can build up in the body causing sensitivity to sunlight and visible skin damage (hence the common name of FE). Chronic wasting and/or death may occur at the time of damage or months later when the animal is under stress, e.g. parturition.

Early research in sheep showed that gamma-glutamyltransferase (GGT) levels in blood measured 2-3 weeks after dosing with the toxin are positively correlated to post-mortem liver damage scores and to losses in live weight (Towers and Stratton 1978). GGT levels (with a natural log transformation) are now used as the proxy for measuring responses to the toxin, in animals not suspected of having suffered any other liver-damaging process.

The first research in dairy cattle involved the daughters of young progeny test sires subjected to a natural challenge in 1989. This demonstrated that FE was also heritable in dairy cattle, as in sheep, and was subsequently followed by limited evaluation of dairy sires by both direct performance testing and/or progeny testing. However, due to the costs and risks involved (possible losses of potentially very valuable bulls), this work ceased after a short period. Morris *et al.* (2013) provide a review of the genetic work performed in both sheep and cattle since the early 1980s.

Sampling of all cows from clinically-affected dairy herds in the upper North Island from 2004-11 provided GGT and pedigree data on ~15,000 cows from 70 herds. These data plus pedigree information on sires allowed AgResearch to update the heritability estimates and provide FE breeding values (BV) for all animals including sires. The current estimate of the heritability for

$\log_e$ GGT is 0.34 ± 0.02 (Cullen *et al*., 2011).

With the introduction of genomic selection, it is now possible to short-circuit the process of identifying elite sires for milk production instead of using the traditional sire-proving approach which takes a minimum of 5 years. Genomic selection allows the bulls that will progress to progeny-testing to be identified by 6 months of age. The remaining group of bulls with reduced genomic predictions is then challenged with sporidesmin to identify the most resistant bulls amongst this group. Farmers could then compare an FE-tolerant bull with its breeding worth (BW – the NZ national breeding objective for dairy cattle) and make informed decisions about bull selection for breeding. A joint bid by two farmers (Mr and Mrs Burt) and AgResearch to DairyNZ's 'On Farm Innovation Fund' in 2010 was successful in obtaining funds to test the concept of sequential selection of dairy bulls (BW prediction, then FE tests). CRV Ambreed Ltd. (CRV), given their past associations in this work, was approached to be a partner in this process.

A protocol was devised for FE testing in the dairy bull industry, and evaluated here. It allows artificial challenge of young bulls with the FE toxin, in order to rank them for tolerance without causing photosensitisation and its severe effect on animal welfare.

## METHODS

**New Testing Protocol.** Animal ethics approval was obtained from the Ruakura Animal Ethics Committee, and a pilot trial was run at CRV's facility near Hamilton in November 2010. This tested a new protocol with an artificial sporidesmin challenge, using a dose rate of 0.25 mg sporidesmin (in suspension in water) per kg of bull live weight, to be administered by stomach tube. Eleven surplus 2009-born bulls (6 Jersey and 5 Holstein-Friesian) were challenged with sporidesmin in this manner at 16 months of age. The agreed protocol was that bulls would be housed in stalls with 24-hour access to shade and fed solely with silage and supplements. The premise was that, if sources of chlorophyll were removed from the diet, phylloerythrin levels would be kept low and thus not cause animals to develop photosensitivity as a result of liver and bile duct damage. GGT levels were measured on these 11 bulls before dosing, to ensure there was no existing damage. The initial sporidesmin dose rate chosen was not sufficiently high to identify the targeted 10% FE-tolerant bulls, so 5 of the non-responders (one bull was removed because of poor semen production) were re-dosed at a higher rate (0.30 mg/kg) 6 weeks later. GGT levels for all bulls were measured at 14, 21 and 28 or 35 days post-dosing, and then every 14 days for those bulls with levels remaining above 200 iu/l. All animals were inspected daily by CRV's veterinary and farm staff for signs of ill health. Live weight was measured 3-5 days before dosing to prepare individual sporidesmin doses and again when bulls were sampled for GGT at day 21 post-dosing.

This protocol and dose rate was to be the basis of future work with ~50 young bulls per year, starting with 'Year 1' in 2011. Although the initial aim was to test non-elite bulls (on genomic BW), CRV were confident enough with the results of this Pilot Trial that 90% of bulls in Year 1 were elite animals. In 'Year 2', 2012, all bulls were elite animals. The risk was reduced by the ability to pre-screen bulls by predicting parent-average FE BVs.

**Year 1.** In March 2011, after 51 8-month old bulls (2010-born) were pre-selected on the basis of having the most negative (favourable) parent-average BVs, and the ethical exclusion of 6 bulls because of evidence of a previous 'natural' FE challenge, the main trial began. Forty-five young bulls were challenged with a sporidesmin dose of 0.30 mg/kg at the same CRV facility and using the same feeding regimes as previously.

**Year 2.** In March 2012, the programme continued with another 50 8-month old bulls (2011-born), with the same pre-screening procedure and the same protocol. The sporidesmin dose rate was reduced slightly to 0.27 mg/kg to reduce potential risks to animals from unique blood-lines which were included in the group.

## RESULTS AND DISCUSSION

**Pilot Trial.** The normal range for GGT is 0-40 iu/l and levels >5000 iu/l have been observed in FE-affected cows on dairy farms. The maximum GGT level attained in the initial dosing was 853 iu/l with 5/11 bulls having levels greater than 40 iu/l. Morris *et al*. (2009) observed that elevated phylloerythrin levels were only detected when GGT levels were greater than ~600 iu/l and not all animals above this threshold exhibited clinical signs of FE. Only one bull surpassed this threshold so it was not possible to draw any conclusions about the protocol protecting animals from severe FE. For the 5 non-responders redosed at 0.30 mg/kg, the highest GGT level was 188 iu/l. No photosensitisation was observed.

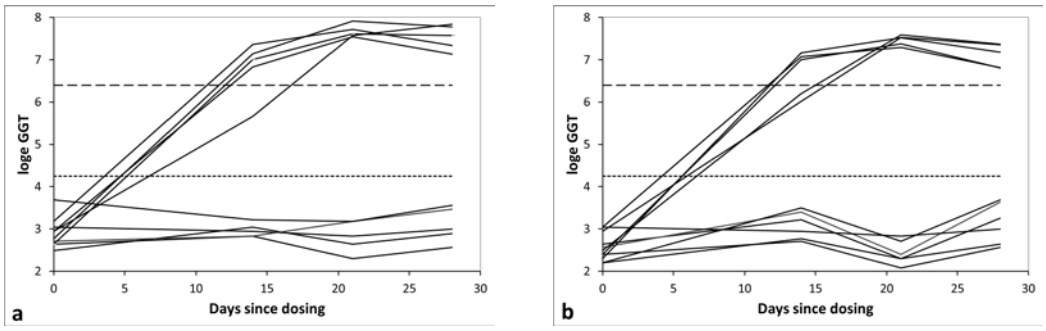


**Figure 1: $\log_e$ GGT levels recorded over the 28 days from dosing for the 5 bulls with the lowest and highest GGT levels at 21 days post-dosing for both the a) 2011 and b) 2012 dosing rounds (dotted line is equivalent to a GGT value of 70 iu/l, a level below which any liver damage may be minimal; and the dashed line to 600 iu/l – the level above which clinical signs of FE due to photosensitisation might be observed).**

**Year 1.** 38 of 45 of bulls had elevated GGT levels (maximum 2736 iu/l). Figure 1a shows the GGT time series for each of 5 bulls having the lowest and highest GGT levels at 21 days post-dosing. The trends for the remaining 31 bulls with intermediate values for GGT are not shown but follow similarly; the repeatability for $\log_e$ GGT in serially sampled animals is 0.86 ± 0.004 (Cullen *et al*. 2011). The repeatability over days 14, 21 and 28 for these data were 0.85. The high GGT levels were in the range where it was expected that some bulls would show clinical FE signs due to photosensitisation (but none observed provided support for the chosen protocol).

**Year 2.** The second year of dosing 8-month old bulls showed similar results (Figure 1b) to Year 1; 43 of 50 bulls had elevated GGT levels. No photosensitisation was observed.

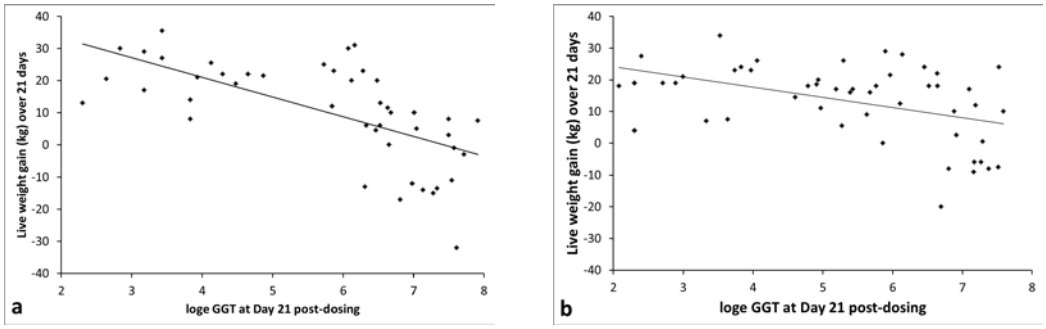


**Figure 2: Live weight gain plotted against $\log_e$ GGT levels at 21 days post-dosing for the a) 2011 and b) 2012 dosing rounds.**

Although no clinical signs of FE were observed in the 106 bulls challenged over the 3 batches of animals, it was evident that the liver damage, as measured by increased GGT levels did have a dramatic effect on live weight. This is probably due to the suppression of appetite through the bull feeling unwell. This was more evident in 2011 with the slightly higher dose rate. The regression estimates of live weight gain/loss on $\log_e$ GGT at 21 days post-dosing were -6.1 ($P < 0.0001$) and -3.2 ($P < 0.01$) kg/$\log_e$ GGT unit for 2011 and 2012 respectively.

The data and pedigrees previously collected from the on-farm sampling of dairy cows has allowed the calculation of reliable (> 60%) breeding values for $\log_e$ GGT for approximately 200 industry sires. Incorporating the results from this work into the data has provided the comparative ranking of sires from this study (reliabilities ranging from 36 to 49%) to be compared to other sires which have been used widely in industry. CRV are now marketing teams of both Jersey and Holstein-Friesian FE-Tolerant bulls to industry. The FE BVs for the 13 bulls in CRV's 2013 FE team (born in 2009-11) along with the ~200 bulls with FE reliabilities greater than 60% have been plotted against their birth-year in Figure 3. The team average BV $\log_e$ GGT of -0.24 is superior to 87% of the widely-used industry sires born since the 1980s.

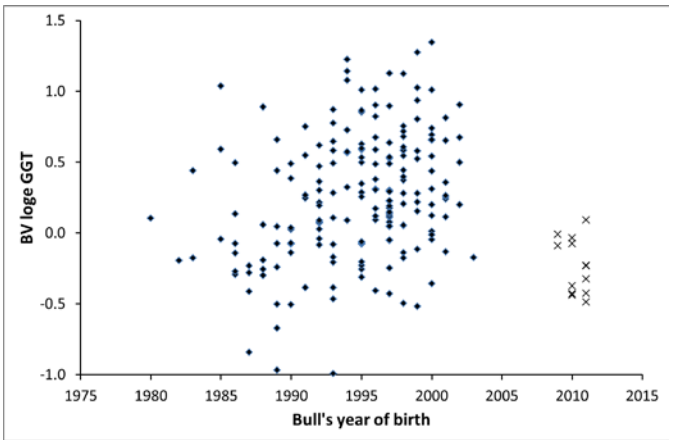


**Figure 3: Breeding values for $\log_e$ GGT for industry sires (reliability > 0.60) (symbol = ♦) and bulls in CRV Ambreed's 2013 FE Team (symbol = ×).**

This method of genetic improvement of a disease trait like FE is long and slow, as demonstrated by the sheep industry progress with FE over 25 years. It is anticipated that genomic selection will play a role to expedite more rapid gains in the future.

**ACKNOWLEDGEMENTS**

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