THE ROYAL SOCIETY

Heritable variation in resistance to gastro-intestinal nematodes in an unmanaged mammal population

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The impact of parasites on natural populations has received considerable attention from evolutionary biologists in recent years. Central to a number of theoretical developments during this period is the assumption of additive genetic variation in resistance to parasites. However, very few studies have estimated the heritability of parasite resistance under field conditions, and those that have are mainly restricted to birds and their ectoparasites. In this paper, to our knowledge, we show for the first time in a free-ranging mammal population, Soay sheep (*Ovis aries*) living on the islands of St Kilda, that there is significant heritable variation in resistance to gastrointestinal nematodes. This result is consistent with earlier studies on this population which have indicated locus-specific associations with parasite resistance. We discuss our results in the context of current studies examining heritable resistance to parasites in domestic sheep and the possible mechanisms of selective maintenance of genetic variation for resistance to gastrointestinal nematodes in the St Kilda Soay sheep population.

Keywords: *Teladorsagia circumcincta*; heritability; generalized linear models; parasites; ungulate; sheep

1. INTRODUCTION

The impact of parasites on host survival, fecundity and general fitness can be considerable (Gulland 1995; Grenfell & Gulland 1995; Møller 1997), and recent theoretical advances suggest that parasites may play an important role in a number of areas impinging on both natural and domestic animal populations. These include life-history evolution (Møller 1997), sexual selection (Hamilton & Zuk 1982; Read 1988; Maynard Smith 1991), population dynamics (Anderson & May 1978, 1979), the maintenance of genetic variation (Hamilton 1982; Potts & Wakeland 1990; Hill *et al*. 1991) and the evolution of sex (Jaenike 1978; Hamilton 1980, Hamilton *et al*. 1990; Howard & Lively 1994). An implicit assumption of many of these theoretical models is that resistance to parasites and pathogens has a significant additive genetic component. For example, the Hamilton–Zuk hypothesis for the evolution of secondary sexual characters assumes that by choosing males with elaborate traits (and low parasite loads) females are gaining good resistance genes for their offspring.

Despite this interest, we are aware of just two studies of natural vertebrate populations that have demonstrated a heritable component to parasite resistance, and both of these involve birds infested with ectoparasites (see reviews by Read 1990; Clayton 1991; Zuk 1992). Møller (1990) demonstrated heritable resistance to mites in a population of barn swallows (*Hirundo rustica*) by moving chicks between nests at an early age and comparing their parasite loads with those of their true and surrogate parents. In most natural populations, however, cross-fostering experiments are not feasible and heritable parasite resistance can only be inferred by a positive correlation between parent and offspring parasite loads. This is the method used by Boulinier *et al*. (1997), in their study of tick resistance in a cliff-nesting population of kittiwakes (*Rissa tridactyla*). Since ectoparasites can be directly transmitted between individuals, this latter study provides only weak support for heritable parasite resistance. This is less of a problem for studies of resistance to endoparasites, such as nematodes, but if parents and offspring share a common environment then this can inflate any estimate of parasite heritability. In many animals, fathers do not provide any parental care and hence sire heritabilities may be unbiased and a comparison of sire and dam heritabilities can provide information on bias due to maternal effects.

We are aware of no studies that have estimated the additive genetic variation of parasite resistance in an unmanaged population of mammals, or indeed any free-ranging population infected with an endoparasite. In contrast, there has been considerable research effort

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Received 25 January 1999 *Accepted* 4 March 1999

devoted to quantifying innate resistance to endoparasites in many managed populations of domestic mammals, including sheep, cattle and goats (Gruner 1991; MacKinnon *et al*. 1991; Bisset *et al*. 1992; Stear & Murray 1994; Woolaston & Piper 1996). These studies have been stimulated by the occurrence of widespread anthelmintic resistance in parasites of domestic livestock and are motivated by the possibility of selectively breeding for host resistance to particular parasites. Heritability estimates of faecal egg count (a measure of nematode worm burden) in domestic sheep range between 0.13 ± 0.07 (McEwan *et al*. 1992) and 0.53 ± 0.15 (Baker *et al*. 1994). Studies of domestic livestock have several advantages over most comparable studies of natural populations. Not only do they usually have detailed pedigree data, but they are also able to standardize, or at least control for, many of the non-genetic sources of variation in parasitism rates (Stear & Murray 1994).

In this paper, we examine the amount of heritable variation in resistance to gastrointestinal nematodes in a free-ranging population of Soay sheep living on the island of Hirta in the St Kilda archipelago, off the north-west coast of Scotland. This population has been intensively monitored for over a decade (Clutton-Brock *et al*. 1991, 1992) with detailed life history, phenotypic and genetic data collected on the vast majority of the study area population. Thus we are able to assess the importance of genetic influences on parasitism and to determine non-genetic sources of variation in parasitism rates (e.g. those related to age, sex, body size, etc.) that might contribute significantly to parasite loads independent of genotype.

Soay sheep on St Kilda undergo periodic population crashes when up to 70% of the sheep may die over winter (Grenfell *et al*. 1992). Although the proximate cause of death is protein-energy malnutrition, both observational and experimental evidence suggest that gastrointestinal nematodes, especially the strongyle nematode *Teladorsagia circumcincta*, play an important role in determining the number and identity of the sheep that die each winter (Gulland 1992; Gulland & Fox 1992; Gulland *et al*. 1993; Illius *et al*. 1995). Thus, nematode parasites are an important selective force in this population. Despite this, the presence of locus-specific associations with parasite resistance (Gulland *et al*. 1993; Smith 1996; Paterson *et al*. 1998) suggests that there is significant additive genetic variation in parasite resistance in the population. In this paper, to our knowledge, we show for the first time in a free-ranging, naturally regulated mammal population that there is a significant heritable component to resistance to gastrointestinal nematodes, as measured by faecal egg count.

2. METHODS

(a) *Study population*

Soay sheep (*Ovis aries*) are the most primitive breed of domestic sheep in Europe and closely resemble the original wild species (Clutton-Brock 1981). They have survived unmanaged on the St Kilda archipelago (59° 49′ N, 08° 34′ W) for between 1000 and 2000 years (Boyd & Jewell 1974). Historically, the population has been restricted to the small island of Soay (99 ha), but in 1932, 107 sheep were introduced to the much larger island of Hirta (638 ha). The Hirta population now comprises 700–2000 animals, and our study population in the Village Bay area includes about one-third of the total Hirta population.

Since 1985, *ca*. 95% of individuals born in the study area each year have been caught and individually tagged soon after birth (Clutton-Brock *et al*. 1992). At this time lambs were weighed and sampled for genetic analyses. In summer each year, about 65% of the study area population were caught and measured, and animals that had not previously been handled were sampled for genetic analysis.

(b) *Parasite data*

Since monitoring natural life histories is part of the study, a non-destructive measure of parasitism was used. Parasitism rates were estimated by regular monitoring of faecal egg counts (FECs). This is the density of gastrointestinal nematode eggs in the faeces and is a standard method of quantifying parasite resistance. Eighty per cent of gastrointestinal nematodes in this population are the strongyle nematode *T. circumcincta* and the damage observed to the abomasum at post-mortem examination is consistent with this species being a major selective force in this population (Gulland & Fox 1992). Since 1988, a single faecal sample has been collected from each animal caught during the annual summer catch-up. During each spring and autumn, multiple faecal samples have been collected from free-ranging females and males respectively (Gulland *et al*. 1993). Strongyle FEC were determined from 3 g faecal samples using a modified McMaster technique (MAFF 1971).

Since FECs are the product of the number and fecundity of adult female worms a host harbours, it measures resistance to parasite establishment, parasite growth and reproductive rates, although it cannot distinguish between them. However, analyses of Soay sheep dying naturally on St Kilda (Grenfell *et al*. 1995) and of a culled population of Soay sheep on the island of Lundy, in the Bristol Channel (Grenfell *et al*. 1995; H. E. G. Boyd, personal communication) indicate that there is a strong positive correlation between strongyle worm burden and FEC in sheep not treated with anthelmintics. Similar correlations have also been shown in domestic sheep (Bisset *et al*. 1992; Stear *et al*. 1995*a*,*b*).

(c) *Molecular determination of paternity*

Blood and tissue samples taken at the time of tagging or subsequent capture were used to genotype each individual at up to 17 loci (six proteins and 11 microsatellites, see Smith (1996), Bancroft *et al*. (1995) and Pemberton *et al*. (1999), for details). These data were used to infer paternity. Because lambs were tagged soon after birth, while still attended by their mothers, we are confident of maternal identification and genetic data for mother–offspring pairs to confirm our observations. In the rut, both sexes of Soay sheep are highly promiscuous and mating observations are poor predictors of paternity for any particular lamb (Bancroft 1993; Coltman *et al*. 1999). Since six-month-old ram lambs regularly gain paternities (Bancroft 1993; Stevenson & Bancroft 1995; Pemberton *et al*. 1996; Smith 1996), all males alive in a rut were considered as candidate fathers for each lamb, and paternal relationships were inferred genetically.

Paternity analysis was carried out using a computer program to compare genotypes of candidate males for the presence of appropriate allelic combinations for each lamb, as determined by comparison of mother–offspring genotypes. Exclusion probabilities (the probability that an unrelated male chosen at random would have the appropriate allelic combination) and

log-likelihood scores were calculated for each ram–lamb pair according to population allele frequency data and paternal genotype (Marshall *et al*. 1998), which allowed identification of paternity at given levels of confidence. Paternity was assigned with greater than 80% confidence for 665 lambs (317 of which had a confidence of 95%) (T.C. Marshall, personal communication; Pemberton *et al*. 1996). These paternities together with the known maternal information were used in the analysis of the heritability of FECs.

(d) *Estimation of residual FECs*

Calculating the heritability of FECs presents a number of statistical difficulties. The first is that FEC varies markedly between individuals due to a variety of non-genetic factors that are beyond the control of the investigators, including population density, age, sex and body weight (Gulland & Fox 1992; Smith 1996; Wilson *et al*. 1996). The second is that the distribution of FEC commonly conforms to the negative binomial (where the majority of the worms are harboured by a minority of the population), whereas standard methods for calculating heritability assume a normal error distribution (Wilson *et al*. 1996, Wilson & Grenfell 1997). Here we minimize these problems by calculating the heritability of residual FEC. This is the residual value after fitting a generalized linear model (GLM) that includes non-genetic factors as explanatory terms (see Smith (1996) and Paterson *et al*. (1998) for details of the non-genetic factors tested), and which explicitly defines a negative binomial error distribution (Wilson *et al*. 1996; Wilson & Grenfell 1997). Not only does this method provide an individual-specific measure of parasite resistance after controlling for non-genetic variation, but it also provides a value that is normally distributed and so conforms to the main assumption of standard parametric methods. The GLMs used to calculate residual FECs were generated using the *glm.nb* procedure (Venables & Ripley 1997) in the *Splus* statistical package (Mathsoft Inc. 1993) and are described in detail elsewhere (Smith 1996; Wilson *et al*. 1996).

FECs were measured each spring, summer and autumn between 1988 and 1994. Spring samples were restricted to females to coincide with their lambing period and the autumn samples were restricted to males to coincide with their annual rut. Thus, although it was possible to estimate the heritability of summer residual FECs for both sexes, the heritability of spring residual FECs could be estimated only for females, and autumn residual FECs only for males.

The summer GLMs are based on a single FEC measure taken each year. Sampling in the spring and autumn involved repeat samples from individuals over a period of time. For these GLMs, the geometric mean FECs provide a single measure of FEC for each individual in each year of analysis. Heritability estimates were based on the residual FEC for an individual in a particular season, averaged over all years of measurement.

Heritability estimates involving both sexes were based on residual FEC values generated from joint-sex GLMs (with sex as a factor in the model), whereas those involving a single sex were generated from single sex GLMs. The GLMs used to calculate residual FECs generally included animals of all ages. However, because the heritability of parasite resistance may vary with age, additional summer GLMs were generated which were restricted to lambs sampled in their first summer (aged four months).

(e) *Heritability analysis*

In this analysis we measured the narrow sense heritability of residual FECs, calculated by two methods: offspring–parent regression and sib analysis. Heritability (and its standard error, s.e.) is most simply calculated by the regression of mean offspring values on parent values for a particular trait (residual FEC in this instance). The heritability of the trait is then estimated by the regression coefficient (in the case of regressions involving the mid-parental value), or twice the regression coefficient (in the case of regressions involving a single parent) (Falconer & Mackay 1996). The standard error of the heritability is simply the standard error (or twice the standard error) of the regression coefficient. These estimates were checked for robustness using standard bootstrapping methods (Manly 1991) based on 2500 replicates (analyses not shown, see Smith 1996). To test for significance from zero, we used standard parametric methods (analysis of variance, ANOVA), and more robust randomization methods (i.e. permutation; Manly 1991), again based on 2500 replicates. These two methods produced similar p-values, and both are shown for comparison.

In a sib analysis, the variation in the trait values of offspring is partitioned between that which is explained by the identity of the parent(s) and that due to other unexplained effects (which will include dominance and environmental effects). Where males mate with more than one female in a given breeding season, the identity of the mother (dam) should be nested within that of the father (sire) to account for the presence of both full and half siblings in the data set. However, on St Kilda, both males and females mate promiscuously and so there are very few full-sibs (55 out of 665 lambs with known paternity (8.3%) have a known full-sib). Therefore, a nested approach is inappropriate for the present analyses and only half-sib heritability estimates can be determined. In standard sib analyses, ANOVA methods are used to determine the proportion of phenotypic variance explained by the identity of the parent, which is included in the model as a fixed effect. In reality, the parentage of the lambs is not controlled and hence ANOVA may introduce biases into estimates of heritability (Falconer & Mackay 1996). The identity of the parent should therefore be treated as a random factor in the statistical analysis. Further biases can be introduced into heritability estimates when the number of offspring per parent varies. Both of these problems can be reduced by using restricted maximum likelihood (REML; Shaw 1987; Meyer 1990). Thus, in the present analysis, we used the *varcomp* procedure and the REML method in *Splus* (Venables & Ripley 1997) to estimate the appropriate variance components. From these, half-sib heritabilities were calculated as four times the intra-class correlation coefficient (Falconer & Mackay 1996), and approximate standard errors were calculated following Roff (1997). Significance levels for heritability estimates were again calculated using both standard ANOVA methods and randomization based on 2500 replicates (Manly 1991; see Smith (1996) for details).

One potential source of bias that might be introduced into these analyses is due to variation in the number of offspring per parent (though this is minimized by using REML). This is particularly likely for sire estimates. Although they may be few, those males which do have large numbers of offspring may have an important effect on the results of this analysis. To examine this possibility, we conducted two sets of analyses. In both sets, we included only families with two or more offspring, so that the within-family variance could be more accurately measured (in practice, this had little impact of the magnitude heritability estimates, but their significance levels were reduced by virtue of smaller sample sizes). In the first set of analyses we included all of the remaining families, and in the second we restricted the analysis to individuals with less than a given number of

Table 1. *Heritabilities (± standard errors) for residual FECs, based on offspring–parent regression analysis*

(*n*, number of offspring–parent pairs involved in the analysis. n/a, number of pairs too small to perform analysis. Significance values were determined by both standard ANOVA (p_{ANOVA}) and randomization (p_{random}), see text for details. Significant estimates are in bold type.)

		sire				dam				mid-parent			
offspring	season	*n*	$h^2 \pm$ s.e.	p_{ANOVA}	p_{random}	*n*	$h^2 \pm$ s.e.	p_{ANOVA}	p_{random}	*n*	$h^2 \pm$ s.e.	p_{ANOVA}	p_{random}
female	summer	52	0.090 ± 0.312	0.490	0.498	**96**	**0.407 ± 0.174**	**0.012**	**0.016**	81	0.132 ± 0.183	0.237	0.239
male	summer	48	0.087 ± 0.255	0.312	0.364	69	0.056 ± 0.195	0.387	0.403	66	0.076 ± 0.187	0.342	0.341
all	summer	88	0.085 ± 0.225	0.353	0.350	148	0.093 ± 0.128	0.235	0.236	147	0.166 ± 0.128	0.099	0.095
female	spring	n/a	n/a	n/a	n/a	113	0.271 ± 0.241	0.132	0.132	n/a	n/a	n/a	n/a
male	autumn	21	0.599 ± 0.464	0.106	0.108	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a

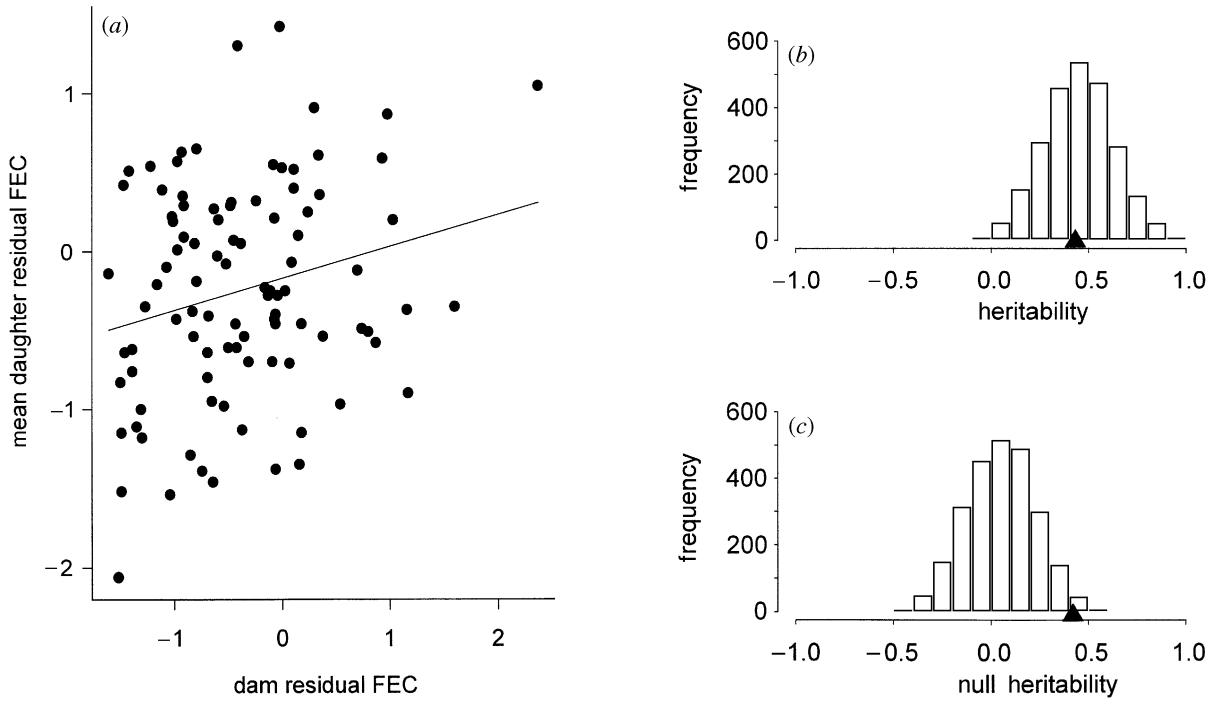

Figure 1. Offspring–parent regression for mean daughter summer residual FEC against dam summer residual FEC: (*a*) shows the regression (equation for regression line: $y = -0.171 + 0.203x$); (*b*) shows the bootstrapped heritability distribution; and (*c*) shows the null distribution generated by randomization, the mean of which is approximately zero (−0.002). Less than 2% (38 out of 2500) of the randomized heritability estimates were as large as that observed (see main text). The observed heritability estimate (0.407) is indicated by a solid triangle in figures (*b*) and (*c*). The jackknifed mean ± standard error was 0.406 ± 0.190.

offspring. These analyses indicated that in general there was no significant effect of sibship size on heritability estimates. However, for very small sibships (five or fewer offspring), heritabilities did change and significance levels were reduced. For comparison, we also present these analyses.

3. RESULTS

(a) *Offspring–parent regression analysis*

The results of the regression analysis are shown in table 1. Heritability estimates based on regression analysis were all positive, and ranged between 0.056 and 0.599. The probability of all 11 estimates being positive purely by chance alone is less than 1% ($p < 0.001$, binomial test). If one considers just those tests that are truly independent ($n = 6$), then this probability is reduced to $p = 0.015$. Moreover, the combined probability (Sokal & Rohlf 1981) of the six independent tests is also suggestive of heritable variation in residual FECs (Fisher's combined probability test: based on probabilities generated by standard ANOVAs: $p = 0.027$; based on probabilities generated by randomization: $p = 0.037$). However, just one of the 11 heritability estimates was significantly greater than zero; this was based on the mean daughter–dam regression of summer residual FEC (figure 1). This relationship was also significant when residual FEC was determined solely in lambs (results not shown).

(b) *Sib analysis*

The results of the main sib analysis are shown in table 2*a*. Heritabilities ranged from approximately zero to 0.688 for sire estimates and from approximately zero to 0.305 for dam estimates. Three of the sire and two of the dam estimates were significantly greater than zero (based on both standard ANOVA and randomization methods). None of the heritabilities based on lamb residual FECs

Table 2. *Heritabilities (± standard errors) for residual FECs, based on sib analysis for (a) all families and (b) families comprising less than six offspring*

(Significance levels were calculated via both standard ANOVA (p_{ANOVA}) and randomization (p_{random}). Significant estimates are shown in bold type; n_{fam}, number of families (parents) used in the analysis; n_{off}, number of offspring used. The heritability calculations assumed that all offspring were half-sibs; the inclusion of a small number of full-sibs may mean that some of the estimates are slightly inflated.)

(*a*)

		sire					dam				
offspring	season	n_{fam}	n_{off}	$h^2 \pm$ s.e.	p_{ANOVA}	p_{random}	n_{fam}	n_{off}	$h^2 \pm$ s.e.	p_{ANOVA}	p_{random}
females	summer	**65**	**235**	**0.688 ± 0.287**	**0.001**	**0.002**	**90**	**347**	**0.305 ± 0.220**	**0.046**	**0.032**
males	summer	48	191	0.227 ± 0.286	0.223	0.140	72	262	0.246 ± 0.252	0.230	0.138
all	summer	**101**	**500**	**0.233 ± 0.166**	**0.007**	**0.014**	**141**	**757**	**0.263 ± 0.135**	**0.011**	**0.001**
females	spring	**88**	**374**	**0.403 ± 0.216**	**0.008**	**0.016**	71	317	0.155 ± 0.202	0.078	0.149
males	autumn	27	77	0.000 ± 0.484	0.867	0.880	40	111	0.223 ± 0.433	0.366	0.328

(*b*)

		sire					dam				
offspring	season	n_{fam}	n_{off}	$h^2 \pm$ s.e.	p_{ANOVA}	p_{random}	n_{fam}	n_{off}	$h^2 \pm$ s.e.	p_{ANOVA}	p_{random}
females	summer	**58**	**174**	**1.245 ± 0.341**	**0.001**	**0.001**	73	215	0.217 ± 0.291	0.244	0.198
males	summer	40	112	0.129 ± 0.413	0.493	0.349	61	184	0.001 ± 0.296	0.779	0.850
all	summer	74	221	0.264 ± 0.287	0.051	0.141	94	295	0.244 ± 0.240	0.251	0.102
females	spring	**68**	**197**	**0.554 ± 0.320**	**0.027**	**0.029**	51	159	0.240 ± 0.330	0.162	0.222
males	autumn	26	69	0.000 ± 0.540	0.835	0.910	40	111	0.223 ± 0.433	0.366	0.328

were significantly greater than zero (results not shown). As with the regression analyses the significant heritability estimates involved female offspring, either alone or in combination with males from a joint-sex GLM.

When animals with very large numbers of offspring were removed from the analysis, the heritabilities remained largely unchanged. However, when animals contributing greater than five offspring were excluded from the analysis, only the sire–daughter heritabilities for summer and spring retained their significance (table 2*b*). These results suggest that although the heritability estimates shown in table 2*a* are extremely robust (particularly the sire–daughter estimates), animals contributing large numbers of offspring to the analysis may inflate some of the heritability estimates.

4. DISCUSSION

We have shown that there is significant heritable variation in parasite resistance in the Hirta Soay sheep population. Significant estimates were derived from regression analyses and sib analyses and were shown to be robust by randomization methods. Significant heritability estimates ranged from 0.233 to 0.688, which is towards the upper end of the range of heritabilities previously reported in domestic sheep (Albers *et al*. 1987; Bisset *et al*. 1992, 1994; McEwan *et al*. 1992; Bishop *et al*. 1996; Woolaston & Piper 1996). However, direct comparison with studies in domestic sheep may not be appropriate because these generally involve single parasitic infections within specific anthelmintic regimes, whereas the present study involved a continual mixed infection of strongyle nematodes and no routine anthelmintic treatment.

The two methods for estimating heritability gave slightly different results. Using the offspring–parent regression method all of the estimated heritabilities were positive, but only the mean daughter–dam summer estimate was significantly greater than zero. Using the sib analysis method, not only was this estimate significant, but so were four others. There are a number of possible explanations why the two methods gave different results. First, the sample sizes are smaller for the regression analyses. Second, the regression method relies on accurate measures of FEC in both the parent and the offspring, whereas only offspring FEC (and the identity of the parent) is required for the sib analysis; hence there is more noise in the regression estimates. Third, the sib analysis included a small number of full-sibs, which may have inflated the heritability estimate and contributed a large dominance genetic variance (V_D), a component of the phenotypic variance which contributes to similarity between full-sibs but not between parents and their offspring (Hoffmann & Parsons 1997).

Previous studies of the heritability of FECs in domestic sheep have rarely measured sex-specific heritabilities. However, in doing so here, we find that all the significant estimates of heritability of residual FECs in Soays involve the trait measured in females. This suggests that genetic factors are more important in determining phenotypic variation in FECs in females relative to males. Earlier modelling of FECs in Soay sheep has shown that males exhibit much weaker systematic patterns of variation and fail to show the decline in FEC with age that is a conspicuous feature of infections in females (Smith 1996; Wilson *et al*. 1996). These observations are consistent with the hypothesis that females develop much stronger acquired

immunity to their parasites than males (Murray *et al*. 1971; Jeffcoate *et al*. 1990) and that this acquired immunity is under genetic control.

The demonstration of significant sire effects in the sib analysis is especially important because they are unlikely to involve the common environment effects which may confound dam estimates of heritability. Common maternal environment effects have been shown to be an important influence on mother–offspring resemblance in other parasitological studies (Bishop *et al*. 1996; Stear *et al*. 1997*a*). Male Soay sheep do not contribute any form of parental care and, because male immigration into the study area is common during the annual rut, there may be little overlap in the home ranges of most sire–offspring combinations. The confidence level for assigning paternities was set at 80% for this study and as such may have the effect of reducing sire heritability estimates as a proportion of the paternities may have been wrongly assigned. It is interesting, therefore, that the sire heritabilities for female FECs in both summer and spring were extremely robust and more than twice as large as those for the equivalent dam heritabilities. We believe that this may be a result of a sex difference in the span and timing of breeding production. On average, females produce one or two offspring each year over a period of four or five years. Males, on the other hand, tend to produce most of their offspring over a period of just two or three years. Thus, the amount of unexplained environmental variation in offspring FECs will tend to be larger for dams than for sires, simply because their offspring are exposed to more environments (years). This will have the effect of reducing the relative magnitude of the additive genetic variation in FEC. Future investigations will explore this possibility further.

Heritability estimates generated from the spring data (a time of possible hormonal and nutritional stress for females at lambing) and autumn data (a time of possible stress for males in the rut) were similar to those estimated from the (relatively stress-free) summer data, although there was a tendency for the summer estimates to be slightly higher. It is possible that during the spring and autumn, environmental influences (including climatic factors) become more important in generating variation in either the number of infective larvae available on the pasture, or the sheep's feeding or immunological responses.

Heritability estimates based on residual FECs from lambs aged four months generally proved non-significant and small (results not shown). This may reflect the smaller sample sizes involved, the importance of (unidentified) environmental effects on FECs in animals of this age, or the failure of young animals to develop genetically based acquired immunity. Recently Stear *et al*. (1997*a*,*b*; see also Bishop *et al*. 1996) used a sib analysis to estimate the heritability of resistance to mixed nematode infections in Scottish blackface lambs aged one to six months. They found that heritability of FECs increased from 0.06 ± 0.11 in one-month-old lambs to 0.21 ± 0.11 in the same animals aged four months, to 0.33 ± 0.15 in six-month-old lambs. Thus, in this breed of sheep at least, the heritability of FECs increased with age and the development of acquired immunity, and was significantly greater than zero by the time the sheep were four months old. Interestingly, Stear *et al*. (1997*b*) found that when these animals were sacrificed at six months of age, the heritability of worm burden was not significantly different from zero, but worm length and fecundity were. Thus, it appears that the heritable component of FECs in young animals is not resistance to worm establishment, but resistance to worm growth and egg production (see also Smith *et al*. 1987).

The presence of heritable variation in parasite resistance in the Hirta population allows scope for selection mediated through fluctuating selection, frequency or density-dependent processes, and/or through sexual selection. Although the relative importance of each of these mechanisms has yet to be established, it is pertinent to note that several locus-specific associations with FECs have recently been revealed in the Hirta population (Gulland *et al*. 1993; Smith 1996; Paterson *et al*. 1998). These involve two protein loci, transferrin and adenosine deaminase, the microsatellite locus Maf45 and microsatellites within the ovine major histocompatibility complex region. In each case, specific alleles were associated with either reduced or increased FEC. These results may indicate a coevolutionary relationship between host resistance and parasite virulence genes and support theories of frequency-dependent selection maintaining additive genetic variation in the population. However, other processes, including sexual selection, may also be involved and further investigations are clearly required.

We are grateful to the National Trust for Scotland and to Scottish Natural Heritage for permission to work on St Kilda and for their assistance in many aspects of the work. We also gratefully acknowledge the generous assistance and support provided on St Kilda by Army units stationed on St Kilda, Benbecula and the mainland. We thank Mike Stear and three anonymous referees for valuable comments on earlier drafts of this paper. Members of the St Kilda Soay Sheep Project and associated volunteers helped with fieldwork. This work was supported by the Natural Environment Research Council and Biotechnology and Biological Sciences Research Council.

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