GENETIC VARIABILITY OF RESISTANCE INDICATORS FOR GASTROINTESTINAL NEMATODE INFECTION IN ANGORA AND CASHMERE GOATS

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INTRODUCTION

The widespread occurrence of anthelmintic resistance in gastrointestinal nematode (GIN) populations affecting sheep and goats is of major concern to these industries, and is responsible for a concerted research effort into alternative control measures (Zajac and Gipson, 2000 ; Terill *et al*., 2001). The determination of resistance in grazing ruminants is generally based on measurement of faecal egg count (FEC) (Baker *et al.,* 1998) and selection has been generally on this variable. However, a range of alternative phenotypic traits is available (Mandonnet *et al.,* 1996) and may prove superior alone or in combination with FEC. These traits include those associated with the immune response to GIN, such as specific antibody levels (AB) and blood eosinophil counts (EOS), or those associated with the pathological effects of infection such as packed cell volume (PCV) an indicator of anaemia (Sheep, Pernthaner, *et al.,* 1995 ; Goats, Mandonnet *et al.*, 2001). To make use of these variables we need to know their heritabilites and the phenotypic and genetic relationships between them, an area where information on the goat is lacking relative to the sheep. The objective of this study is therefore to characterise the basic genetic parameters for FEC, PCV and EOS in two goat breeds during natural and induced infections with gastrointestinal nematodes. This report is preliminary in nature, representing data from the first of 5 years of data to be collected.

MATERIALS AND METHODS

This study involved investigation of host genetic resistance to GIN infection in 234 mixed sex progeny of 6 Angora bucks and 250 female progeny of 9 Cashmere bucks, on two commercial properties in the Northern Tablelands of New South Wales (NSW), Australia. To provide a uniform early immunological stimulus, half of the kids were orally vaccinated with irradiated (L_3) larvae of *Trichostrongylus colubriformis* (Black scour worm) at 1 and 2 months of age (5 000 and 14 000 larvae respectively). All kids were subjected to natural nematode infection up to 5 months of age with FEC taken at 3 and 5 months of age, followed in each case by anthelmintic treatment to terminate the infection. One week after anthelmintic treatment at 5 months, all kids were challenged with an oral dose of 10,000 infective L_3 larvae of *T. colubriformis.* Faecal egg count (FEC), were determined for individual kids at 3 (FEC3) and 5 (FEC5) months of age during natural challenge and at days 28 (FEC6.25) and 35 (FEC6.5) after the artificial challenge. Individual FEC was determined using the modified McMaster floatation technique (MAFF, 1986). Blood samples were collected from individual kids by jugular venipuncture and analysed for blood parameters (including PCV and EOS) at 2, 3, 5 and 6.25 months old using an automated haematology analyser (Cell-Dyn® 3500, Abbott, Norfolk, VA USA) with-in 48 hours of collection.

Statistical analyses. Exploratory data analysis and the analysis of fixed effects were performed using S-PLUS software (MathSoft, 1999). The distribution of FEC and EOS data were skewed, they were cube root and log transformed to CFEC and LEOS, respectively, to normalise their distribution. For Angora kids the effects of sire, vaccination, birth type (single or twin), age (in days, fitted as covariate), sex, and their 1st order interactions were fitted in the model for variance component estimation. The effects fitted for the Cashmere data were sire, vaccination, birth type, management group and their 1st order interactions. Heritabilities for these traits (FEC, PCV and EOS) were estimated using a sire model in a univariate analysis of ASREML (Gilmour *et al.,* 1999) fitting the significant effects only. A multivariate analysis of ASREML was performed to estimate the phenotypic correlations between these traits. Due to the small size of the data the genetic correlation was not estimated.

RESULTS AND DISCUSSION

Test of fixed effects. Sire effects in FEC were significant at 5 months in both breeds, and at 6.5 months in Angora and 6.25 months in Cashmere goats. In Angora goats, significant sire differences were found for EOS at 5 months and for PCV at all ages except for PCV at 2 months (PCV2). For the Cashmere goats significant sire effects ($P < 0.001$) were found for EOS and PCV at all ages except for PCV2. This is in contrast to the result of (Mandonnet *et al.*, 1996) who observed no significant sire effects for PCV at 6 and 10 months old in Creole goats. For the Angoras sex was significant ($P < 0.05$) for EOS at 5 months and for PCV at all ages, EOS was not influenced by age but PCV2 and PCV3 were. In the Cashmeres, there were significant ($P < 0.05$) effects of birth type on EOS5, PCV2 and PCV3. Management group effects were found for EOS at all ages but not for PCV. Oral vaccination of kids early in life with irradiated L_3 larvae of *T. colubriformis* had no influence on FEC at 3, 5 and 6.25 months in either breed, but significantly ($P < 0.01$) increased FEC at 35 days (FEC6.5) post artificial challenge in Angora kids (3496 ± 175 vs. 2812 ± 171 for vaccinated and control treatments respectively). The lack of response to vaccination during natural challenge may have been due to the fact that natural infection was predominantly due to a different species, *Haemonchus contortus*, although failure of vaccination has also been described in lambs under similar conditions (Windon, 1991). The significant elevation of FEC following later challenge with the homologous species (*T. colubriformis*) is a novel finding and suggests that some form of immunological tolerance may have been induced by the vaccination regimen used.

Genetic analyses. Table 1 shows the heritability estimates (± standard errors) and phenotypic correlations for LEOS and PCV at 2, 3, 5 and 6.25 months and CFEC at 3, 5, 6.25 and 6.5 months old in Angora and Cashmere kids. In Angora goats, the heritability estimates for LEOS, PCV and CFEC ranged between 0.10 ± 0.13 to 0.24 ± 0.21, 0.18 ± 0.17 to 0.74 ± 0.43 and 0.10 ± 0.13 to 0.25±0.21, respectively, and were highest for the 3 traits at 5 months (natural infection, predominantly with *Haemonchus contortus*). In Cashmere goats, the estimates for LEOS ranged from 0.28 ± 0.20 to 0.66 ± 0.34, for PCV from 0.09 ± 0.12 to 0.51 ± 0.29 and 0.04 ± 0.10 to 0.23 ± 0.19 for CFEC. The heritability estimates for FEC were generally lower in this study than the estimates of 0.33 ± 0.06 in Creole goat (Mandonnet *et al.*, 2001) and 0.37 ± 0.18 in Scottish Cashmeres (Jackson, 1999). Estimates for PCV agreed with the report of 0.10 to 0.33 for Creole goats (Mandonnet *et al.,* 2001).

Table1. Estimates of heritability (± s.e.) and phenotypic correlation for faecal egg count (CFEC), blood eosinophil count (LEOS) and packed cell volume (PCV) measured at 3, 5 and 6 months old in Angora and Cashmere goats with and without artificial challenge

Heritability			Phenotypic correlation		
Traits*	Angora	Cashmere	Traits	Angora	Cashmere
Natural challenge					
$LEOS_2$	0.14 ± 0.15	0.66 ± 0.34			
PCV_2	0.18 ± 0.18	0.09 ± 0.12			
$CFEC_3$	NE	0.04 ± 0.10	PCV_3 : $CFEC_3$	-0.17 (0.08)	-0.23 (0.04)
$LEOS_3$	0.15 ± 0.16	0.62 ± 0.33	$LEOS_3$: $CFEC_3$	0.05 (0.01)	-0.03 (0.01)
PCV_3	0.18 ± 0.17	0.29 ± 0.20	$LEOS_3$: PVC_3	-0.03 (0.01)	-0.02 (0.01)
$CFEC_5$	0.25 ± 0.21	0.23 ± 0.19	PCV_5 : $CFEC_5$	-0.06 (0.06)	0.21 (0.00)
$LEOS_5$	0.24 ± 0.21	0.46 ± 0.27	$LEOS_5$: $CFEC_5$	0.08 (0.01)	-0.01 (0.01)
PCV_5	0.74 ± 0.43	0.47 ± 0.27	LEOS5 : PCV_5	-0.14 (0.01)	-0.01 (0.01)
Artificial challenge					
$CFEC_{6.25}$	0.10 ±0.13	0.19 ± 0.18	$PCV_{6.25}$: $FEC_{6.25}$	-0.02 (0.06)	-0.05 (0.09)
$LEOS_{6.25}$	0.10 ± 0.13	0.28 ± 0.20	$LEOS_{6.25}$: $CFEC_{6.25}$	-0.20 (0.01)	-0.11 (0.02)
$PCV_{6.25}$	0.29 ± 0.23	0.51 ± 0.29	$LEOS_{6.25}$: $PCV_{6.25}$	0.15 (0.01)	0.09 (0.01)
$CFEC_{6.25}$	0.23±0.21	0.07±0.09			

NE = not estimable, CFEC = cube root transformed FEC, LOES = natural log transformed EOS ; *subscript refers to the month of measurement.

Phenotypic correlation. The phenotypic correlation between FEC and EOS was -0.20 and -0.11 in Angora and Cashmere goats, respectively, and most significant, at 6 months. This agrees with Pernthaner *et al.*, (1995) who obtained a negative correlation between FEC and EOS in sheep for both *T. colubriformis* and *H. contortus* infection. The strongest association between the EOS and PCV was also, at 6 months in both Angora (0.15) and Cashmere (0.09) goats. However, the most significant phenotypic relationships between FEC and PCV was negative and at 3 months (-0.17 for Angora and -0.23 for Cashmere). The direction of all of these associations is consistent with the known role of eosinophils in combating GIN infections (Balic *et al.*, 2000) and the negative effect of infection, particularly with *H. contortus*, on PCV. In general the phenotypic correlations for these traits were generally low, indicating considerable independence between the traits. Also, the inconsistency in the relationships over time, suggest that it would be unwise to base selection decisions for parasite resistance on either PCV or EOS. However, the heritability estimates for FEC in both breeds of goat were mostly generally moderate, indicating that there is a reasonable within-breed genetic variation for GIN resistance, which may be exploited in selection programs. This is consistent with findings in Creole (Mandonnet *et al.*, 2001) and Scottish Cashmere goats (Jackson *et al.*, 1999) but not in Fijian goats (Woolaston *et al.,* 1995). It should be noted that only a small number of sire groups has been used to date (6 Angora and 9 Cashmeres) and that as we build up the data set our estimates of genetic parameters will improve substantially.

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