

# Genetic parameters for faecal egg count following mixed, natural, predominantly *Ostertagia circumcincta* infection and relationships with live weight in young lambs

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## Abstract

Faecal egg counts and live weights were measured on approximately 200 predominantly twin-born Scottish Blackface lambs each year for 3 years, from 1 to 6 months of age. Measurements were made at 4-week intervals following anthelmintic treatment. Heritability estimates (with s.e.s) of log transformed faecal egg count at each age were 0.01, 0.00, 0.12 (0.10), 0.14 (0.12), 0.15 (0.07) and 0.22 (0.13), for ages 1 to 6 months respectively. Therefore, genetic variation exists for acquired but apparently not for innate resistance to infection. Maternal common environmental effects (with s.e.s) were 0.36 (0.11), 0.20 (0.05), 0.27 (0.09), 0.06 (0.08), 0.15 (0.09) and 0.16 (0.08), for ages 1 to 6 months respectively. Genetic correlations between faecal egg counts in lambs older than 3 months were not significantly less than 1.0, indicating that faecal egg counts at different ages are expressions of the same trait. Phenotypic correlations between faecal egg counts were generally positive but small. Measurement error contributed one-third of the observed variation for individual egg counts. The heritability of mean faecal egg count from 3 to 6 months was 0.33 (s.e. 0.15), indicating that selection decisions can be made more accurately using multiple egg counts per animal. Phenotypic correlations between faecal egg counts and live weight were generally negative but close to zero. However, genetic correlations between faecal egg counts and live weight in lambs older than 3 months were close to -1.0, indicating that resistance to gastrointestinal parasites may be an important genetic determinant of growth rate in this environment.

**Keywords:** faecal egg counts, genetic parameters, live weight, *Ostertagia*, sheep.

## Introduction

Profitable sheep farming relies, in part, upon the efficient control of nematode infection. Currently, the major control strategy is the use of anthelmintics, however this strategy is threatened by the evolution of drug resistance in parasite populations (Jackson, 1993). An alternative, or additional, method for helping to control nematode infection is the selective breeding of sheep with enhanced resistance to infection (Stear and Murray, 1994), especially as selective breeding schemes in Australia and New Zealand have been remarkably successful in improving resistance of sheep to infection with *Haemonchus contortus*, *Trichostrongylus colubriformis* and to mixed, natural infections (Piper, 1987; Windon, 1990; Baker *et al.*, 1991; Woolaston and Piper, 1996).

In northern Europe, a prevalent and economically important parasite is the abomasal nematode *Ostertagia (Teladorsagia) circumcincta*, and this parasite dominates the parasitic fauna of grazing sheep. The genetic parameters of resistance to infection with predominantly *O. circumcincta* are not known, but they are required before the feasibility of selective breeding can be assessed. In addition, a knowledge of the sources of variation among sheep is essential for understanding the infection and for developing sustainable strategies for reducing the effects of parasitic disease.

The aims of this paper are: firstly, to partition the variation observed in faecal egg count in lambs following natural, predominantly *O. circumcincta* infection into components attributable to genetic,

maternal and measurement error effects, and hence to calculate genetic parameters, secondly, to investigate relationships between faecal egg counts at different ages; and, thirdly, to investigate relationships between faecal egg count and live weight.

## Material and methods

### *Animals and experimental design*

Data were collected from a commercial flock of Scottish Blackface sheep on an upland farm in south-west Strathclyde, Scotland. Whilst grazing, these sheep were exposed to natural, mixed, predominantly *O. circumcincta* infection. A total of 185, 188 and 194 lambs were studied in 1992, 1993 and 1994, respectively. The lambs were sired by a total of 23 rams, were predominantly twin born and each year were born within a 2-week period. Lambs were kept in two separate fields prior to weaning each year and after weaning, at about 16 weeks of age, all lambs were kept together on one field to minimize variation in exposure to infective larvae.

Each year faecal samples were collected from the rectum when lambs were 4 weeks of age on average, and thereafter at 4-week intervals until the lambs were 24 weeks of age (26 weeks in 1992 and 1993), giving six samples per animal. Faecal egg counts were made from a 3-g sample of faeces using the modified McMaster technique (Gordon and Whitlock, 1939; Bairden, 1991). Each egg count represented 50 eggs per g. These counts will be referred to as EPG1, EPG2 ... EPG6. In 1993 duplicate aliquots from the same faecal sample were counted for EPG4, EPG5 and EPG6, and in 1994 quadruplicate samples were counted for EPG6. The majority of larvae recovered from culture were *O. circumcincta*. Other parasites identified from the faecal samples but not analysed were from the genera *Strongyloides*, *Nematodirus* and *Eimeria*. After collection of each faecal sample, all lambs were treated with a broad spectrum anthelmintic (Albendazole sulphoxide) which was given at the recommended dose rate of 5 mg/kg body weight, based on the weight of the heaviest lamb at the time of treatment. The efficacy of the anthelmintic treatment was tested with a faecal egg count reduction test and there was no evidence of drug resistance within this flock. Live weight was also measured at each of the first five sampling dates each year, and will be referred to as WT1, ... WT5.

### *Data analysis*

Preliminary analyses of all traits were undertaken using the GENSTAT statistical package (Lawes Agricultural Trust, 1983) to determine significant fixed effects and test the distributional assumptions

for the faecal egg count data. All traits were subsequently analysed fitting fixed effects of sex, birth type and the interaction of field and year of birth, with age at sampling fitted as a covariate. All faecal egg counts (FEC) were positively skewed and were transformed by  $\log(\text{FEC} + 1)$  prior to analyses.

Genetic parameters were estimated by residual maximum likelihood (REML), fitting an animal model and using a derivative free algorithm to solve the equations (DFREML) (Meyer, 1989). For univariate analyses, additive genetic and maternal common environment components of variance were estimated. Additionally, for the traits of EPG4, EPG5 and EPG6 where replicated egg counts were available, individual animal variance components were also estimated. The data structure was not suitable for estimating maternal genetic effects, and the maternal common environment component may include maternal genetic effects.

Phenotypic and genetic correlations between traits were estimated using the bivariate technique outlined by Thompson *et al.* (1995). Fitting the same models to each trait as for the univariate analyses did not lead to convergence in the cases attempted, possibly because there were up to 11 (co)variance components to be estimated in each bivariate analysis. Therefore, a simplified model was used in which all dam information was deleted from the data and only the additive genetic and individual animal components of variance were fitted, along with the appropriate covariances and also repeated record effects, where necessary.

Standard errors for each parameter of interest were calculated from the second derivative of the log likelihood, evaluated at the parameter estimate. To enable this derivative to be calculated, a log likelihood profile was obtained by fixing the parameter at points at, and on either side of, the REML parameter estimate and then re-maximizing the likelihood for all other variance components.

## Results

Mean values and phenotypic standard deviations for faecal egg count,  $\log(\text{FEC} + 1)$  and live weight at each of the sampling times are shown in Table 1. Also shown are the maximum values for FEC at each time. A previous paper has discussed the distribution of faecal egg counts using the data collected in 1992 and 1993, and all samples taken from weaning (EPG4) onwards conformed to a negative binomial distribution (Stear *et al.*, 1995a).

Heritabilities ( $h^2$ ), maternal common environmental ( $c^2$ ) and individual lamb ( $i^2$ ) effects are shown in

**Table 1** Mean values and phenotypic standard deviations for faecal egg count (EPG) and live weight (WT) measured at months 1 to 6

	EPG1	EPG2	EPG3	EPG4	EPG5	EPG6
Age (weeks)	4	8	12	16	20	24
Mean (eggs per g)	77	218	317	224	205	220
$\sigma_p$ †	475	264	398	260	297	222
Maximum	8100	3500	3200	2300	2700	1950
	log(EPG1+1)	log(EPG2+1)	log(EPG3+1)	log(EPG4+1)	log(EPG5+1)	log(EPG6+1)
Mean	1.09	4.55	4.84	3.90	3.53	3.70
$\sigma_p$	2.18	1.71	1.70	2.35	2.44	2.22
	WT1	WT2	WT3	WT4	WT5	
Mean (kg)	10.7	17.8	23.0	27.2	29.7	
$\sigma_p$	1.96	2.67	3.43	3.58	3.12	

†  $\sigma_p$  = phenotypic standard deviation.

**Table 2** Heritabilities, maternal common environment and individual animal effects for faecal egg count (EPG) measured at months 1 to 6†

	EPG1	EPG2	EPG3	EPG4	EPG5	EPG6
$h^2$ ‡	0.01	0.00	0.12	0.14	0.15	0.22
s.e.			0.10	0.12	0.07	0.13
$c^2$ ‡	0.36	0.20	0.27	0.06	0.15	0.16
s.e.	0.11	0.05	0.09	0.08	0.09	0.08
$i^2$ ‡			0.50	0.31	0.32	

† All data log transformed prior to analyses.

‡  $h^2$  = heritability;  $c^2$  = maternal common-environment effect;  $i^2$  = individual animal effect.

Table 2. The  $i^2$  effects were estimated for cases where replicate egg counts were made. The heritabilities of single egg counts were essentially zero at 4 and 8 weeks of age, then increased to a maximum of 0.22 at 24 weeks. In contrast, maternal common environmental ( $c^2$ ) effects (which may include maternal genetic effects) were large in the very young lambs and decreased as the animal matured, but were still evident 8 weeks after weaning (0.16). The individual lamb ( $i^2$ ) effects were large, as may be expected, accounting for up to half of the variation at the last three sampling times. The true measurement error ( $e^2$ ), as defined by the variation between replicated egg counts from the same faecal sample, can be estimated from the variance components in Table 2 for EPG4, EPG5 and EPG6 (i.e.  $e^2 = 1 - h^2 - c^2 - i^2$ ). The  $e^2$  values at these three sampling times were 0.30, 0.39 and 0.30, respectively. In other words, approximately one-third of the variation observed among animals in faecal egg counts was due solely to measurement error.

An alternative method for estimating  $h^2$  values for EPG4, EPG5 and EPG6, where there are replicated measurements, is to estimate heritabilities choosing just one measurement per animal per sampling time, repeating this across all possible replicate

combinations. Doing this and averaging the values obtained resulted in  $h^2$  and  $c^2$  values of 0.14 and 0.06 for EPG4, 0.16 and 0.13 for EPG5 and 0.21 and 0.14 for EPG6. These values are close to those shown in Table 2.

Simultaneous analysis of the data from EPG3 to EPG6 gave a heritability estimate for a single egg count of 0.12 (s.e. 0.06), a maternal common environmental effect of 0.06, an individual animal effect common to all measurements ( $i^2$  permanent) of 0.01, and an individual animal effect specific to each measurement ( $i^2$  temporary) of 0.49. The measurement error effect was 0.31. The relatively low heritability and maternal effects suggest that the true genetic correlation among egg counts may be less than one and the nature of the maternal influence may differ between sample dates.

Genetic and phenotypic correlations between faecal egg counts at different times are shown in Table 3. No attempt was made to calculate genetic correlations with EPG1 and EPG2 because these traits expressed essentially no genetic variation. Residual correlations, obtained from univariate

**Table 3** Phenotypic (above diagonal) and genetic (below diagonal) correlations between faecal egg count (EPG) at months 1 to 6††

	EPG3	EPG4	EPG5	EPG6
EPG1	0.09	0.01	0.02	0.06
EPG2	-0.05	-0.07	-0.07	-0.04
EPG3		0.16	0.14	0.22
EPG4	1.00		0.24	0.25
EPG5	0.69	0.92		0.22
s.e.	0.26	0.25		
EPG6	0.99	0.80	0.82	
s.e.	0.23	0.33	0.25	

† All data log transformed prior to analyses.

†† Phenotypic correlation (EPG1, EPG2) = -0.01

GENSTAT REML, rather than phenotypic correlations in the true sense, are presented for these two traits, although for all traits residual and phenotypic correlations were almost identical. All genetic correlations between EPG3, EPG4, EPG5 and EPG6 were large, positive and not significantly different from 1.0, indicating that faecal egg counts on these four occasions may effectively be expressions of the same trait, or closely related traits.

The pattern of phenotypic correlations between successive faecal egg counts differs from that for genetic correlations. From EPG3 onwards, all measurements were positively correlated with each other, although this correlation was moderate, generally in the range 0.15 to 0.25. Faecal egg counts at 8 weeks were weakly, negatively, correlated with subsequent measurements, and all correlations with EPG1 were close to zero.

Heritabilities and maternal common environmental effects for live weight are shown in Table 4, along with correlations with faecal egg count. Again, no attempt was made to calculate genetic correlations with EPG1 or EPG2, and residual correlations are shown for these two traits. All live weight heritability values were large, with the exception of the 20-week value, although they are compromised by large standard errors which limit their interpretation. The maternal common environment values were also very high, confirming the strong maternal effects often observed in this breed. Interestingly, the genetic but not the phenotypic correlations among live weight and faecal egg count were large and negative. Phenotypic correlations with growth rate immediately prior to, or immediately after, the time of sampling were similar to those presented in Table 4.

## Discussion

The number of eggs counted in the faeces is a complex interaction between the level of parasite

**Table 4** Heritabilities and maternal common environment effects for live weight (WT), and phenotypic and genetic correlations with faecal egg count†

	WT1	WT2	WT3	WT4	WT5
$h^2$	0.41	0.50	0.43	0.28	0.14
s.e.	0.28	0.30	0.16	0.25	0.14
	0.49	0.43	0.47	0.46	0.38
s.e.	0.13	0.14	0.11	0.12	0.09
$r_p$	0.09	-0.00	-0.13	-0.01	-0.06
$r_g$			-0.90	-1.00	-0.63
s.e.			0.28		0.32

† Faecal egg count data log transformed prior to analyses.

burden in the host and the fecundity of the adult female parasites, both of which may be regulated by density-dependent factors outside the direct control of the host. Therefore, there may not be a linear relationship between faecal egg count and either an animal's immune response or the actual parasite burden. As such, faecal egg counts may be considered to simply be an indicator of resistance/susceptibility to parasite infection. Actual non-linear relationships between faecal egg counts and true worm burdens are explored elsewhere (M. J. Stear and S. C. Bishop, unpublished results).

At the population level, many factors may lead to variation in faecal egg counts. Variation between lambs over and above that explicable by genetic background and maternal effects will exist at any given time due simply to, for example, chance effects in exposure to larvae, variations in acquired immunity, variations in density-dependent factors, etc. When actual faecal samples are taken there will also be variation due to the faecal sample collected, that is whether or not the sample is representative in terms of the number of eggs present per unit of faecal material. These factors will all contribute to the observed  $i^2$  effects. Finally, having obtained the faecal sample, there will inevitably be measurement error, leading to the  $e^2$  effects observed for EPG4, EPG5 and EPG6.

The existence of additive genetic effects has been reported in Australia and New Zealand for egg counts following infection with *H. contortus* (Woolaston *et al.*, 1991; Woolaston and Piper, 1996), *T. colubriformis* (Windon, 1990) or mixed natural infections (Baker *et al.*, 1991; Cummins *et al.*, 1991; Bisset *et al.*, 1992; Douch *et al.*, 1995, amongst others). Additive genetic effects for faecal egg counts have also been reported in African sheep (Baker *et al.*, 1994). Although the estimated heritabilities for FEC need not be similar for studies which have different parasite species, different environmental conditions and different experimental protocols, the estimate at 6 months of age (0.22) is remarkably similar to most published estimates which usually lie between 0.2 and 0.3.

In this study there was little or no detectable genetic variation in faecal egg counts in very young animals. This indicates that there is either no innate resistance to infection or that there is no genetic variation in innate resistance, with genetic variation only existing for acquired resistance. The mechanisms of acquired resistance are not known for any parasite, but experimental results suggest that variation in faecal egg counts following infection with *O. circumcincta* in sheep is due to variation in parasite-specific IgA responses which regulate parasite fecundity, and

also to variation in the production of globule leucocytes which are associated with decreased worm burdens (Smith *et al.*, 1985; Stear *et al.*, 1995b).

Maternal common environmental effects accounted for a large but variable proportion of the observed variation in faecal egg counts. There are several possible maternal influences. Firstly, maternal antibodies in milk and colostrum might influence egg counts directly or by interfering with the development of effective immunity in the lamb. The consistent negative correlation between egg counts at 2 months of age and subsequent egg counts could be due to maternal antibodies: those lambs with most maternal antibody are best protected at a young age, but develop immunity more slowly or less effectively. Alternatively, chance differences in parasite exposure might lead to higher counts initially but subsequently to better immune responses and lower counts. A second potential maternal influence could be grazing behaviour. Lambs were kept with their mothers until 4 months of age and lambs often grazed together with their dams. Therefore, dams which tended to graze more heavily infected areas would have lambs with higher than average exposures. Learnt grazing behaviour may also account for maternal effects persisting beyond weaning. Additionally, ewe milk production might affect the amount of grass lambs eat prior to weaning, and hence their exposure to infective larvae.

The genetic correlations presented in this paper indicate that faecal egg counts from 3 to 6 months of age may be expressions of the same trait. The phenotypic correlations between EPG3, EPG4, EPG5 and EPG6 are, however, considerably lower than the equivalent genetic correlations. This may simply reflect the variation inherent in faecal egg counts, especially as these sheep were given an effective anthelmintic at each sampling date. In terms of variance components, the only terms which contribute to a phenotypic correlation in this example are the genetic covariance, the maternal covariance and the permanent  $i^2$  component. Simultaneous analyses of EPG3 to EPG6 (see **Results**) suggested that the latter two components may be small, hence the small observed phenotypic correlations.

The large observed measurement error, approximately one-third of the variation, highlights the importance of distinguishing between the heritability of a single egg count and that of mean faecal egg count. Taking the average faecal egg count from several replicated counts from the same sample will reduce the measurement error and thus increase the heritability. For example, taking the mean of two

replicated counts will, theoretically, halve the measurement error term and increase the heritability for EPG6 to 0.26, although the maximum value obtainable by this means will only be 0.31. A more effective means of increasing the heritability may be to average faecal egg counts across time, as this will reduce both the  $e^2$  and the temporary  $i^2$  effects, and is justified by the high estimated genetic correlations between sampling dates. Assuming that faecal egg counts are made on two separate occasions, then the heritability should increase from 0.12 to 0.20, and for four sampling times to 0.31. Averaging all faecal egg counts per animal from EPG3 to EPG6, including the replicated counts, gave a heritability of 0.33 (s.e. 0.15), compared with an approximate predicted value of 0.32 obtained when  $i^2$  (temporary) and  $e^2$  were scaled by the average number of sampling times per animal and the average number of faecal egg counts made per animal, respectively.

The large, negative genetic correlations between faecal egg count and live weight indicate that resistance to gastrointestinal parasites is probably an important determinant of growth rate, in this environment. These genetic correlations are somewhat stronger than those previously reported between faecal egg count and live weight, most of which are close to and not significantly different from zero (e.g. Bisset *et al.*, 1992; Baker *et al.*, 1991; Eady *et al.*, 1994; McEwan *et al.*, 1995; Douch *et al.*, 1995), although those reported by Bisset *et al.* (1994) for female lambs under natural infection were stronger, being close to -0.5. As pointed out by Eady *et al.* (1994), there is considerable inconsistency amongst reports of genetic correlations (both estimated and implied by correlated responses to selection) between faecal egg counts and production traits. It is not clear why the correlations in this paper differ from previously published results but there does appear to be a strong case for monitoring both faecal egg count and live weight in selective breeding schemes in the United Kingdom, especially if the level of parasite exposure varies between years.

The phenotypic correlations between live weight and faecal egg count were low, in agreement with most published results. The same arguments may be used to rationalize these results as were used to account for the low correlations between successive faecal egg counts. In other words, a single faecal egg count is a very variable entity. Moreover, live weight at any given age is a cumulative effect of all the experiences that the lamb has had up to that age, whereas a faecal egg count simply describes the state of being at that point in time, for the current infection. Correlations were also calculated between live weight and the mean faecal egg count up to that point in time. Again, the genetic correlations were

always close to -1.0, whereas the phenotypic correlations were negative but small.

In summary, we have demonstrated that genetic variation exists for acquired, but not for innate resistance to infection to *O. circumcincta* infection in Scottish Blackface lambs under Scottish grassland conditions, with maternal common environment effects also contributing significantly to the variation. In this data set, faecal egg counts taken at different ages are, genetically, strongly correlated with each other and genetically negatively correlated with live weight. Therefore, selective breeding, using the average of several faecal egg counts, may offer a desirable and feasible option in assisting in the control of nematode infections.

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