

## Lifetime survival of Jersey-sired cows following natural challenge with facial eczema during first lactation

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**Abstract** During a serious natural outbreak of facial eczema (FE) in March–May 1989 in the Northland, Auckland, and Taranaki regions of New Zealand, over 1500 Jersey-sired 1st-lactation heifers in 60 spring-calving herds were blood sampled between 24 April and the end of May. The objective was to monitor their response to the FE challenge, as measured by serum activity of the enzyme gamma-glutamyltransferase (GGT); 43 (72%) of the herds contained heifers that had been exposed. In these herds, compared with the remaining (“non-exposed”) herds, the cumulative percentages of heifers (still present for the blood sample) which were culled or had died were not significantly affected by type of herd. In the exposed herds only, the difference in  $\log_e$  GGT between cows culled or dead by the end of the 1st lactation and those that survived was  $0.52 \pm 0.23 \log_e$  i.u. litre<sup>-1</sup> ( $P < 0.05$ ). The survival of animals to the end of the 2nd lactation in exposed herds was 5.9% lower in heifers with serum GGT  $> 100$  i.u. litre<sup>-1</sup> than in those with lower GGT levels ( $P = 0.05$ ); the survival difference in heifers with serum GGT above or below a threshold of 200 i.u. litre<sup>-1</sup> was 9.1%

( $P < 0.01$ ). From all animals blood sampled in the exposed herds (daughters from 65 sire groups), the heritabilities of survival past the end of the 1st, 2nd, 3rd, and 6th lactations following the FE field challenge were  $0.01 \pm 0.03$ ,  $0.02 \pm 0.05$ ,  $0.02 \pm 0.05$ , and  $0.00 \pm 0.05$ , respectively. The heritability estimate for  $\log_e$  GGT in the FE challenge season was  $0.32 \pm 0.10$ . This study has shown significant negative associations between the GGT level after an FE challenge and cow survival.

**Keywords** facial eczema; dairy cows; liver enzyme; survival; heritability; GGT

### INTRODUCTION

Facial eczema (FE) is a disease of grazing ruminants and is common in the autumn in northern New Zealand. It occurs as a result of ingesting *Pithomyces chartarum* fungal spores containing the toxic compound, sporidesmin. In susceptible cattle and sheep, the toxin causes liver injury, resulting in sensitivity to sunlight and loss of production (Towers & Smith 1978). The severity of liver injury can be determined in the live animal from a blood sample, by measuring serum activity of gamma-glutamyltransferase (GGT) (Towers & Stratton 1978).

Resistance or susceptibility to FE is heritable ( $0.42 \pm 0.09$ ) in sheep (Campbell et al. 1981) and in cattle ( $0.29 \pm 0.15$  in Holstein-Friesians and  $0.77 \pm 0.13$  in Jerseys; Morris et al. 1998). The trait will respond to directional selection in sheep (Morris et al. 1995), and in cattle (Morris et al. 1991a, 1998).

The present study was set up in 1989 to monitor the effects of a natural FE challenge on dairy cow survival. A series of 60 herds was monitored, using 1986-born animals in their 1st lactation (the 1988/89 lactation season). Herds were selected from the Livestock Improvement Corporation's (LIC) "1985" Jersey Sire-Proving-Scheme (SPS), so that large numbers of young-sire groups were repre-

sented, with an average of about 20 daughters per sire being sampled. The aim of the study was to monitor genetic and phenotypic effects of FE on cow survival, and estimate genetic effects on log-transformed GGT following a natural FE challenge. The summary of long-time survival is now presented, including survival records up to a possible 14 years of age.

## MATERIALS AND METHODS

### Herd and animal sampling

The survey was undertaken in the late autumn of 1989 to evaluate the FE susceptibility of daughters by Jersey sires represented in herds in northern New Zealand. Three FE-affected regions of New Zealand were chosen, Northland, Auckland, and Taranaki. With assistance from LIC, all 65 Jersey SPS herds within these regions (50% of the national total of "1985" Jersey SPS herds) were identified and contacted and 60 were visited; 14, 25, and 21 by region, respectively. All cows by the Jersey SPS sires in these herds were sampled between 24 April and the end of May 1989. These cows were born in late winter/spring 1986, and were in their 1st lactation in the 1988/89 season when the FE outbreak occurred. The outbreak was slightly later in the season than usual, with clinical cases generally not observed until March in that year but persisting through until at least June (Ministry of Agriculture and Fisheries/Agriquality (now Gribbles Group Limited) Ruakura Animal Health Laboratory unpubl. "Optigrow trend" data; A. J. Fraser pers. comm. 2001). The animals in the study were daughters of 76 Jersey SPS bulls used by artificial insemination (the 1985 mating design), with semen from each bull used across many herds. Most of the 76 were young bulls to be progeny tested, but 8 "link" sires were included, to provide reference-sire connections with previous years of LIC progeny-test data.

All Jersey-sired 2-year-olds still present in the 60 survey herds were blood sampled from the tail into 10 ml vacutainer tubes. Herds were sampled at the rate of up to 5 per day, and all blood samples were sent to Ruakura for serum GGT assay using standard procedures at 37°C. The farmers were not informed of the GGT results.

### Data analyses

GGT results were obtained from 1523 Jersey-sired 2-year-olds in the 60 herds, an average of 25.4

animals per herd (Morris et al. 1990). Herds ( $n = 17$ ) found to have no more than one animal showing elevated GGT ( $>30$  i.u. litre<sup>-1</sup>) were said to have received no (effective) challenge, and had no estimable sire variance ("non-exposed" herds). The remaining herds ( $n = 43$ ) experienced some degree of natural challenge, according to GGT results, and the majority of analyses were restricted to between-cow analyses of animals in these herds ("exposed" herds).

Figures extracted from 59 of the 60 herds show the following statistics (the single missing "herd" being a composite herd from other sources): there were 3830 daughters sired by the Jersey SPS bulls and born in 1986 in these herds; 2534 of these were reared, and 2081 calved as 2-year-olds in 1988. A total of 1435 or 69.0% of those calving were blood sampled. If there had been a bias before blood sampling in 2-year-olds towards more culling of FE-affected animals, a lower percentage of animals would have been blood sampled in the "exposed" herds, but there was a slight tendency in the opposite direction (percentages blood sampled in the exposed versus non-exposed herds being 70.3 and 65.2%, respectively;  $P < 0.05$ ).

In our earlier published analyses of these herds (Morris et al. 1990, 1991b), the temporary tag number within the herd ("herd-test number") was used for identification and for locating corresponding pedigree data. For the present study of cows' lifetime survival, where individual cows had to be traced potentially through a number of different herds, the nationally unique "animal key" was used, with approval of the Dairy Herd Improvement Tribunal and assistance from LIC. A total of 1480 of the original 1523 records (animals with herd-test number) were matched with the animal key, and the original heritability estimate for log<sub>e</sub> GGT (Morris et al. 1990) was re-calculated for a revised data set ( $n = 1480$  animals, before then selecting on sires' progeny group size, and FE status of herd). Cow lifetime survival (age at death or at final disposal) was calculated for the 1480 animals from the date of birth to either the date of death or (if no date of death was entered) to the "out date" in the last herd where the animal was recorded. The "out date" was the last known date for which an animal was recorded as physically present in the last herd; this was preferred to the last milk herd-test date (known as the "end date") because of the possibility of a dry period or carryover period after the last end date. The lifetime file was extracted by LIC staff on 22 June 2000, so the 1986-born

cows under study had a potential lifetime of up to 14 years (the beginning of the 13th lactation).

Cow lifetime survival data were pooled into age groups, counting from April/May 1989 (the time of blood sampling for FE, near the end of their 1st lactation), i.e., those surviving to <3.00 years (end of 1st lactation), to <4.00 years of age, etc., and these categorical data were analysed by chi-square, using herd type (exposed or non-exposed herds) and age-group at death as classification factors. Differences in  $\log_e$  GGT between survivors and those not recorded as survivors (called “non-survivors”) were analysed by least squares, using the JMP package from SAS (1995); herd and a binomial code for survival to different ages were fitted as fixed effects. The  $\log_e$  GGT  $\times$  survival relationship was also investigated in the reverse manner, i.e., by comparing the survival of cows with GGTs above or below a given threshold level. Threshold GGTs of 50, 100, and 200 i.u. litre<sup>-1</sup> were tested. In addition, logistic regressions were used to investigate the probability of survival against  $\log_e$  GGT.

Finally, sire-model heritability estimates of cow survival and of  $\log_e$  GGT were carried out (progeny groups of at least 10 daughters each, comprising 65 sires and 1068 daughters) using restricted maximum likelihood (REML) procedures (Johnson & Thompson 1995). Data on the dam of each cow were not used in this study, because only one year-group of animals was under analysis. However, the sires’ own pedigree data were included in the REML analyses, thereby taking account of common links in the sires’ ancestry. A fixed effect in the REML analyses was fitted for the effect of herd-of-origin (i.e., the herd in which each cow was originally blood-sampled as a 2-year-old). For survival, this fixed effect could only account for the 2-year-old herd, and from then on at least some cows in many of the herds became widely dispersed, for which it was not possible to make further statistical adjustment.

## RESULTS

### Exposed versus non-exposed herds

The cumulative percentages of animals culled by the end of each age group (lactation) up to June 2000 were compared for cows from the 43 exposed and 17 non-exposed herds (Fig. 1). Survival percentages to the end of the 1st, 2nd, 3rd, or 6th lactation were not significantly affected by herd-

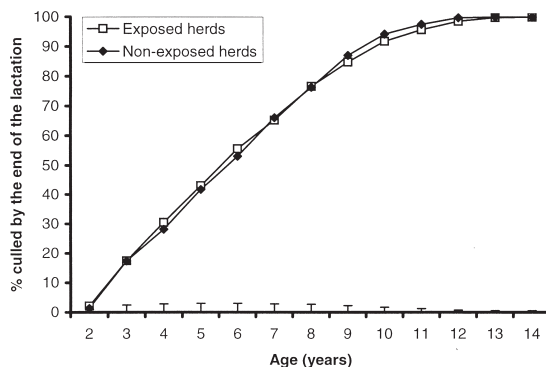


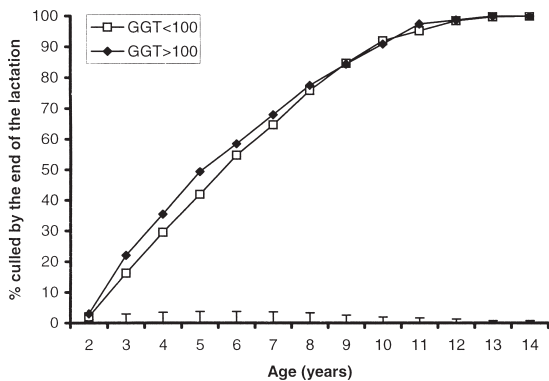
Fig. 1 Cumulative distribution of age at disposal in exposed and non-exposed herds (standard errors of differences between means are shown along the  $x$ -axis).

type. There were, however, highly significant differences in  $\log_e$  GGT among the 43 herds classified as having been exposed to FE ( $P < 0.001$ ).

### Survival and $\log_e$ GGT within exposed herds

Restricting analysis to the 43 exposed herds only, the phenotypic relationship between survival and  $\log_e$  GGT was analysed in two ways. Firstly, least-squares mean  $\log_e$  GGTs for non-survivors ( $n = 24$ ) and survivors ( $n = 1044$ ) after the 1st lactation were 4.31 and 3.79  $\log_e$  i.u. litre<sup>-1</sup>, respectively, giving a difference of  $0.52 \pm 0.23 \log_e$  i.u. litre<sup>-1</sup> ( $P < 0.05$ ). For the 1044 remaining, no subsequent differences in  $\log_e$  GGT (as measured in 1989) between later culls and later survivors were significant, indicating no long term carryover effects of a 1st-lactation challenge.

Secondly, the differences in survival were calculated between animals above or below thresholds of 50, 100, or 200 i.u. litre<sup>-1</sup> in the FE challenge year (3.91, 4.61, and 5.30  $\log_e$  i.u. litre<sup>-1</sup>, respectively). There were (from the 1068 animals analysed), 71.3% with a GGT < 50 i.u. litre<sup>-1</sup>, 7.1% between 50 and 100 i.u. litre<sup>-1</sup>, 6.4% between 100 and 200 i.u. litre<sup>-1</sup>, and 15.3% above 200 i.u. litre<sup>-1</sup>. For the 50 i.u. litre<sup>-1</sup> threshold, the percentage of non-survivors (cows culled/died) by the end of 2nd lactation was 16.7% for those below, and 19.5% for those above the threshold (and did not differ significantly;  $P < 0.27$ ), but for 100 i.u. litre<sup>-1</sup> the corresponding values were 16.2 and 22.1% (136/837 and 51/231;  $P = 0.05$ ) (Fig. 2), and for 200 i.u. litre<sup>-1</sup> 16.1 and 25.2% (146/905 and 41/163;  $P < 0.01$ ). Logistic regression analyses of survival



**Fig. 2** Cumulative distribution of age at disposal in exposed herds for animals above or below a GGT enzyme activity level of 100 i.u. litre<sup>-1</sup> (standard errors of differences between means are shown along the *x*-axis).

on log<sub>e</sub> GGT (not shown) led to a similar conclusion: the odds of survival fell by 0.90 (SE 0.05) as log<sub>e</sub> GGT increased by one unit. A regression analysis of age at final culling against log<sub>e</sub> GGT within herd (treating “herd” as a random effect) gave a coefficient of -0.08 (SE 0.07) years per log<sub>e</sub> unit, which was not significant, but the sign was in the expected negative direction.

### Heritability estimates

Restricting data to those cows in sire groups of 10 or more and managed within exposed herds (*n* = 1068 animals with records), the heritability estimates for cow survival to the end of the 1st, 2nd, 3rd, or 6th lactation were all very low and not significantly different from zero. Individual estimates were: 1st, 0.01 ± 0.03; 2nd, 0.02 ± 0.05; 3rd, 0.02 ± 0.05; and 6th, 0.00 ± 0.05.

For the same 1068 animals, the heritability estimate for log<sub>e</sub> GGT was 0.32 ± 0.10. The phenotypic SD of log<sub>e</sub> GGT was 1.08 log<sub>e</sub> units, with the corresponding genetic SD 0.62 log<sub>e</sub> units.

## DISCUSSION

### Exposed versus non-exposed herds

In analysing genetic differences among sire groups, the prevalence of exposed versus non-exposed herds could be the result of a number of possible factors, such as geographical differences among herds

which undoubtedly affected the level of natural sporidesmin challenge experienced on pasture, effects of weather/microclimate of the farm, or the differing abilities of herd-owners and their staff to protect stock against FE. Although we tested for differences in cow survival between these two herd-types, many other factors probably influenced herd-owners’ culling decisions, especially pregnancy status of the cows.

Only one sample for GGT was taken from each animal in this project. In other studies we have found a high repeatability (0.85) of serial GGT samples after challenge (Morris et al. 1998). This suggests that one blood sample should be enough to classify animals, as long as they have received the same exposure (i.e., they have grazed together).

For the survival statistics, it was necessary to follow contemporary within-herd groups of animals over a lifetime. For example, there were cases where an entire herd (or a large subgroup) was relocated, although the original owner may or may not have moved with the herd; there were cases where groups of animals were transferred to the ownership of some other relative (e.g., a son or daughter) on the same property; there were also many sales of stock to a nearby or to a very distant region of the country. It was not possible to code contemporary groups to each cow for all of her lifetime, so there is a considerable amount of chance involved as to where a long-time survivor may end up lactating in, say, her 10th lactation, with or without any subsequent FE challenge, relative to her original 1st-lactation contemporaries.

The apparent very low culling rate in the remainder of the 1st lactation (Fig. 2) was probably because some cows which were to be culled for non-pregnancy or late pregnancy had already been removed from the main herd by the time the blood sample was taken for this study. However, they were sold but not necessarily slaughtered, as explained later. In the same way, the overall figure of 69.0% of calved cows blood sampled in the following April/May is lower than might be expected among 2-year-olds, but this is explained because of small numbers of 2-year-olds grazing away from the main herd after having been dried off, and not necessarily accessible for blood sampling. The percentage was not lower in exposed than in non-exposed herds.

### Survival and log<sub>e</sub> GGT within exposed herds

On a within-herd basis, the comparisons of survival and the relationships between log<sub>e</sub> GGT and

survival reflected expressions of performance among animals, some of which were possibly genetic in nature. The mean  $\log_e$  GGT of survivors was  $0.52 \pm 0.23 \log_e$  units lower than for non-survivors at the end of the 1st lactation ( $P < 0.05$ ). We published an estimate of this in 1991 (Morris et al. 1991b) as  $0.35 \pm 0.15 \log_e$  units ( $P < 0.05$ ), but some cows that were nominally culled by farmers by 1991 (sold but not slaughtered) have since calved in a recorded herd and been re-instated on the national database.

A significant difference in survival to the end of 2nd lactation was found for those with GGT above or below 100 i.u. litre<sup>-1</sup>, and for those with GGT above or below 200 i.u. litre<sup>-1</sup>. The same trend (although not significant) was found for those with GGT above or below 50 i.u. litre<sup>-1</sup>. For animals surviving at least to the end of the 1st lactation, there was no long term carryover effect of a 1st-lactation challenge.

Considering FE-challenged animals retained by a farmer who may have access to GGT data (unlike the protocol in our study), a related question is whether subsequent production is affected. This has been evaluated recently by Hopkinson et al. (2002). They monitored the performance for 12 months (after a single blood sample, taken in April 1999) of 116 cows affected by FE in one herd, comprising "all that recovered sufficiently from clinical symptoms and were present in the herd in the following season". Cows retained were considered by the herd-owner to have recovered from FE, based on lack of photosensitivity. No relationship was found between production levels for the season and plasma concentrations of GGT or glutamate dehydrogenase in the survivors from the previous season.

### Heritability estimates

Our heritability estimate for  $\log_e$  GGT in 1990 was  $0.31 \pm 0.10$  (Morris et al. 1990), compared with the present estimate here of  $0.32 \pm 0.10$ . There were 43 animals out of the original 1523 which could not be matched on the nationally unique animal key (or 20 out of 1088 after selecting on progeny-group size and exposed-herd status).

In the interval between 1990 and the present work, an analysis of another study by LIC of the FE susceptibility of 265 Jersey bulls as a result of dosing with sporidesmin (2 calf crops) or pasture challenge (1 calf crop) was published by Morris et al. (1998), with a heritability estimate of  $0.77 \pm 0.13$ . We have combined data from the two studies (some

sires in common), providing a combined heritability estimate for  $\log_e$  GGT in Jerseys of  $0.48 \pm 0.10$ . A heritability study of  $\log_e$  GGT in 257 Friesian bulls gave estimates of  $0.29 \pm 0.15$  and  $0.46 \pm 0.27$  from univariate and multivariate analyses, respectively (Morris et al. 1998). In summary, although there may be differences in heritability between breeds or between FE-challenge procedures, which require further study, there can be little doubt that there is heritable variation for  $\log_e$  GGT in cattle. This is consistent with our findings in sheep (Morris et al. 1995), where genetic progress has been made in response to selection pressure applied to increase or reduce susceptibility to FE. In addition, there is the possibility of a major gene segregating in some Jerseys, which may explain some of the difference between the results from our two Jersey studies.

The heritability estimates for cow survival were all low, averaging about 0.01. Much larger numbers of sires and records are needed to provide a precise estimate for a trait with a truly low estimate. Cue et al. (1996) published heritability estimates for survival from 1st to 2nd lactation and from 1st to 3rd lactation, based on New Zealand data from heifers calving between 1987 and 1991. Their estimates for Jerseys were 0.05 and 0.09 (SE c. 0.015), and for Holstein-Friesians 0.03 and 0.05 (c. SE 0.015).

### CONCLUSIONS

We have shown significant effects for the following: differences in  $\log_e$  GGT between survivors and non-survivors, and differences in cow survival to the end of the 2nd lactation according to GGT status in the season of FE challenge. The revised heritability estimate for  $\log_e$  GGT from this study with Jerseys was  $0.32 \pm 0.10$ , and the combined estimates across all Jersey data gave a value of  $0.48 \pm 0.10$ , from which the implications are that genetic progress could be made in selecting for increased resistance to FE.

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