Australian Government
Rural Industries Research and Development Corporation

Breeding Fibre Goats for Resistance to Worm Infections

Gastrointestinal, nematode or helminth

By S. Walkden-Brown, B. Sunduimijid, M. Olayemi, J. Van Der Werf and A. Ruvinsky
(School of Rural Science and Agriculture, University of New England)
and L. Le Jambre
(CSIRO Livestock Industries)

April 2008

RIRDC Publication No 07/184
RIRDC Project No UNE 69A

ISBN 1 74151 579 3
ISSN 1440-6845

Breeding Fibre Goats for Resistance to Worm Infections*: Gastrointestinal, nematode or helminth)*
Publication No. 07/184
Project No.UNE-69A

Researcher Contact Details
Associate Professor Stephen W. Walkden-Brown
Animal Science W49
University of New England
Armidale, NSW 2351

Phone: 02-6773 5152
Fax: 02-6773 3922
Email: swalkden@une.edu.au

In submitting this report, the researcher has agreed to RIRDC publishing this material in its edited form.

RIRDC Contact Details
Rural Industries Research and Development Corporation
Level 2, 15 National Circuit
BARTON ACT 2600
PO Box 4776
KINGSTON ACT 2604

Phone: 02 6271 4100
Fax: 02 6271 4199
Email: rirdc@rirdc.gov.au.
Web: http://www.rirdc.gov.au

Published in April 2008

Foreword

Gastro-intestinal helminth (worm) infection (GIH) is the major health problem of grazing goats world-wide, including Australian fibre goats. In recent years the sheep industry has made considerable progress in describing the genetic component of resistance to infection and incorporating this trait in breeding programs. The goat industry has lagged behind in this, primarily because the genetic parameters for resistance traits, and their association with production traits have not been defined in Australian goat breeds. Successful incorporation of genetic resistance to gastro-intestinal helminth infection in goats could result in reduced anthelmintic (drench) usage and concurrent reduction in the selection pressure for anthelmintic resistance, and also improved efficiency of fibre production.

The major aim of the project was to obtain genetic parameter estimates for worm resistance traits and production traits in Australian Cashmere and Angora goats, and phenotypic and genetic correlations amongst them.

The project results are of interest to geneticists and goat breeders worldwide, with special relevance to Australia. The use of worm infections with worm species common in both Northern and Southern regions in Australia means that the results should have broad application in the Australian industry.

The findings support other studies in showing that rapid genetic progress can be made in selection for fleece traits and moderate progress for bodyweight. Selection for resistance to worm infection is likely to result in slow genetic progress with no evidence of negative correlated responses in production traits. For the full benefits of the information included in this report to be captured, wider adoption of industry genetic evaluation programs (such as MOPLAN) is required. Such programs enable significant progress to be made, even with traits of low to moderate heritability, due to accurate estimation of breeding values.

The project has also developed and validated a panel of microsatellite markers which can be used for parentage analysis in goats. An attempt to control worm infections using early oral vaccination with radiation-attenuated larvae was unsuccessful and should not be pursued further.

This project was funded from industry levies plus matching funds and RIRDC Core Funds provided by the Australian Government.

This report, an addition to RIRDC's diverse range of over 1700 research publications, forms part of our Rare Natural Fibres R&D program, which aims to facilitate the development of new and established industries based on rare natural fibres.

Most of our publications are available for viewing, downloading or purchasing online through our website:

- downloads at www.rirdc.gov.au/fullreports/index.html
- purchases at www.rirdc.gov.au/eshop

Peter O'Brien
Managing Director
Rural Industries Research and Development Corporation

Acknowledgments

We thank Tony Brown and Margaret Harris for their unstinting support of this project despite many obstacles and hardships, and for the hard work they put in to it both practically, and intellectually. Without their support and access to their goat herds there would have been no project. We also thank their spouses, Judy Brown and Jim Harris, for their indulgence with this complication to their already busy lives. Dr Brad Crook was an important investigator early in the life of the project and we thank him for his contribution. We are grateful for the excellent technical support of Neil Baillie (UNE) and Dominic Niemeyer (CSIRO) - again the project could not have run without them. Other key contributors at various stages of the project include Simon Stachiw, Michael Raue, Lisa Mascord and Warren Flood all from UNE. This report draws significantly on the PhD theses of Bolormaa Sunduimijid (Sunduimijid, 2005) and Muyiwa Olayemi (Olayemi, 2004) who are co-authors of this report.

Abbreviations

ANSTO	The Australian Nuclear Science and Technology Organisation
BAS	Circulating basophil count
CSD	Mean cashmere diameter
CSW	Cashmere (down) weight
CSY	Cashmere yield % (=CSW/GFW *100)
CURVE	Mean fibre curvature
CV (%)	Coefficient of variation of MFD
EBV	Estimated breeding value
ELISA	Enzyme Linked Immunosorbent Assay
EOS	Blood eosinophil count
GFW	Greasy fleece weight
GIN	Gastrointestinal nematodiasis. Roundworm infection of the gut.
HD	Mean hair diameter
HGB	Blood haemoglobin content
IgG	Immunoglobulin G. Also used as the abbreviation for the analysed variable, specific circulating IgG directed against *T. colubriformis*.
ILV	Irradiated larval vaccine (or vaccination)
KEMP%	Percentage of kemp fibres
LYM	Blood lymphocyte count
MCH	Mean corpuscular haemoglobin
MCHC	Mean corpuscular haemoglobin content
MCV	Mean corpuscular volume (red cell volume)
MED%	Percentage of medullation (hollowness) in fibre snippets
MFD	Mean fibre diameter
MFD_SD	Standard deviation of MFD
MON	Blood monocyte count
NEU	Blood neutrophil count
PCV	Packed cell volume
PCR	Polymerase chain reaction
RBC	Blood red cell (erythrocyte) count
WCC	Blood white cell (leucocyte) count
WEC	Faecal worm egg count

Contents

Executive Summary

What is the report about?

This report details a long-term study into the feasibility of breeding Angora and Cashmere goats for resistance to worm (gastrointestinal nematode or helminth) infections. The research is important because gastro-intestinal nematodiasis (GIN) is the major health problem of grazing goats word-wide, including Australian fibre goats. The problem is exacerbated by a widespread, and growing, problem with anthelmintic resistance in worm populations on goat farms. This makes ongoing reliance on chemical control of GIN a risky and possibly non-sustainable approach. Selection for genetic resistance in the host is an alternative control measure which could be used in conjunction with chemical and other control measures to place worm control on a more sustainable footing into the future.

Who is the report targeted at?

The report is targeted primarily at geneticists and animal breeders in the fibre goat industry. It is also targeted at policy makers within the industry. Parasitologists servicing the goat industry will also find the information in this report useful.

Background

At the time this project commenced there was emerging evidence from overseas studies of genetic variation in resistance to GIN in goats that could be exploited to select for resistance to infection. No such information was present for Australian goats. At that point the sheep industry had made considerable progress in describing the genetic component of resistance to infection and incorporating this trait in breeding programs. The goat industry has lagged behind in this, both because of less use of industry-wide performance recording schemes (eg. Sheep Genetics Australia) and also because the genetic parameters for resistance traits, and their association with production traits had not been defined in Australian goat breeds. Successful incorporation of genetic resistance to gastro-intestinal helminth infection in goats could result in reduced anthelmintic (drench) usage and concurrent reduction in the selection pressure for anthelmintic resistance, and improved efficiency of fibre production.

Aims/Objectives

The major aims of the project were as follows:

- to obtain genetic parameter estimates for worm resistance traits and production traits in Australian Cashmere and Angora goats, and phenotypic and genetic correlations amongst them;
- to develop a DNA marker set for parentage analysis and to have the quantitative genetic analysis based on DNA verified pedigrees;
- to provide an evaluation of potential markers for worm infection or resistance in goats other than faecal worm egg count (WEC);
- to evaluate the use of irradiated larval vaccines for control of worm infections in goats; and
- to obtain new information on the effects of worm infections in goats of the two breeds in the New England environment.

The beneficiaries of the project are likely to be geneticists, parasitologists and animal breeders in the fibre goat industry who have a new and comprehensive set of genetic parameter estimates for Angora and Cashmere goats under commercial Australian conditions. Ultimately goat producers themselves should be the beneficiaries as decision-making regarding breeding programs and parasite control is put on a more scientific footing.

Methods used

The project utilised one commercial Angora and one commercial Cashmere resource herd, both near Barraba in Northern NSW (Latitude 30.38˚ S; Longitude 150.61˚ E). In both herds does were mated in single sire groups in March/April. In the Angora herd does were regrouped into a single mob after joining and they kidded down as one mob. In the Cashmere herd, does were allocated to different "management groups" after joining and kidded down in these groups, each containing does mated to each of the sires used. Link sires were used across years to account for year effects. Both male and female kids were included in the analysis for Angora goats, but only female kids were included for cashmere goats. Kids were weighed, and had blood and faecal samples collected at 3 and 5 months of age after exposure to natural worm challenge from the pastures they were grazing. At the 5-month sampling the kids were treated with anthelmintic and a week later each kid was orally dosed with 10,000 infective larvae of *Trichostrongylus colubriformis* (black scour worm). Four weeks later the animals were again weighed, and had blood and faecal samples collected. A week after this, another faecal sample was collected and the animals were treated with anthelmintic to terminate the artificial challenge. Bodyweight and fleece data were collected at the first two annual shearings for Cashmere goats, and for the first 3 6-monthly shearings for Angora goats. For cashmere goats the project dataset included kids from the 2000, 2001, 2002 and 2003 kid drops, while for the Angora goats only the first 3 of these kid drops were included.

WEC was estimated using a modification of the standard McMaster technique, and fleece samples were tested for a range of fibre variables using the OFDA 100 after calibration with AWTO mohair tops. Blood samples were assessed for a wide range of haematological variables using a Cell Dyn® 3500 automated haematology analyser.

Pedigree assignment was tested using DNA tests (PCR) developed and validated by the project, using a set of 14 microsatellite markers. In total, 18 production and parasite associated traits were analysed to estimate (co) variance components, heritabilities and correlations. Where necessary data were transformed prior to analysis. Fixed effects were estimated using a sire model with sire fitted as a random effect. Estimation of variance components for the estimation of genetic parameters utilised four single-trait models fitting the animal as a random effect. Model 1 included significant fixed effects only; Model 2 added the direct additive effect of the animal; Model 3 added the with permanent environment effect of the dam and model 4 added the additive and permanent environmental effect of the animal itself. Genetic parameters were estimated from the model giving the best fit beyond which no further improvement occurred (mostly model 2).

Results and key findings

The project was successfully implemented but drought conditions in 2002, carrying over into 2003 resulted in very low levels of natural worm challenge of kids in these years. The main findings of the project are as follows:

- Both Angora and Cashmere goats are able to mount significant effective immune responses to worm infection by 5 months of age that are capable of limiting subsequent infections.
- Despite this, early vaccination at 1 and 2 months of age with bolus challenges of radiation-attenuated infective worm larvae failed to induce a protective immune response.
- A DNA test for parentage analysis was developed and validated. The panel of microsatellite markers used provided a very high level of power (99.7%) of exclusion of non-true parents. The overall pedigree error rate of 12.7% was within the range reported for other species. As expected ascertaining the dam by mothering up at marking reduced dam pedigree accuracy only relative to mothering up at birth (14.6% and 9.8% error rate respectively).
- Heritability estimates for faecal worm egg count (WEC) were within the range of published estimates for other goat populations and were generally low (range 0.2-0.22), indicating that genetic progress for selection on this trait is likely to be low to moderate and somewhat lower than may be seen in sheep. This is particularly true if selection is based on phenotypic evaluation of sires (as is the case in much of the Cashmere industry). If however, progeny and other relative data

is captured in industry performance recoding schemes such as MOPLAN, accurate EBVs can be calculated, even for traits of low heritability.

- Although the heritability estimates of some parasite-associated traits measure in blood were high or very high insufficient information is available to recommend selection based on these variables in the place of WEC. This is mainly due to the lack of estimable genetic correlations. For several of these variables this report contains the first estimates of heritability made for these variables in goats.
- The heritability estimates for liveweight and fleece traits for cashmere goats were mostly within the range of other published estimates, with moderate to high heritability for most traits. This indicates that rapid genetic progress can be made by genetic selection for these traits although a strong unfavourable genetic correlation between cashmere down weight and diameter requires that both traits be measured for optimum genetic progress.
- The heritability estimates for liveweight and fleece traits for Angora goats tended to be higher than most other published estimates. Once again this indicates that rapid genetic progress can be made by genetic selection for these traits although a strong unfavourable genetic correlation between fleece weight and mean fibre diameter requires that both traits be measured for optimum genetic progress.
- The project has produced the first genetic parameter estimates for the trait of fibre curvature in goats. The trait is moderately heritable in both breeds of goats and has a strong negative genetic correlation with fibre diameter suggesting that crimp frequency can be used as a marker for fibre diameter in both breeds of goat.
- Genetic associations between parasite-associated traits and production traits were non-estimable. Phenotypic associations were in the expected directions for both breeds, and did not suggest that selection for parasite-associated traits would lead to negative effects on production traits.

Implications for relevant stakeholders

The major implications of this project are that it has provided an additional set of genetic parameter estimates for Australian Cashmere and Angora goats to complement those of Pattie and Restall (1991) and Gifford et al (1991) for cashmere goats, and Gifford et al (1989) for Angora goats. The parameter estimates were obtained in current commercial animals in commercial herds in typical goat production areas. For the first time, traits associated with worm infection and resistance to it were included in the analysis and the trait of fibre curvature was also included. Also for the first time the analysis was based up pedigree data validated by DNA testing. The findings support other studies in showing that rapid genetic progress can be made in selection for fleece traits and moderate progress for bodyweight. Selection for resistance to worm infection is likely to result in slow genetic progress with no evidence of negative correlated responses in production traits.

The project has also developed and validated a panel of microsatellite markers that can be used for parentage analysis in goats. An attempt to control worm infections using early oral vaccination with radiation-attenuated larvae was unsuccessful and should not be pursued further.

Recommendations

- The inclusion of selection for resistance to worm infection on the basis of individual WEC measurements will result in slow genetic gains in this trait. Inclusion of the trait should only be considered where additional information from relatives would allow accurate estimates of EBV (eg. within MOPLAN). Selection for this trait based on sire measurements alone is unlikely to be worthwhile due to the low heritability. Inclusion of WEC in the selection index requires careful evaluation as not only will the response to selection be relatively slow, but applying significant selection differential to WEC will reduce the selection differential able to put on traits of high heritability and economic value such as fleece traits.
- Given the lack of estimable genetic correlations between WEC and production traits in this dataset, published values from overseas studies will need to be used. Based on such studies the genetic correlation between WEC and liveweight is not different from 0 but tending towards positive, that between WEC and fleece variables is weakly positive and that between WEC and PCV is moderately to strongly negative.
- Given environmental variation in the level of parasite challenge, individual tests of WEC should always be preceded by a bulk WEC test indicating significant worm burdens in the herd (eg. at least 500 epg in regions with *Haemonchus contortus* present).
- Given the moderate to very high heritabilities of some parasite-associated blood traits and the increasing availability of automated haematology analysis, support should be given to determining genetic correlations between these traits and WEC to more fully evaluate whether they are viable alternative measures of resistance and/or resilience to GIN. The physiological consequences of extreme values for blood constituents also need to be determined.
- Given the difficulty of using selection for genetic resistance to control worms, research emphasis should be applied to other control measures suitable for inclusion in an integrated worm control program in goats. Two approaches supported by the findings of this project are detailed below.
  - The evidence in this study that immune regulation of worm burdens is related to the level of prior worm challenge indicates that parasite control methods that rely on maintaining some parasite burden in the host are more likely to be successful than those that totally suppress worm infections intermittently. Tactical worm control based upon close monitoring of WEC and treatment intervention only when the WEC exceeds a given threshold based on worm species involved, animal condition, feed availability and climatic conditions is likely to maintain immunity in the population and help regulate infections. Such an approach has been successfully used in sheep (Kahn *et al.*, 2006; Scrivener *et al.*, 2006).
  - The marked year to year variation in worm burdens associated with drought conditions reinforces the susceptibility of the free-living stages of gastro-intestinal nematodes to adverse environmental conditions (Review: O'Connor *et al.*, 2006) and our ability to exploit this weakness using grazing management practices such as alternate grazing with cattle (Southcott and Barger, 1975) and intensive rotational grazing (Colvin *et al.*, 2007).
- fEarly oral vaccination of kids with bolus doses of irradiated worm larvae cannot be recommended as a practical worm control method and there is little evidence that it is likely to ever be so.
- The findings of this project would support the use of an on-farm measure of fibre curvature (eg. "crimp" frequency) as an indirect measure of fibre fineness in both Angora and Cashmere goats. However full objective measurement in a laboratory will maximise genetic progress.
- The project results should be published in the scientific literature and be made widely available to industry.

1. Introduction and Review

1.1 Project Overview

This project was designed to provide genetic parameter estimates for worm resistance traits and production traits in Australian Cashmere and Angora goats, and phenotypic and genetic correlations amongst them. At the time on initiation of this project (2000), estimates of genetic parameters for worm resistance were readily available for sheep and selection for increased resistance to worm infection had become commonplace in sheep breeding programs as a result. Estimates were also beginning to be published for goat populations in other countries but not Australia. The project therefore had the important task of providing the first heritability estimates for worm resistance traits in Australian goats (specifically Australian fibre goats), and correlations with production traits. Subsidiary objectives were:

- to evaluate the use of irradiated larval vaccines for control of worm infections in goats;
- to develop a DNA marker set for parentage analysis and to have the quantitative genetic analysis based on DNA verified pedigrees;
- to provide an evaluation of potential markers for worm infection or resistance in goats other than faecal worm egg count; and
- to provide information on the effects of worm infections in goats of the two breeds in the New England environment.

The project animals were commercial Cashmere and Angora goats on two properties near Barraba in Northern NSW.

1.2 The problem of gastro-intestinal nematode infection in goats

1.2.1 Species, distribution and lifecycle

Parasitic disease caused by helminths (worms) is of primary importance in small ruminant production systems worldwide. There are three groups of helminths, nematodes (roundworms), cestodes (tapeworms) and trematodes (flukes). Gastro-intestinal nematodiasis (GIN, roundworm infection) in sheep and goats is ubiquitous in all but arid and semi-arid environments and is associated with production loss, treatment costs and mortality. There is evidence that goats are more susceptible than sheep to nematode infection and never develop the same level of immunity that sheep do (Le Jambre and Royal, 1976; Le Jambre, 1984; Pomroy and Charleston, 1989). This is possibly due to the evolutionary history of goats as browsers, rather than grazers, which would have limited exposure to infective worm larvae, which are rarely found above 20 cm in the pasture sward. This means that goats are heavily reliant on use of anthelmintics to control nematode infections and as for sheep there is a severe problem with anthelmintic resistance amongst the main species of nematodes affecting sheep and goats, both in Australia (Besier et al 2003) and overseas (Pomroy, 1996; Hoste et al 2002). Indeed there is a widespread view amongst parasitologists that anthelmintic resistance problems in goats are greater than in sheep, possibly due to the higher requirement for treatment in adults, and also due to goats' ability to metabolise and inactivate anthelmintics faster than sheep (Rickard, 1990).

The major gastrointestinal nematodes affecting sheep and goats in Australia are shown in Table 1.1.

H. contortus is the most pathogenic being a large haematophagous (blood sucking) parasite than can induce severe anaemia and death. *T. colubriformis* and *Ostertagia* spp. are less pathogenic than *H. contortus* and cause weakness, reduced productivity, progressive weight loss and diarrhoea. The relative importance of each parasite species varies with rainfall amount and seasonal distribution (Donald *et al.*, 1978). Worm problems are greatest in areas with an annual rainfall of 350mm or greater. *H. contortus* is susceptible to cold and desiccation and predominates in the northern summer rainfall zones while *Ostertagia* spp. are tolerant of cold and desiccation and so are abundant in the

southern winter rainfall zones (O'Connor *et al.*, 2006). *T. colubriformis* has wide temperature and desiccation tolerance and is important in all zones. Depending on seasonal conditions any of the three parasites may be important in each of the zones.

Table 1.1 Major gastrointestinal nematodes of sheep and goats in Australia (adapted from Donald *et al.*, 1978).

Specific name	Common name	Habitat in host	Disease mechanisms
Haemonchus contortus	Barber's pole worm	Abomasum	Loss of blood, protein, and appetite (mild)
Trichostrongylus spp.	Black scour worm	Small Intestine	Loss of appetite (severe), disturbed protein metabolism, diarrhoea.
Ostertagia (Teladorsagia) circumcincta	Small brown stomach worm	Abomasum	Loss of appetite (severe), disturbed protein metabolism, diarrhoea.
Nematodirus spp.	Thin necked intestinal worm	Small Intestine	Loss of appetite, disturbed protein metabolism diarrhoea.
Oesophagostomum spp.	Large bowel/Nodule worm	Large Intestine	Loss of appetite, disturbed protein metabolism
Chabertia spp.	Large mouthed bowel worm	Large Intestine	Loss of blood, appetite and disturbed protein metabolism

The life cycle of nematodes consists of a parasitic phase in the host and a free-living phase in the environment. Generally, the life cycle consists of seven stages, the egg, four larval stages (L1, L2, L3, L4) and two adult stages. Sometimes the sexually immature adult stages are called L5. The adult female worm mates with a male and lays fertile eggs that leave the goat's intestinal tract with the faeces. Depending on temperature and moisture availability, the eggs hatch to L1 in the faeces. These feed on bacteria in the faeces and moult to the L2 stage which also feeds on bacteria within the faecal pellet. The L2 undergoes a partial moult to the L3 stage which is completely ensheathed, precluding further feeding. It will therefore die if not ingested by the host. This stage leaves the faecal pellet and migrates on to nearby herbage awaiting ingestion by the host. Once ingested by the host, L3 exsheaths in the rumen and then moults to L4. The L4 enter the abomasal or intestinal mucosa and undergo further growth through L5 until sexual maturity in appropriate section of the host gastrointestinal tract. Sexually mature females produce eggs. The duration of full lifecycle varies widely depending on environmental conditions. However the prepatent period in the host (from ingestion of L3 to adult egg production) is less variable ranging from 19-21 days for *H. contortus* and 20-25 days for *T. colubriformis* (Kaufmann, 1996). Reproduction occurs only in the host and parasite numbers cannot increase in the environment, the upper limit of environmental numbers being the number of eggs shed in host faeces.

1.2.2 Approaches to control

Worm control strategies can be broadly classified according to their focus: on the pathogen (chemotherapy and anthelmintics), the environment (grazing management, biosecurity), or the animal (vaccination, host nutrition, selection of resistant animals).

Anthelmintic treatment

The use of chemicals that selectively kill helminths (anthelmintics) remains the main means of controlling gastrointestinal nematodiasis worldwide. Because of their comparatively high toxicity, early treatments such as copper sulphate and nicotine sulphate were replaced by phenothiazine in 1940s and 1950s. In the 1960s, two new much safer classes of broad spectrum anthelmintic were introduced and their initial representatives, thiabendazole (of the benzimidazole class) and levamisole, were widely used as an effective control of sheep and goat nematodes (Vlassoff and McKenna, 1994). A number of other benzimidazole compounds with improved features (spectrum of activity, duration of activity) were released in the 1970s and in the 1980s and a third group of anthelmintics, the macrocyclic lactone class (milbemycins and avermectins) compounds were introduced (Vlassoff and

McKenna, 1994). Hoste *et al.* (2002) conducted surveys on anthelmintic resistance over the previous 15 years on dairy goat farms in France and found a constant increase in the prevalence rate of anthelmintic resistance, particularly, benzimidazole resistance (83 to 100%), which involved the most prevalent nematode species of goats such as *T. colubriformis*, *H. contortus* and *Teladorsagia circumcincta*. The prevalence of anthelmintic resistance in goats is high relative to sheep (Pomroy, 1996) causing concern in the sheep industry as the same parasites generally affect the two species and they are controlled by the same anthelmintics. The development of resistance has been associated with a high frequency of anthelmintic treatment (Pritchard, 1990), however the issue of what factors predispose to selection for resistance is complex and includes the level of unselected refugia on pasture at the time of anthelmintic treatment (van Wyk, 2001). Effective quarantine treatment of all purchased animals is also of critical importance to prevent the inadvertent introduction of multiple-resistant worm strains onto a property.

Grazing management

This worm control method aims to reduce or eliminate parasite infection by providing animals with a clean or relatively uncontaminated pasture or by preventing ingestion of large numbers of worm larvae from heavily contaminated pasture. There are several strategies used to avoid pasture contamination. Pasture spelling can greatly reduce parasite numbers in deferred grazing systems if the pasture is rested for at least 6 months during the cool season and 3 months in the warm part of the year (Scarfe, 2003) but this is often impractical in terms of optimum pasture utilisation. On the other hand, very intensive rotational grazing systems in which animals graze pasture for 2-5 days only with long rest periods (35-80, days depending on time of year) have been shown to be particularly effective in controlling infections with *H. contortus* in goats in tropical regions (Barger *et al.*, 1994) and sheep in temperate regions (Colvin *et al.*, 2007). An alternative approach is grazing with different livestock species that do not share the same parasites. Most parasites of sheep and goats do not cross infect cattle (with some exceptions) and this can be successfully exploited by rotating pastures with the different species (e.g. Barger and Southcott, 1978). This effectively makes pasture spelling practical by having an alternative host graze the pasture during the long spelling periods required for significant larval die off to occur. A combination of chemical treatment and grazing management is the basis of "Smart grazing" for worm control in sheep over summer in winter rainfall areas (Niven *et al.*, 2001). It is a way of more reliably preparing clean pasture for spring-born weaners to graze during their first winter. During the preparation period, dry sheep are grazed at 2.5-3 times the normal stocking rates for one month after each of two summer drenches (usually November & February). The paddock is destocked during the intervening period, and so the average stocking rates over summer are unchanged compared to conventionally prepared weaner paddocks (those grazed continuously by mature wethers). Smart grazing is currently being adapted for northern summer rainfall regions under the Australian Wool Innovation funded project EC306 "Integrated Parasite Management in Sheep (IPM-s)" project. Practical application of different parasite management approaches including grazing management is feature of this project (see http://www.wool.com.au/Animal_Health/Integrated_parasite_management_/IPM_-_sheep/page__2244.aspx)

Nutritional strategies

Manipulation of host nutrition is a well-documented means of improving control of GIN infections. Protein supplementation can increase the rate of acquisition of immunity and resistance to reinfection (Coop and Holmes, 1996). Goats receiving a high protein diet had decreased faecal egg counts compared with those with normal protein diet during lactation (Hoste *et al.*, 2005). Hoste *et al.* (2005)'s review showed that the response of goats to supplementary feeding was characterized by an improvement in resilience, while effects on host resistance were less evident. Resilience is ability of the host to maintain production in the face of infection with parasites while resistance is the ability to resist or throw off infection. However access to urea-mollases blocks improved both resilience and resistance in gazing East African goat kids in an environment dominated by *Haemonchus contortus* (Waruiru *et al.*, 2004). These effects are broadly consistent with those seen in sheep for which there is an extensive literature and many reviews (e.g. Coop and Holmes, 1996; Van Houtert and Sykes, 1996; Walkden-Brown and Kahn, 2002).

Vaccination

Vaccination has long been considered as a promising alternative for control of GIN infections in sheep and goats. This is based on the observation that immune-based resistance to infection develops in sheep and goats, and almost complete resistance to infection can be induced in sheep under experimental conditions of constant high challenge, although this is not replicated in the field. Vaccines may be live (usually irradiated to preclude full patency), incompletely defined mixtures of parasite antigens or defined vaccines using known antigens. Ruiz *et al.* (2004) found that the faecal egg count of goats vaccinated with *Haemonchus* cysteine proteases (61±2.9) was significantly lower ($P<0.001$) than that of unvaccinated goats (550±13.5). The majority of attempts to develop commercial vaccines for GIN have been focused on identifying protein antigens, including "hidden" antigens such as those of the parasite gut, which the host is not normally exposed to. Proteins isolated from the surface of the gut of GIN, especially *H. contortus*, have proved to be effective protective antigens and several are being progressed towards recombinant protein-based vaccines (Knox and Smith, 2001). Vervelde *et al.* (2003) showed that vaccination with excretory/secretory (ES) glycoproteins in alhydrogel induced significant specific IgG, IgA, and IgE antibody responses and was effective in immunizing lambs against *H. contortus*. Vaccination with irradiated larvae has also been used to induce protective immunity in sheep and this area has been well reviewed (Bain, 1999).

Breeding for resistance or resilience to GIN

There are two important concepts regarding the host response to parasitism: resistance (or susceptibility) and resilience (or tolerance). Resistance is the ability of the host to resist infection while resilience is the ability of the host to limit the pathological and production consequences of infection. Breeding for resistance to gastro-intestinal nematodes has been demonstrated to be highly effective in decreasing worm burdens and worm egg output in sheep. Evidence for genetic variation in worm burdens has been found both within, and between, host populations and parasite species. Resistance to nematodes can be quantified by directly measuring faecal worm egg counts (WEC) or indirectly by measuring specific antibody responses to infection or blood cell traits after an extended period of exposure to natural or artificial challenge with selected GIN species. By contrast, selection for resilience can be done based on direct measurement of the production traits such as growth and fibre production traits and this will have been taking place by default within the industry for some time. Most studies have been concerned with investigating the feasibility and implications of breeding for resistance to GIN (Baker *et al.*, 2001; Woolaston and Windon, 2001; Vagenas *et al.*, 2002) rather than for resilience (Albers *et al.*, 1987; Bisset *et al.*, 1994). Whether it is preferable to improve resistance or resilience is controversial. There have been some arguments that selecting resistant hosts is a direct challenge to the parasites' existence and as parasites can adapt to anthelmintics, they can probably also respond to selection by genetically changed hosts (Le Jambre, 1993). Resilience on the other hand allows worms and host to co-exist, without necessarily placing any selection pressure on the parasite (Woolaston and Eady, 1995). Gray (1995) noted that breeding programs should not have resistance to parasites as their sole objective and that it is important to take into account genetic relationships between disease resistance and other traits such as live weight, fleece weight or resistance to other diseases, as well as the economic weights of all the traits in the breeding objective. Many authors (e.g. Baker *et al.*, 1999; Piper *et al.*, 1978; Mandonnet *et al.*, 2001; and Morris *et al.*, 1997) have reported existence of genetic variation to GIN within and between sheep or goat breeds, which is the essential element for breeding programs.

1.2.3 Mechanisms of host resistance and resilience to nematodes

The immune response to internal nematodes is based on the establishment of different immune cell effector populations (e.g. neutrophil, eosinophil, and basophil granulocytes, monocytes, mast cells, megakaryocytes, and lymphocytes) and implementation of antibody and cell-mediated defence mechanisms that impair the establishment and well-being of nematodes (Balic *et al.*, 2000). Immunity generally manifests itself as one or more of:

- reduced establishment of incoming L3;
- reduced size, fecundity and lifespan of adult nematodes;
- expulsion of the adult nematode population.

Defence mechanisms are generally highly complex and involve a number of components of innate and acquired immunity. Innate immunity is rapid and involves generalized non-specific responses against a broad range of pathogens without prior exposure to the pathogen, whereas adaptive immunity or resistance is a specific immune response to a pathogen and it develops more slowly than innate immunity. Adaptive immunity involves recognition of specific antigenic epitopes and development of a very specific immune response (antibody mediated, cell-mediated or both) with associated development of memory cells (T and B lymphocytes) which provide immunological memory and an improved or anamnestic response to subsequent challenge from the same parasite. A resistance mechanism that is effective against one particular nematode species and stage of development may be ineffective against a different development stage of the same species or against the same stage of another nematode species (Balic *et al.*, 2000). Despite this specificity of response, resistance to one nematode species is highly correlated with resistance to another in sheep (Gruner *et al.*, 2004). Thus selection for resistance to challenge by one nematode species will result in correlated selection for resistance to the other key nematode species.

The heritability of WEC in lambs naturally infected with *Teladorsagia circumcincta* increases with age indicating that genetic variation is due to an acquired response rather than an innate response (Stear *et al.*, 1997; Stear *et al.*, 1999). The results of Stear *et al.* (1999) indicate that genetic variation in faecal egg counts in lambs is due to variation in adult female worm length and fecundity rather than worm number, although in adult sheep both worm number and fecundity are regulated. In the UNE Golden Ram herd, Gill (1991) tested immune responsiveness of genetically resistant sheep to secondary infection with *H. contortus* and found that the genetically resistant line had significantly lower WEC and exhibited significantly higher circulating specific antibody (IgG) levels and mucosal eosinophilia in response to a secondary challenge infection than the non-resistant line. Again genetic resistance resulted from the expression of acquired immune responses rather of innate responses. Eosinophil responses in Merino sheep selected for either increased resistance (resistant line) or susceptibility (non-resistant line) to *T. colubriformis* infection after irradiated larval (L3) vaccination were significantly higher in the resistant line and associated with lower WEC (Dawkins *et al.*, 1989). Strain and Stear (2001) suggested that in lambs infected with *H. contortus*, secretion of IgA is a major defence mechanism because it suppresses worm growth and fecundity. IgA responses were greater to L3 than L4 larvae of *H. contortus*. The authors also found that protein supplementation had positive effects on magnitude of the IgA response to H. contortus. However, a diversity of immune mechanisms exists, and IgG, IgE, eosinophils and globular leucocytes in the gut have all been shown to be associated with effective immune responses in various studies.

In sheep there is peri-parturient relaxation of immunity to gastro-intestinal nematodes that extends well into the lactation period and results in a peri-parturient rise in WEC (Connan, 1967). This is also evident in goats with does that had given birth and were lactating having significantly higher WEC than dry does (Vlassoff *et al.*, 1999). However, in goats, there is less development of resistance with age and exposure so there is less divergence in level of infection between young and adult animals. Le Jambre and Royal (1976) compared the worm burdens in 15-month-old grazing Merino sheep and Angora goats for over a 4 month period and found that the ability of Angora goats to develop an immune response against worms of different species was poor compared with those of Merino sheep.

1.3 Genetic and phenotypic parameters for worm resistance traits

1.3.1 Heritability of faecal worm egg count and packed cell volume

A number of studies have reported heritability estimates for resistance to nematodes in goats (Table 1.2). The estimates of heritability for faecal worm egg counts (WEC) range from 0.04 to 0.37. The heritability of WEC in Fijian and Saanen goats in New Zealand appears to be very low. The highest estimates (0.37) have come from Scottish and Greole goat populations. In Greole goats, heritability estimates for WEC increase from 0.14 to 0.33 with age, whereas such a trend was not apparent for Small East African goats. Mandonnet *et al.* (2001) reported that the highest heritability estimates of WEC (0.37) at weaning were reduced by 0.17 after including maternal effects. Generally, the trend towards increasing heritability of WEC with age is inconsistent possibly due to the relatively small data sets involved. Goats are more susceptible to gastrointestinal nematodiasis than sheep (Miller *et al.*, 2006) and the heritability estimates for WEC tend to be somewhat lower than those for sheep (Baker *et al.*, 2001). The explanation for a reduced immune response in goats than sheep given in some studies is that goats are browsers rather than grazers, thus have less exposure to nematode larvae in the pasture and have been facing less intense natural selection for resistance to worms (Baker *et al.*, 2001).

The PCV of goats has been found to be a low to moderately heritable trait in regions where *Haemonchus contortus* is a major parasite. Estimates of heritability for PCV have ranged from 0.11-0.33 and appear to be in the same range as in sheep (Baker *et al.*, 1998; Baker *et al.*, 2001). Baker *et al.* (2001) pointed out that the maternal effects were not significant or very small at all measurement ages for WEC, but were significant at some measurement ages for PCV.

Table 1.2 Heritability estimates for faecal egg counts (WEC) and packed cell volume (PCV) in goats

Traits	heritability	Age (months)	Breed of goats	Country	Reference
WEC	0.05*	>12	Saanen	New Zealand	(Morris *et al.*, 1997)
WEC	0.04	<12	Fijian goat	Fiji	(Woolaston *et al.*, 1992)
WEC	0.08	>12	Fijian goat	Fiji	(Woolaston *et al.*, 1992)
WEC	0.37	3	Greole	India	(Mandonnet *et al.*, 2001)
WEC (PCV)	0.14 (0.20)	4	Greole	India	(Mandonnet *et al.*, 2001)
WEC (PCV)	0.17 (0.10)	8	Greole	India	(Mandonnet *et al.*, 2001)
WEC (PCV)	0.33 (0.33)	10	Greole	India	(Mandonnet *et al.*, 2001)
WEC (PCV)	0.15 (0.25)	4.5	Small East African	Kenya	(Baker *et al.*, 2001)
WEC (PCV)	0.16 (0.11)	8	Small East African	Kenya	(Baker *et al.*, 2001)
WEC (PCV)	0.12 (0.29)	10	Small East African	Kenya	(Baker *et al.*, 2001)
WEC	0.37	NA	Scottish Cashmere	Scotland	(Jackson, 2000)
WEC	0.17	NA	Scottish Cashmere	Scotland	(Vagenas *et al.*, 2002)
WEC	0.32*	NA	Scottish Cashmere	Scotland	(Vagenas *et al.*, 2002)

WEC= transformed faecal worm egg count; PCV=packed cell volume; NA= not available; * = mean of more than 3 records

1.3.2 Repeatability of faecal worm egg count and packed cell volume

Repeatability is a measure of the similarity between multiple measures and represents the upper limit for heritability. Estimates of heritability based on repeated measurements are expected to be higher, because the amount of variation due to non-genetic permanent environment that appears in phenotypic variance reduces as the number of records increases (Falconer and Mackay, 1996). Compared with a single measurement, the mean of two or more measurements therefore has a higher heritability $h_n^2 = nh^2/(1+r(n-1))$ Where h_n^2 is the heritability of the mean of *n* repeated measurements, h^2 is the heritability of a single measurement, and *r* is the repeatability (Falconer and Mackay, 1989). For example, Vagenas *et al.* (2002) in studies of Scottish Cashmere goats found that the heritability of a single WEC was 0.17 while the heritability of the mean WEC of 4-11 measurements was 0.32. Woolaston *et al.* (1992) in studies of Fijian goats found a permanent environmental effect of zero, which indicates that the repeatability was not higher than heritability. Similarly, the permanent environment effects among Small East African goats at 4.5, 8 and 10 months of age were non-significant for logWEC (LWEC) or PCV (Baker *et al.*, 2001). In pen-tested Merino sheep at 3, 5, 7, 9, and 11 weeks of ages, Woolaston and Windon (2001) estimated heritability for LWEC, and found that repeatability for LWEC of five counts (0.39) was not greater than the heritability of any single LWEC (range 0.33 to 0.39). On the other hand, in sheep infected with *H. contortus*, the repeatabilities for PCV and LWEC (0.51 and 0.46 respectively) were high compared with heritability estimates (0.15 and 0.31, respectively) (Vanimisetti *et al.*, 2004).

1.3.3 Genetic and phenotypic correlations

Estimates of genetic correlation express the extent to which genes affect two different traits. Correlation estimates are essential in assessing the suitability of indicator traits as indirect criteria in breeding programs (O'Meara and Raadsma, 1995). On the other hand, the phenotypic correlation indicates the extent of associations between two traits.

Correlations between resistance traits

Highly negative genetic correlations (0.56-0.79) between WEC and PCV and positive genetic correlations (0.37-0.58) between WEC and eosinophil concentration have been reported in goats under tropical conditions (Baker *et al.*, 2001; Mandonnet *et al.*, 2001). Costa *et al.* (2000) also found a highly significant and negative relationship between transformed worm faecal egg counts and PCV or haemoglobin (genetic correlations: -0.45 and -0.53, respectively) in goats infected with *H. contortus* in Brazil. Strong negative genetic correlations between WEC and IgA activity or eosinophil counts were found in Scottish Blackface lambs infected with *Teladorsagia circumcincta* (-0.78, s.e. 0.18 and -0.97, s.e. 0.11, respectively) as well as between resistance-related traits and worm length or worm burdens (Davies *et al.*, 2005). However, the accuracy of the estimates is generally not very high.

Correlations between resistance and production traits

Genetic correlations between WEC or PCV and live weight in Greole goats (Mandonnet *et al.*, 2001) were non-significant or slightly favourable, whereas in goats under African conditions (Baker *et al.*, 2001), the genetic correlation between WEC and live weight was not significant, and the phenotypic correlations were negative, averaging -0.10. Genetic and phenotypic correlations between PCV and live weight at different ages were all positive and moderate (Baker *et al.*, 2001). For crossbred cashmere-producing goats in the UK, production traits (cashmere length, yield, and diameter, and live weight) were not genetically and phenotypically correlated with WEC (not significantly different from zero). Morris *et al.* (1997) reported that the genetic correlations between WEC and milk yield or yield of fat and protein in milk for Saanen does in New Zealand were moderately negative (-0.21 and -0.17), and the phenotypic correlations were also negative but not significant (-0.07 and -0.07).

1.4 Genetic and phenotypic parameters for production traits

1.4.1 Heritability estimates for production traits

The estimate of the heritability depends on the magnitude of all components of variance. The literature shows that substantial variation exists between heritability estimates for most production traits

obtained from different studies of fibre producing goat populations or breeds. All the genetic components of variance are influenced by gene frequencies and therefore the magnitude of genetic components (heritability) may differ from one population to another, according to the past history of the population (Falconer and Mackay, 1996). However, these differences could also be due to limited accuracy resulting from a small number of measurements, to population size, to sampling errors or to possible biases in the estimates of heritability.

Heritability estimates fleece traits and live weight in Angoras

The range of heritability estimates for live weight, greasy fleece weight, and some other fleece characters for different populations of Angora goats provided by different authors is summarized in Table 1.3. From this it can be said that greasy fleece weight (GFW) is moderately to highly heritable with the highest estimates reported for Australian and Texan Angora goats (0.45 and 0.40) and the lowest reported for South African Angora goats (0.19 and 0.22). Estimates of heritability for mean fibre diameter (MFD) were variable, ranging from 0.12 to 0.51. Again, the Australian and Texan goat populations had very similar estimates for MFD, but they were the lowest among the studies (0.14 and 0.12), the highest estimates (0.51) being in studies on French and New Zealand Angora goats with South African goats intermediate (0.26 and 0.30). Staple length had low heritability in French and Australian Angora goats. Allain and Roguet (2005) reported that heritability estimates for kemp and medullation content were moderate. However, heritability estimates for medullation and kemp content for New Zealand and Argentinean Angora goats were not significantly different from zero. The heritability estimates of live weight (LWT) in the referred studies varied widely ranging from 0.13 in Australian Angora goats to 0.50 in Texan Angora goats. The heritability estimates for LWT in the other Angora populations were moderate.

Heritability estimates for fleece traits and live weight in Cashmere goats

Published heritability estimates are summarised in Table 1.4. Heritability estimates for greasy fleece weight and live weight are lower than for the other cashmere traits. Cashmere length and diameter are highly heritable, whereas the estimates for cashmere yield and cashmere weight are moderate to high. Pattie and Restall (1989) in studies of Australian feral goats found that the heritability of all cashmere traits was high, ranging from 0.29 to 0.90 and that cashmere weights were extremely variable (CV=42%). The moderate to high heritability estimates indicate that all the cashmere traits would respond effectively to individual or mass selection.

Table 1.3. Literature estimates of heritability for production traits in Angora goats

Character	Heritability	Country	Reference
Greasy fleece weight (GFW)	0.45	Australia (South)	(Gifford *et al*., 1991)
	0.19	South Africa	(Snyman and Olivier, 1996)
	0.22	South Africa	(Snyman and Olivier, 1996)
	0.40	USA (Texas)	(Shelton and Bassett, 1970)
	0.26	Argentina	(Taddeo *et al.*, 1998)
	0.19	France	(Allain and Roguet, 2003)
	0.25	France	(Allain and Roguet, 2005)
	0.25 and 0.36	New Zealand	(Nicoll *et al.*, 1989)
Mean fibre diameter (MFD)	0.14	Australia (South)	(Gifford *et al*., 1991)
	0.26	South Africa	(Snyman and Olivier, 1996)
	0.30	South Africa	(Snyman and Olivier, 1996)
	0.12	USA (Texas)	(Shelton and Bassett, 1970)
	0.19	Turkey	(Yalcin *et al.*, 1979)
	0.33	Argentina	(Taddeo *et al.*, 1998)
	0.32	France	(Allain and Roguet, 2003)
	0.51	France	(Allain and Roguet, 2005)
	0.51	New Zealand	(Nicoll *et al*., 1989)
Staple length (STL)	0.13	Australia (South)	(Gifford *et al.*, 1991)
	0.18	France	(Allain and Roguet, 2003)
Percent Medullation (%MED)	0.02	New Zealand	(Nicoll *et al*., 1989)
	0.10	Argentina	(Taddeo *et al.*, 1998)
	0.23	France	(Allain and Roguet, 2005)
Percent Kemp (%KEMP)	0.16	New Zealand	(Nicoll *et al*., 1989)
	0.32	France	(Allain and Roguet, 2005)
Live weight (LWT)	0.13	Australia (South)	(Gifford *et al.*, 1991)
	0.29	South Africa	(Snyman and Olivier, 1996)
	0.35	South Africa	(Snyman and Olivier, 1996)
	0.50	USA (Texas)	(Shelton and Bassett, 1970)
	0.24	Turkey	(Yalcin *et al.*, 1979)
	0.24	New Zealand	(Nicoll *et al*., 1989)

Estimates of maternal effects for production traits

Since the dam contributes environmentally (e.g., by producing milk) to the performance of the progeny, it is important to estimate the maternal effects (either genetic or environmental effects in cause), particularly for traits measured before and a shortly after weaning. If the genetic component of maternal effects is negligible, then maternal effects can be ignored in genetic improvement programs although they need to be accounted for in genetic evaluation. If they are significant, they need to be considered when constructing breeding programs. However, for estimating maternal effects, it is essential to have adequate information (e.g. multiple offspring per dam, genetic relationships among dams if estimating a genetic component, and dam performance records when estimating the correlation between additive genetic and maternal genetic effects). Maternal effects for all cashmere characteristics in the studies of Scottish Cashmere goats were negligible and not significant, but those for live weight were significant (Bishop and Russel, 1996; Vagenas *et al.*, 2002). The heritability estimate for live weight changed from 0.50 to 0.22 after fitting maternal effects. Maternal effects for fleece traits as well as live weight were found to be non-significant in Inner Mongolian Cashmere goats and Argentinean Angora goats (Taddeo *et al.*, 1998; Zhou *et al.*, 2002).

Table 1.4. Range of estimates of heritability and genetic and phenotypic correlations for live weight and cashmere traits in Cashmere goats

Traits	Heritability (range)	Correlation (range)	
		Genetic	Phenotypic
Live weight	0.10 to 0.35		
Greasy fleece weight		0.17 to 0.17	0.21 to 0.36
Cashmere yield		-0.24 to -0.39	-0.07 to -0.12
Cashmere weight		-0.13 to -0.34	-0.08 to 0.12
Cashmere diameter		-0.06 to -0.25	0.12 to 0.23
Cashmere length		-0.31 to 0.02	-0.05 to 0.08
Greasy fleece weight	0.14 to 0.45		
Cashmere yield		-0.23 to 0.75	-0.11 to 0.14
Cashmere weight		0.14 to 0.98	0.41 to 0.77
Cashmere diameter		-0.04 to 0.44	0.18 to 0.45
Cashmere length		0.05	0.04
Cashmere yield	0.23 to 0.90		
Cashmere weight		0.85 to 0.92	0.56 to 0.80
Cashmere diameter		0.00 to 0.81	0.25 to 0.40
Cashmere length		0.87	0.57
Cashmere weight	0.20 to 0.68		
Cashmere diameter		0.04 to 0.81	0.39 to 0.52
Cashmere length		0.90	0.65
Cashmere diameter	0.32 to 0.99		
Cashmere length		0.50 to 0.60	0.32 to 0.35
Cashmere length	0.57 to 0.70		

Sources: Gifford *et al.* (1989); Pattie and Restall (1991b); Bigham *et al.* (1993); Bishop and Russel (1996); Zhou *et al.* (2002); Vagenas *et al.* (2002).

Repeatability estimates for production traits

Estimates of repeatability of production traits in Cashmere goats over repeated shearings were moderate to high and varied between 0.39 and 0.63 (Pattie and Restall, 1991b; Zhou *et al.*, 2002). Estimates of repeatability for GFW and MFD in Argentinean and South African Angora were found to be reasonably high, 0.41 and 0.64 for GFW and 0.68 and 0.70 for MFD, respectively (Taddeo *et al.*, 1998; Snyman and Olivier, 1999). In addition, Australian Angora goats reported by Gifford *et al.* (1991) demonstrated moderate repeatability of GFW, CFW and %MED (range: 0.39-0.45) and low repeatability for MFD, %KEMP and LWT (range: 0.18-0.30).

1.4.2 Genetic and phenotypic correlations amongst production traits

The changes that will occur in the offspring of selected animals are indicated by genetic correlations, whereas phenotypic correlations express the associations between traits within the same animals. Understanding the mechanisms involved in changes in the production traits leads to improved selection indices and ultimately higher financial returns (Merchant and Riach, 2003). Fleece traits have been found to be strongly correlated but unfavourable relationships exist among some desirable traits which affect the potential for genetic improvement of Angora and Cashmere goats.

Angora goats

From Table 1.5, it can be seen that moderate to strong, but positive correlations between GFW, MFD, STL and LWT exist amongst the different Angora strains. The unfavourable genetic and phenotypic relationships between fleece weight and live weight, and fibre diameter demonstrate that selection for high fleece weight and/or increased live weight alone will increase fibre diameter. For Argentinean Angoras, selection for high fleece weight would result in a decrease of medullated fibre content, whereas this appears the opposite of what has been observed in New Zealand Angoras. Medullated fibres and kemp appear to be positively associated both phenotypically and genetically. Medullation has a negative genetic correlation with fleece weight, but positive phenotypic association.

Table 1.5. Literature estimates of genetic and phenotypic correlations between production traits in Angoras

Traits	Correlation		Country	Reference
	Genetic	Phenotypic		
GFW & MFD		0.37	Australia (South)	(Gifford *et al.*, 1991)
	0.55	0.57	South Africa	(Snyman and Olivier, 1996)
	0.75	0.22	USA (Texas)	(Shelton and Bassett, 1970)
	-0.28	0.14	Turkey	(Yalcin, 1982)
	0.51		Argentina	(Taddeo *et al.*, 1998)
	0.35	0.64	France	(Allain and Roguet, 2003)
	0.55	0.98	New Zealand	(Nicoll *et al.*, 1989)
GFW & STL		0.40	Australia (South)	(Gifford *et al.*, 1991)
	0.39		USA (Texas)	(Shelton and Bassett, 1970)
	-0.33		Turkey	(Yalcin, 1982)
	0.25	0.11	France	(Allain and Roguet, 2003)
GFW & %MED	-0.72		Argentina	(Taddeo *et al.*, 1998)
		0.14	Australia (South)	(Gifford *et al.*, 1991)
	-0.04	0.49	New Zealand	(Nicoll *et al.*, 1989)
MFD & STL		0.28	Australia (South)	(Gifford *et al.*, 1991)
MFD & %MED		0.39	Australia (South)	(Gifford *et al.*, 1991)
	0.32	0.54	New Zealand	(Nicoll *et al.*, 1989)
	-0.18		Argentina	(Taddeo *et al.*, 1998)
MFD & %KEMP	-0.02	0.04	New Zealand	(Nicoll *et al.*, 1989)
%MED & %KEMP	0.07	0.84	New Zealand	(Nicoll *et al.*, 1989)
	0.70		France	(Allain and Roguet, 2003)

LWT & GFW		0.39	Australia (South)	(Gifford *et al.*, 1991)
	0.67	0.57	South Africa	(Snyman and Olivier, 1996)
	-0.27	0.10	USA (Texas)	(Shelton and Bassett, 1970)
	0.17	0.19	Turkey	(Yalcin, 1982)
	0.54	-0.24	New Zealand	(Nicoll *et al.*, 1989)
LWT & MFD		0.23	Australia (South)	(Gifford *et al.*, 1991)
	0.56	0.55	South Africa	(Snyman and Olivier, 1996)
	0.00	0.13	USA (Texas)	(Shelton and Bassett, 1970)
	0.14	0.26	Turkey	(Yalcin, 1982)
	0.37	0.19	New Zealand	(Nicoll *et al.*, 1989)

Cashmere goats

Table 1.4 presents the range of genetic and phenotypic correlations between live weight, greasy fleece weight, cashmere yield, cashmere weight, diameter and length obtained from different studies of Cashmere goats. Unfavourable relationships exist between body weight and fleece traits. Increased body weight therefore could result in decreased cashmere yield, weight and length, and increased cashmere diameter. Greasy fleece weight and the other cashmere traits (excluding cashmere length) are positively and strongly correlated (genetically and phenotypically) in South Australian cashmere goats (Gifford *et al.*, 1989). In Australian feral goats negative but non-significant associations between cashmere weight and yield are reported (Pattie and Restall, 1991) indicating that selection for high cashmere production would not increase cashmere yield as well. Yield and diameter measurements are expensive compared with other measurements of down characteristics. Cashmere weight and cashmere yield are both strongly and positively correlated in all studied breeds, except unselected Australian feral goats and therefore, cashmere weight may be used as an indirect measure of cashmere yield. As with Angora goats, strong positive correlations (unfavourable) between down weight (down length) and down diameter are observed in Cashmere goats. Pattie and Restall (1991) suggested two-stage indexes to reduce cashmere diameter without decreasing in the production of other cashmere traits in Australian feral goats. McDonald (1988) reported a significant linear relationship between cashmere length and weight for 1329 male and female goats. This strong positive correlation means cashmere length can be used as an indirect measurement for cashmere weight, because its measurement is much easier and cheaper.

1.5 Genetic and phenotypic correlations between WEC and production traits in sheep and goats

It has been shown that resistance to infection by nematode parasites may not necessarily equate to resistance to the effects of the parasite challenge in grazing animals (Bisset *et al.*, 1996). The association between FEC and productivity varies in magnitude and direction depending on the breed and the environment in which the evaluation was done.

1.5.1 Genetic correlation between resistance and growth

In studies with sheep there is a high variability in estimates of genetic correlation between productivity (e.g. lamb growth rate) and resistance. Table 1.6 summarises the genetic correlations between WEC and growth in sheep. The estimated genetic correlation between WEC and growth rate varies from strongly favourable (i.e. a large negative correlation; (Bishop *et al.*, 1996), through neutral or moderately favourable (Albers *et al.*, 1987; Eady *et al.*, 1998) to moderately unfavourable (a positive correlation) (McEwan *et al.*, 1995). However, most studies have shown negative genetic correlations between body weight or live weight gain and WEC that were not significantly different from zero (Baker *et al.*, 2003).

In Western Australia, selection of Merino sheep for resistance to naturally acquired gastrointestinal nematode parasites had no effect on body weight of sheep (Karlsson *et al.*, 1995). Windon *et al* (1984) also indicated that selection for responsiveness has not adversely affected the productive potential. Selection for resistance of lambs under infective conditions should improve growth, but it might not be true that selection for production would improve resistance, as could be inferred from the genetic correlation between WEC and live weight gain (LWG). In New Zealand studies, selection for low WEC resulted in unfavourable correlated responses in body weight in Perendale and Romney flocks (Watson *et al.*, 1986; Morris *et al.*, 1997). Studies with Romney sheep showed a consistent, but low, unfavourable genetic correlation (0.18-0.25) between WEC and body weight at 8 months of age (McEwan *et al.*, 1995). In the Rylington Merino selection line in Western Australia, (Greeff and Karlsson, 1998) reported that WEC had a genetic correlation of 0.12 with body weight. However, most Australian studies have shown neutral to slightly favourable (negative genetic) correlation between WEC and body weigeadyht. (Eady *et al.*, 1998) observed a possible favourable genetic relationship between $WEC^{0.33}$ and body weight, being generally negative and of the order of 0.2. In the simulation study of the epidemiological and genetic relationships between productivity and resistance to GIN in grazing young lambs, (Bishop and Stear, 1999) reported a genetic correlation of -0.27 respectively between live weight and WEC.

Selection for productive traits might affect the genetic correlation between resistance and growth. In an earlier study (McEwan *et al.*, 1992) assessed the effect of selection for production traits on gastrointestinal nematode parasite resistance in Romney sheep using five lines, which were selected for one of the following: number of lambs born, 100-day weight, hogget fleece weight, or a production index consisting of all these three traits. There was also a randomly bred line. It was found that, between lines, the total WEC was positively correlated with live weight gain (0.95), implying that fast growing lambs harboured more gastrointestinal nematode parasites or were less resistant to parasites. An alternative estimation of genetic variation using half-sib variation within lines revealed a positive genetic correlation between WEC and LWG (0.11) and between WEC and body weight (0.42). Similarly, (Eady *et al.*, 1994) observed positive genetic correlations between WEC and weaning weight and also hogget weight under relatively worm free conditions, but these were not significantly different from zero. In goats, there a favourable genetic relationship between WEC and growth was estimated for Creole kids grazed on infected pasture (Aumont *et al.*, 1998), while the reported genetic correlations between live weight and WEC in Scottish Cashmere goats were not significantly different from zero (Vagenas *et al.*, 2002).

Table 1.6 Genetic (r_g) and phenotypic (r_p) correlations (and their standard errors (s.e.)) between resistance and growth traits in sheep and goats.

Species	Trait 1*	Trait 2	$r_g \pm$ s.e.	r_p	Breed	Infection	Source
Sheep	LWEC	LWG	-0.36±0.23	-0.05	Romney	NatM	Bisset *et al.*, 1992
	LWEC	WWT	0.05±0.22	-0.01	Romney	NatM	
	LWEC	AWT	-0.29±0.22	-0.05	Romney	NatM	
	LWEC	LWG	-0.24±0.17	-0.02	Romney	NatM	Bisset *et al.*, 1994
	WEC	LWG	-0.24±0.17	-0.02	Romney	NatM	
	LWEC	HWT	-0.48±0.13	-0.08	Romney	NatM	
	WEC	LWG	-0.43	-0.06	Romney	NatM	Watson *et al.*, 1986
	WEC	LWG	0.49	-0.06	Romney	NatM	
	WEC	LWG	-0.16±0.21	-0.09	Romney	NatM	Baker *et al.*, 1991
	WEC	LWG	-0.30±0.25	-0.12	Romney	NatM	Douch *et al.*, 1995
	WEC	WWT	-0.16	-0.13	Romney	NatM	
	WEC	LWG	0.01	-0.03	NA	NatM	McEwan *et al.*, 1992
	WEC	LWT100d	0.31	0.00	NA	NatM	
	WEC	LWGuni	-0.02±0.53	NA	NA	ArtHc	Albers *et al.*, 1984
	WEC	LWGinf	-0.76±0.32	NA	NA	ArtHc	
	WEC	WWT	-0.2	NA	Merino	NatM	Eady *et al.*, 1998
	LWEC	WWT	-0.20±0.3	0.02	Merino	NatM	Cummins *et al.*, 1991
	LWEC	YWT	-0.02±0.30	-0.17	Merino	NatM	
	WEC	LWT	-0.63±0.28	-0.13	NA	NatM	Bishop *et al.*, 1996
	LWEC	HWT	-0.42±0.49	0.03	Merino	ArtHc	Piper, 1987
	LWEC	HWT	-0.15	0.01	Merino	NA	Woolaston *et al.*, 1990
	$WEC^{0.5}$	HWT	0.08±0.34	-0.08	Merino	NA	Woolaston *et al.*, 1991
	WEC	HWT	0.12	NA	Merino	NatM	Eady *et al.*, 1994
	LWEC	LWT8mo	0.18-0.25	NA	Romney	NatM	McEwan *et al.*, 1995a
	WEC	LWT	-0.27	-0.10	SCBF	Sim	Bishop, 1999
	WEC15	LWT15	0.12	0.04	Merino	Nat	Greeff & Karlsson, 1998
	LWEC12	LWT12mo	-0.37±0.54	-0.15	R/Massai	NatHc	Baker *et al.*, 2003
	WEC	LWT12mo	-0.05±0.07	-0.02	Merino	NatM	Khusro *et al.*, 2004
	WEC	LWT16mo	-0.10±0.07	-0.06	Merino	NatM	
Goat	QWEC4mo	LWT4mo	0.19±0.20		Creole	NatM	Mandonnet *et al.*, 2001
	QWEC6mo	LWT6mo	-0.03±0.15		Creole	NatM	
	QWEC8mo	LWT8mo	-0.14±0.15		Creole	NatM	
	QWEC10mo	LWT10mo	-0.09±0.14		Creole	NatM	
	LWEC2mo	LWT2mo	0.43±0.21	-0.08	East African	NatM	Baker *et al.*, 2001
	LWEC3mo	LWT3mo	-0.03±0.31	-0.18	East African	NatM	
	LWEC4.5mo	LWT4.5mo	-0.78±0.46	-0.19	East African	NatM	
	LWEC8mo	LWT8mo	0.28±0.37	-0.05	East African	NatM	
	LWEC10mo	LWT10mo	-0.18±0.35	-0.18	East African	NatM	
	LWEC12mo	LWT12mo	0.25±0.39	0.01	East African	NatM	
	CWEC	LWT	0.00±0.15		Scottish Cashmere	NatM	Vagenas *et al.*, 2002

WEC = faecal egg count; LWEC = log transformed WEC; CWEC = Cube root transformed WEC; QWEC = 4^{th} root transformed WEC; LWG = live weight gain; WWT = weaning weight; LWT = live weight; SCB = Scottish Blackface; ArtHc = artificial challenge with *H. contortus*; NatM = natural mixed challenge; YWT = yearling weight; HWT = Hogget weight; AWT = Adult weight.

1.5.2 Genetic correlation between resistance and wool / fleece traits

The correlations between resistance and wool production traits are summarised in Table 1.7.

Table 1.7 Genetic (r_g) and phenotypic (r_p) correlations between worm resistance and fleece traits in sheep and goats.

Species	Trait 1*	Trait 2	$r_g \pm$ s.e.	r_p	Breed	Infection	Source
Sheep	LWEC	GFW	-0.15±0.18	-0.02	Romney	NatM	Bisset *et al.*, 1992
	WEC	GFW	0.30	NA	Merino	NatM	Eady *et al.*, 1994
	$WEC^{0.33}$	GFW	0.15	NA	Merino	NA	Eady *et al.*, 1998
	$WEC^{0.33}$	CFW	0.10	NA	Merino	NA	
	$WEC^{0.33}$	MFD	-0.06	NA	Merino	NA	
	CWEC	GFW	0.21	NA	Merino	NA	
	LWEC	CFW	0.14	0.03	Merino	NA	Woolaston *et al.*, 1990
	LWEC	CFW	0.22	-0.05	Merino	ArtTc	Cummins *et al.*, 1991
	$WEC^{0.5}$	CFW	-0.05	-0.06	Merino	NA	Woolaston *et al.*, 1991
	LWEC	HCFW	0.10	0.12	Merino	ArtHc	Piper, 1987
	LWEC	HMFD	-0.15±0.27	-0.05	Merino	ArtHc	
	LWEC	HGFW	-0.49	-0.07	Merino	NatM	Bisset *et al.*, 1994
	WEC	WG	0.80	NA	Romney	NatM	McEwan *et al.*, 1992
	WEC5	WGRTuinf	-0.02±0.32	NA	NA	ArtHc	
	WEC5	WGRTinf	-0.66±0.28	NA	NA	ArtHc	
	WEC5	MFDuinf	-0.26±0.27	NA	NA	ArtHc	
	WEC5	MFDinf	-0.41±0.24	NA	NA	ArtHc	
	LWEC	MFD	-0.14	-0.08	Merino	NA	Woolaston *et al.*, 1990
	$WEC^{0.5}$	MFD	0.11±0.22	-0.05	Merino	NA	Woolaston *et al.*, 1991
	LWEC	MFD	0±0.3	-0.09	Merino	ArtTc	Cummins *et al.*, 1991
	LWEC15	CFW	0.06	0.02	Merino	NA	Greeff *et al.*, 1998
	LWEC15	MFD	-0.02	-0.02	Merino	NA	
	WEC12mo	CFW12mo	0.11±0.08	0.02	Merino	NatM	Khusro *et al.*, 2004
	WEC12mo	GFW12mo	0.07±0.07	0.04	Merino	NatM	
	WEC12mo	MFD12mo	-0.05±0.07	<0.01	Merino	NatM	
	WEC16mo	CFW16mo	-0.01±0.08	0.02	Merino	NatM	
	WEC16mo	GFW16mo	0.07±0.04	0.03	Merino	NatM	
	WEC16mo	MFD16mo	-0.05±0.02	-0.02	Merino	NatM	
Goat	CWEC	CLength	0.23±0.15	0.06	Scottish	NatM	Vagenas *et al.*, 2002
		CYield	0.16±0.13	0.05	Cashmere	NatM	
		CMFD	0.19±0.16	-0.01		NatM	
		CMFD-SD	0.30±0.19	0.07		NatM	

WEC = faecal egg count; LWEC = log transformed WEC; CWEC = Cube root transformed WEC; WG = Wool growth; WGRT = wool growth rate; CFW = Clean fleece weight; GFW = Greasy fleece weight; MFD = Mean fibre diameter; MFD-SD = Standard deviation of mean fibre diameter; H prefix = Hogget; C prefix = Cashmere.

The pattern of genetic correlations between wool growth and WEC is similar to the correlation between WEC and growth traits. When sheep had not been subjected to specifically intensive selection for wool traits, the genetic correlations between wool growth (clean fleece weight) and WEC were neutral to negative, suggesting that a high resistance to nematodes favours wool growth. This view is supported by findings in Romney sheep (Bisset *et al.*, 1992; Bisset *et al.*, 1994) and in Merino sheep (Piper, 1987; Woolaston *et al.*, 1991). However, unfavourable (positive) genetic relationships between WEC and production have been suggested by several New Zealand studies.

High fleece weight lines have higher WECs (Howse *et al.*, 1992; McEwan *et al.*, 1992; Watson *et al.*, 1992), and studies with Romney sheep showed a consistent, but low, unfavourable genetic correlation (0.18-0.25) between WEC and hogget fleece weight (McEwan *et al.*, 1995). Morris *et al* (1997) in New Zealand concluded that selection for low WEC has resulted in unfavourable correlated responses in 12-month fleece weight in Perendale and Romney flocks. However, Australian studies have shown in most cases neutral to slightly unfavourable (positive) genetic correlation of WEC with clean fleece weight, and neutral with fibre diameter (Eady *et al.*, 1998).

Albers *et al* (1987) found that the genetic correlation between WEC and wool growth was negative under either worm free or infection environments. Such a correlation was high under infection conditions (-0.53 to -0.66) compared with that when sheep were uninfected (-0.02 to -0.17). A contrasting correlation was presented by McEwan *et al.* 1992) in Romney sheep. It was found that between lines WEC was positively correlated with wool growth (0.4). Using sire variation within lines, they also found a significant positive genetic correlation (0.80) between strongyle WEC and hogget fleece weight. This implies that lambs, selectively bred for high wool production, would be less resistant to nematodes.

Garrick (1993) summarised observations of genetic correlations between resistance and production in New Zealand according to selection protocol: either selection for production was carried out prior to selection for resistance or selection for resistance was prior to selection for production. They concluded that if selection for resistance is carried out prior to selection for production the genetic correlations between resistance and production in sheep may be favourable, and if selection for production is carried out prior to selection for resistance, the genetic correlation may be unfavourable. Genetic correlations reported between resistance and fibre diameter have been either neutral (Cummins *et al.*, 1991; Woolaston *et al.*, 1991; Raadsma *et al.*, 1997), or favourable (Albers *et al.*, 1987; Piper, 1987).

Vagenas *et al* (2002) reported that the genetic correlations between Cashmere traits (10 cm^2 patch, fibre length and fibre diameter) and WEC in Scottish Cashmere goats were not significantly different from zero. They concluded that in animals facing the same parasitic challenge, there is no relationship between the animals' ranking on productivity and ranking on WEC, rather than meaning that nematode infection had no effect on these production traits.

Published estimates of the genetic correlations between packed cell volume (PCV) or packed cell volume decline (PCVD) and production (live weight and wool growth) are either negligible, or favourable (Albers *et al.*, 1987; Eady *et al.*, 1998; Greeff and Karlsson, 1998). There are a relatively small number of studies estimating the genetic correlation between resistance and reproduction in sheep. Studies carried out in Australian Merinos showed that this genetic correlation was essentially zero or favourable (Piper, 1987; Woolaston *et al.*, 1991). On the other hand, McMillan *et al.* (1992) found that there was higher litter size and lamb survival in New Zealand Romney sheep selected for low WEC than those selected for high WEC.

1.5.3 Phenotypic correlation between resistance and production traits

Tables 1.6 and 1.7 summarise phenotypic correlations between resistance and production traits. Phenotypic correlation reflects the extent to which performance in one character is associated with performance in the other character. The phenotypic correlations are small, slightly negative (-0.02 to 0.13 and -0.02 to –0.09 for WEC and live weight, respectively) and mostly close to zero. Studies in different lines of Merinos at various locations in Australia showed phenotypic correlations between WEC and production traits that ranged from -0.09 to 0.02 (Eady *et al.*, 1998). This appears to be a common outcome, with low phenotypic correlations occurring in the study of Albers *et al.* (1987) (see Piper and Barger, 1988) and in a range of experiments in New Zealand (Baker *et al.*, 1991; Bisset *et al.*, 1992). In Australian Merino, Eady *et al.* (1998) also observed a small, but generally negative, phenotypic association of $WEC^{0.33}$ with body weight and mean fibre diameter (MFD), indicating that the more resistant sheep tended to be phenotypically heavier and produce coarser wool. However, in studies with Red Massai and Dorper sheep in Kenya Baker *et al* (2003) reported negative significant post weaning phenotypic correlations of an average of –0.15 between LWT and log transformed WEC (LWEC). The predominantly low or close to zero phenotypic correlations between production and WEC in most studies suggests that the measurement of WEC, after infection with GIN would not be a useful indicator of production.

1.5.4 Association between GIN resistance related traits

Phenotypic correlation between LWT and PCV are usually positive and of moderate size (0.2-0.50) in sheep and goats infected with *H. contortus* (Albers *et al.*, 1987; Baker *et al.*, 2001; Baker *et al.*, 2003). Determination of resistance in grazing ruminants is generally based on measurement of WEC (Baker *et al.*, 1998) and selection has been generally on this variable. However, a range of other phenotypic traits is available (Mandonnet *et al.*, 1996) and has been investigated for use as a measure of resistance in their own individual right or in combination with WEC. These traits include those associated with the immune response to gastrointestinal nematode infection such as 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 (in Sheep: Pernthaner, *et al.* 1995; in Goats: Mandonnet *et al.* (2001). Studies in sheep (Blattman *et al.*, 1993; Douch *et al.*, 1995) have indicated that antibody level, particularly IgG, could be used as a phenotypic marker for worm resistance. Low negative genetic and phenotypic correlations of -0.21 and 0.08 between anti-*Trichostrogylus colubriformis* Ab and WEC respectively have been reported in 6-month-old lambs (Douch *et al.*, 1995). The development of acquired immunity to *T. colubriformis* is usually associated with a rise in the number of circulating eosinophils (EOS), with a higher level of eosinophilia found in the animals bred for increased resistance (Dawkins *et al.*, 1989; Rothwell and Sangster, 1993).

Studies showed that eosinophilia is positively correlated with resistance to gastrointestinal nematode infections in sheep (Buddle *et al.*, 1992), and also that eosinophils can damage and kill infective L_3 larvae of the gastrointestinal nematode parasite, *H. contortus*, both in *vitro* (Rainbird *et al.*, 1998) and in *vivo* (Balic *et al.*, 2000). IgE has been shown to stimulate differential release of eosinophil granule proteins and to be more or less effective in inducing eosinophil-mediated parasite killing (Meeusen, 1999). Negative significant phenotypic association between EOS and WEC has been reported in unselected flocks of sheep, which indicates that animals with low WEC are associated with high blood EOS counts (Rothwell and Sangster, 1993; Hohenhaus and Outteridge, 1995; Woolaston *et al.*, 1996). Also reported in sheep were positive genetic and phenotypic correlations for WEC between weaning and hogget age of 0.66 and 0.11, respectively (Greeff and Karlsson, 1998)

PCV has been demonstrated to be an important indicator of resistance to GIN in animals infected with *Haemonchus spp*. An additive genetic breed effect for PCV has been reported to exist in crossbred genotypes of Red Massai and Dorper sheep in Kenya (Baker *et al.*, 1994). Genetic and phenotypic correlation estimates between LWEC and PCV are consistently negative (and therefore favourable) and are of moderate to high magnitude (0.30-0.80) in both sheep and goats (Albers *et al.*, 1987; Baker *et al.*, 1999; 2001 and 2003) when exposed to *H. contortus* infection (Table 1.8).

Table 1.8. Genetic correlations between WEC, PCV and LWT in studies with tropical goats.

Goat type	Age (months	Genetic correlation (±SE)			Source
		WEC-PCV	PCV-LWT	WEC-LWT	
Creole	4	-0.14±0.21	0.47±0.10	0.19±0.20	Mandonnet *et al.*, 2001[a]
	6	-0.47±0.17	0.28±0.12	-0.03±0.15	
	8	-0.67±0.17	0.07±0.18	-0.14±0.15	
	10	-0.06±0.14	0.10±0.14	-0.09±0.14	
East African	2	-0.44±0.23	0.62±0.20	0.43±0.21	Baker *et al.*, 2001[b]
	3	-0.12±0.32	0.82±0.12	-0.03±0.31	
	4.5	-0.95±0.34	0.83±0.12	-0.78±0.46	
	8	-0.32±0.64	0.25±0.47	0.28±0.37	
	10	-0.59±0.29	0.46±0.25	-0.18±0.35	
	12	-0.73±0.38	0.48±0.28	0.25±0.39	

[a]WEC is fourth root transformed.
[b]WEC is log transformed.

These relationships appeared to differ between environments and perhaps the sheep and/or worm populations. The large variability in genetic correlation between WEC and production traits, especially live weight (LWT) in different studies could be as a result of differences in breed, nutritional status (poorer protein nutrition is associated with decreased immune responsiveness), the type of infection (Coop and Holmes, 1996), the species and pathogenecity of the parasites and the actual disease epidemiology. (Bishop and Stear, 1999) indicated that there is an interaction between the genetic resistance of the host population and the epidemiology of the disease, which may affect the genetic relationships between resistance and productivity and such effects need to be accounted for in selection strategies. As resistance has to be estimated under infection, animal production performance in a worm-free environment may differ from that under infection conditions. There are two kinds of infection used for facilitating WEC assessment: artificial infection with either single worm species or mixed worm species and natural mixed infection. Most researchers in Australia use an artificial challenge while the natural infection approach is followed in New Zealand

2. Materials and Methods

2.1 Overview

The Cashmere and Angora goats studied were raised and managed separately under normal commercial conditions on two farms (one breed per farm) near Barraba on the New England Tablelands of New South Wales (NSW), Australia (Latitude 30.38˚ S; Longitude 150.61˚ E). Similar experimental procedures with minor exceptions were followed on both farms. Kids were assessed for resistance to gastrointestinal nematodiasis (GIN) by measuring faecal worm egg counts per gram of faeces (WEC) and parasite-associated traits such as eosinophil counts (EOS), plasma anti-nematode IgG antibody level (Ab) and packed cell volume (PCV) during both natural challenge and after artificial challenge.

In the first year, half of the kids on both sites were vaccinated orally with radiation-attenuated infective larvae of *T. colubriformis* at one month of age to assess immunological responses to an early fixed challenge. In both years, responses to fixed challenge with non-attenuated *T. colubriformis* were assessed following challenge at approximately six months of age. Fleece traits were measured at 6, 12 and 18 months of age in Angora kids, and at 10 and 22 months of age in Cashmeres. Individual live weights were also recorded at each faecal egg collection and shearing for both breeds and also at birth for the Angoras.

2.2 Resource herds and climatic conditions

2.2.1 Angora resource herd

This herd was on the property 'Wiry' owned by Margaret and Jim Harris. Wiry is located 50km west of Barraba in Northern NSW, near the top of Nandewar range. It has an annual rainfall of approximately 1000 mm with slight summer dominance. Initially the owners ran about 700 Angora goats on 809 hectares at this property together with Merino sheep and cattle. About 5000 sheep and 1000 cattle were run on Wiry and the adjacent property "Wongala". At the start of the study the Angora herd comprised 250-300 does of mixed Australian, Texan and South African blood which was being upgraded to a minimum of 87% South African blood. This process was well under way with South African bucks having been used for the previous 5 years and about two thirds of the 250 Angora does used during the 2000 mating had South African sires, while the other third of does from Texan (USA) goat sires. A Texan buck was introduced in this herd in 1993 but subsequently South African bucks were used predominantly.

Three kid drops were included in the study. The number of recorded sires, dams and kids by year are presented in Table 2.1. An average of 56 (range, 50-60) does were randomly mated to each of six Angora bucks in six different paddocks between the end of March and the middle of May in each year. In order to validate comparisons across years, two sires (HILLROSE, YARANPK) in 2000/01 and two sires (PHEZULU1718, YARANPK) in 2001/02 were re-used as link sires. After mating, all does were pooled together into a single herd in which they remained until after kidding. Kidding occurred in a single paddock and individual kid pedigrees were recorded daily by the farm owner/staff. New-born kids were tagged using a temporary tag and their respective dams were recorded in each day. Both male and female progeny were included in the project with a total of 608 Angora kids were recorded for parasite resistance and performance traits.

2.2.2 Cashmere resource herd

This herd was on the property 'Romani' owned by Tony and Judy Brown and located 25km North-East of Barraba in Northern NSW. Romani receives an annual rainfall of about 800mm with slight summer dominance. Initially the owners ran approximately 2000 goats (predominantly Cashmere with some Boer crosses), along with beef cattle on 971 hectares of land at Romani. The cashmere herd was established in 1985 with bucks and does sourced from a wide variety of sources over the years including Kinross, the WA group breeding scheme, Noel Waters, Bess Vickers, Fred Brown and Northumbria. Historically, the Browns practised subjective selection of their stock mainly for Cashmere diameter and yield. Does were mated to selected bucks, in single sire groups of

approximately 60 does. Does kidded in these same groups. To overcome confounding of sire and kidding paddock effects, the project instituted the randomisation of does into different management groups after the joining period. Does then kidded down and reared their kids in these management groups, each containing approximately equal representation of all sires used.

Four kid drops were used in the study. Only female kids were used for the cashmere study as the majority of young males were sold early for capretto. The number of recorded sires, dams and kids by year are presented in Table 2.1. For the first three years (2000-2002), nine Cashmere sires were each joined to approximately 60-70 does in separate paddocks in March-April each year. For the following year (2003), seven sires were mated to 365 does during the mating season. Three sires in 2001 (R1004, R1111, and R0979), one sire in 2002 (R0979) and two sires in 2003 (B2239 and B2453) were re-used from previous years to serve as a link sires across years. After the joining period, the does were reorganized into nine management groups with equal representation of each sire in each management group and the does kidded down in these groups. Kids remained in these management groups until weaning after which they were managed as a single maiden doe mob before being allocated to mating groups the following year. Only kids born in the first six weeks of kidding were included in this study and were identified by tagging with a temporary tag at 4-6.5 weeks of age. Individual kid pedigree was determined by mothering up small groups of kids to their dams at marking time at 6-8 weeks of age. Permanent tags replaced the temporary tags at weaning time at approximately 4-5 months of age. In total, 796 female cashmere kids were recorded for pedigree and had performance measurements made.

Table 2.1 Number of sires, dams and kids used in the Angora and cashmere resource herds. The number of link sires included in the sire total is indicated in parenthesis. Sire totals include only unique sires. Both male and female Angora progeny are included but only female cashmere progeny.

Year	Angora			Cashmere		
	Sires	Dam	Progeny (both sexes)	Sires	Dam	Progeny (female)
2000	6	201	236	9	201	247
2001	6 (2)	173	200	9 (3)	242	286
2002	6 (2)	152	172	9 (1)	85	97
2003				7 (2)	133	166
Total	14	526	608	28	661	796

2.2.3 Climate during the experimental period

Climatic records were not maintained on either resource herd, so data were obtained from the Bureau of Meteorology station at Barraba Post Office, 25km and 50km from the Cashmere and the Angora resource herds respectively. Rainfall and temperature data are summarized in Figure 2.1.

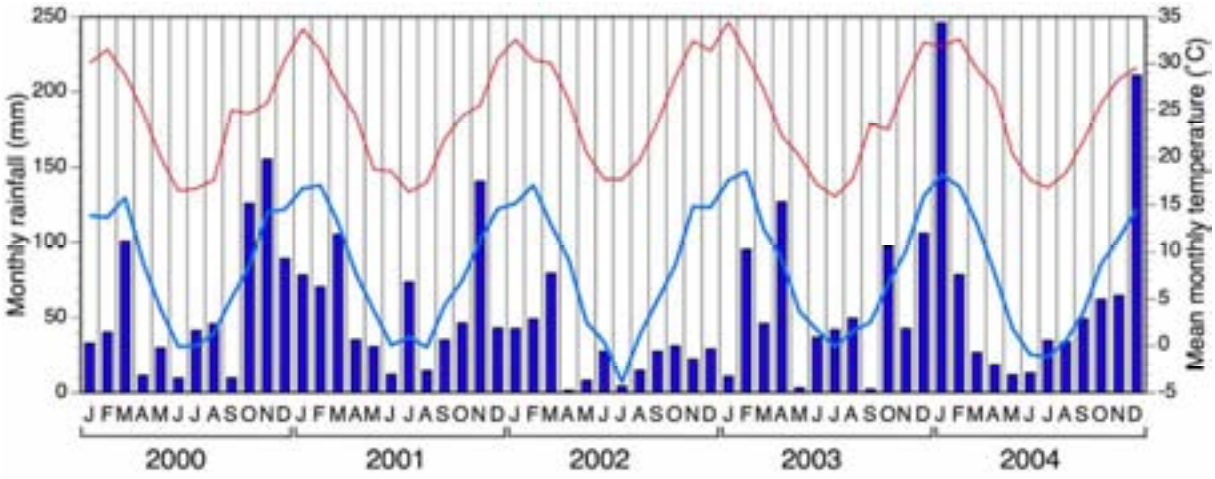

Figure 2.1 Monthly total rainfall (bars) and monthly mean maximum (upper line) and minimum (lower line) temperatures at the Barraba Post Office during the experimental period. Source: Australian Bureau of Meteorology.

The most notable feature is the extreme drought and cold in 2002 which severely reduced natural worm challenge in both 2002 and 2003, in both resource herds. Barraba's average annual rainfall based on 122 years of data is 689mm/yr. The average annual rainfall for the years 2000, 2001, 2002, 2003, 2004 and 2005 was 692, 686, 338, 659 and 850 mm respectively. This put the 2002 rainfall well below the 1st decile of recordings (454 mm) indicating a very major and rare drought event. The July winter average minimum temperature of -3.9˚C was also 4.1˚C colder than the long-term average of 0.2˚C.

2.3 Parasitological treatments

2.3.1 Challenge with irradiated larvae (Year 1 only)

To provide a uniform early immunological stimulus and possible immunological marker for parasite resistance, in year 1 of the project (2000) kids within each sire group in both breeds were randomly allocated into two equal groups: those that received the irradiated vaccination (vaccinated group) and those that did not receive irradiated larval vaccination (unvaccinated control group). The vaccinated group was orally vaccinated with radiation-attenuated L3 larvae (ILV) of *Trichostrongylus colubriformis* at one and two months of age (5000 and 14,000 larvae respectively) while the unvaccinated group were treated with water only. Irradiated larvae are capable of only limited development, and do not produce egg-laying adult worms or the pathology associated with them. The irradiated larvae were of the CSIRO McMaster strain of *T. colubriformis* (fully susceptible to anthelmintics) and were irradiated at the Australian Nuclear Science and Technology Organisation (ANSTO) at the Lucas Heights Nuclear site, Sydney Australia in a gamma pond. Larvae were exposed to approximately 450 Gy of gamma radiation over approximately two hours. The irradiated *T. colubriformis* larvae were stored at 4˚C until used. All the larvae were used within eight weeks of irradiation.

2.3.2 Natural and artificial challenge with gastrointestinal nematodes

In both Angora and Cashmere resource herds, the kids were subjected to initial natural challenge followed by artificial challenge with gastrointestinal nematodes. From birth until around five months of age kids were exposed to natural mixed nematode infections with *Haemonchus* spp., *Trichostrongylus* spp., and *Teladorsagia* spp. by ingesting infective larvae from the pasture they were grazing. The dominant infection during this period was with *Haemonchus contortu*s on both properties. The level of natural challenge was markedly influenced by environmental conditions and infections were interrupted by periodic anthelmintic treatments as required to control the nematodes. Mostly this comprised of a treatment after the 3-month and 5-month samplings. The level of nematode infection was assessed by faecal worm egg counts (WEC) and measures of immunological and blood parameters at 3 and 5 months of age in all kids.

After the 5 month sampling, the natural infections were terminated by an effective short-acting anthelmintic treatment and a week later, an artificial challenge was applied to all animals using infective L_3 larvae of *Trichostrongylus colubriformis* administrated orally at a dose of 10,000 larvae per experimental kid. The larvae were from the CSIRO McMaster strain, (fully susceptible to anthelmintics) derived from animals that were free of ovine Johne's disease. *T. colubriformis* is a common parasite in Northern NSW, and is also abundant in most other high rainfall grazing regions around Australia. Therefore, this species was chosen as the most representative to use as a challenge species. Faecal and blood samples were collected at 28 and 35 days post artificial infection when the kids were approximately 6.25- and 6.5-month old. After collection of samples at day 35 post challenge, the artificial infection was terminated by drenching with an effective anthelmintic.

2.4 Measurements and laboratory procedures

2.4.1 Measurement summary

The timing of sample collections is summarised in Table 2.2

Table 2.2. Timing of sample collections during the project.

Age (months)	Sample/measurement	Angora		Cashmere	
		Yr 1	Yrs 1-3	Yr 1	Yrs 1-4
2	Blood	Yes		Yes	
3	Faeces, blood, weight	Yes	Yes	Yes	Yes
5	Faeces, blood, weight	Yes	Yes	Yes	Yes
6	Fleece	Yes			
6.25	Faeces, blood, weight	Yes	Yes	Yes	Yes (6.5mo)
6.5	Faeces	Yes	Yes	Yes	Yes
10	Fleece, LWT			Yes	Yes
12	Fleece, LWT	Yes	Yes		
18	Fleece, LWT	Yes	Yes		
22	Fleece, LWT			Yes	Yes

2.4.2 Measurement of live weight

Live weights of Angora and Cashmere goats were measured using Ruddweigh scales at about 3, 5, 6.25 and 6.5 months of age, during collection of faecal egg count samples as well as at shearing times. Shearing occurred at 6, 12, and 18 months for Angora goats whereas it occurred at 10 and 22 months for Cashmere goats as shown in Table 2.2. Animals were weighed just before shearing and therefore the live weight at shearing was calculated as pre-shearing live weight less the weight of the shorn fleece.

2.4.3 Faecal worm egg count and larval differentiation

In years 1 and 2 the WEC counts and larval differentiation were performed in the UNE parasitology laboratory while in years 3 and 4 they were performed by a commercial provider. Faeces for parasitological examination were collected from the rectum of Angora and Cashmere kids at approximately 3, 5, 6.25 and 6.5 months of age. The number of worm eggs per gram of faeces (WEC) was counted using a modification of the McMaster floatation technique (MAFF, 1986). At each WEC measurement, a pooled faecal sample for each breed containing faeces from about 90% of the animals sampled was mixed with vermiculite and incubated aerobically at 28˚C for seven days. The larvae were harvested after inversion of the culture over a large petrie dish full of water and recovered from the water. Larvae then differentiated to determine their genus based on the morphology of 100 ex-sheathed L_3 larvae as observed under the microscope at 100 – 250X magnification (MAFF, 1986).

2.4.4 Haematology analysis

Blood samples were collected in order to assess pathological consequences of infection and/or the host immune response after natural and artificial challenge. Blood samples were collected from the jugular vein of individual kids using vacutainers containing ethylene diamine tetra-acetic acid (K_3 EDTA). Samples were stored at 1-4^{o}C in a portable 12V refrigerator for up to 48 hours before being analysed for blood parameters using an automated haematology analyser (Cell-Dyn® 3500, Abbott, Norfolk, VA USA) at CSIRO Livestock Industries, Chiswick, Armidale. Plasma was then removed by centrifugation and samples were stored individually at -20^{o}C until required for enzyme linked immunosorbent assay (ELISA) to determine the levels of specific IgG to *T. colubriformis* antigens in each sample (AB). The variables measured on the Cell Dyn® included counts of total white cells (WCC), eosinophils (EOS), basophils (BAS), neutrophils (NEU), lymphocytes (LYM), monocytes (MON), and red cells (erythrocytes) (RBC), packed cell volume (PCV), haemoglobin content (HGB), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC).

2.4.5 Assay for anti-nematode IgG

To quantify the humoral immune response (antibody response) against fixed *T. colubriformis* challenge at 5 months of age, specific IgG in the serum directed against *T. colubriformis* antigen was measured using an indirect Enzyme Linked Immunosorbent Assay (ELISA). The ELISA was an adaptation of the assay of Gill (1991) as described by Olayemi (2004). The ELISA principle is to detect the presence of specific IgG antibody directed against the antigens attached to the wells of polystyrene plates. The retained antibody is detected by an anti-Goat/Sheep IgG antibody conjugated to an enzyme, which in presence of a suitable substrate produces a coloured reaction. The coloured product is then used to quantify the level of anti-*T. colubriformis* IgG against a standard curve of known concentration. In practice there is significant cross reaction between ELISA assays detecting antibody responses to the three main nematode species (Walkden-Brown and Maslen, 2002), so the results should be interpreted as a non-specific response to nematode infection rather than a specific response to infection with *T. colubriformis* although the artificial challenge at 5 mo with *T. colubriformis* is the major trigger for an increase in AB levels.

2.4.6 Fleece sampling and fibre analyses

Fleeces were weighed on farm and sub-sampled at each relevant shearing. For Angora goats at 6, 12 and 18 months of age, the measurements recorded were greasy fleece weight (GFW), mean fibre diameter (MFD), standard deviation of MFD (MFD_SD), mean fibre curvature (CURVE), percentage of kemp (%KEMP) and percentage of medullation (%MED) was measured to estimate the genetic parameters. Approximately 15-20g of fleece were sampled from the mid side of the body in a labelled plastic bag and brought into the lab for further analysis. GFW was measured at shearing. MFD, MFD_SD, CURVE, %KEMP and %MED were measured using an Optical Based Fibre Diameter Analyser (OFDA 100, BSC Electronics Pty, Perth, WA) after calibration using an AWTO calibration set of mohair tops. MFD variation is expressed as coefficient of variation of fibre diameter (CV (%) $= 100 * s / \bar{x}$, where s is the standard deviation of MFD and $\bar{x}$ is the average MFD).

For cashmere goats at 10 and 22 months of age, greasy fleece weight (GFW), cashmere weight (CSW), cashmere yield (CSY), mean cashmere diameter (CSD), mean fibre curvature (CURVE) and hair diameter (HD) were measured to estimate the genetic parameters. GFW was obtained by weighing the total harvested raw fleece from each animal. Fleece sub-sampling for further analysis was carried out as follows: the complete harvested raw fleece was laid on a flat table and divided into two equal halves. Each half was subdivided into 16 sections, and a pinch (0.5-0.6g) was taken from each of the total of 32 sections and pooled together into a sub-sample (15-20g), which was mixed well and stored until OFDA analysis. OFDA analysis was based on the mean of three replicates per sample, with a total of 6,000-8000 fibres being measured per animal Fibres with diameters of 1 – 35µm were classified as cashmere and those above 35µm as hair and the measurement of HD (>35µm), CSD (1-35µm), and CURVE were extracted from OFDA output. CSY, MFD and CURVE were calculated from the output of the OFDA using the equation of Peterson and Gherardi (1996) as follows:

$$\text{CSY (\%)} = \frac{100\sum_{i=1}^{35} d_i^2 f_i}{\sum_{i=1}^{150} d_i^2 f_i}$$

where d is the fibre diameter and f is the frequency of fibres.

CSW was calculated as CSW = (GFW x CSY)/100.

2.4.7 DNA sample collection and processing

Both blood and ear skin tissue samples were collected from Angora and Cashmere kids. Blood samples were taken from the jugular vein of individual kids using vacutainers containing ethylene diamine tetra-acetic acid (K3 EDTA) while ear skin tissue was sampled using an ear punch which was thoroughly cleaned between animals. Punch samples were placed into individual ziplock plastic bags, and held in a portable 12V refrigerator until reaching the lab where they were placed in a freezer at -20˚C for storage.

For tissue samples a small amount (20-30mg) of ear punch tissue was cut into tiny pieces and digested it at 55^{o}C in a 0.5 ml digestion buffer with Proteinase K at 200µg/ml for 3 hours with occasional spinning. After digestion, genomic DNA was extracted using the phenol/chloroform extraction protocol described by (Sambrook *et al.*, 1989). After that, the DNA pellet was dried for 30 minutes at 37^{o}C and resuspended in 200µl TE buffer (pH=8.0) in an incubator at 65^{o}C for five minutes.

The procedure of DNA extraction from blood was as described by Sunduimijid (2007). Briefly 10 ml of blood was lysed in two volumes of ice cold RBC lysis solution then centrifuged at 2800 x g for 10 minutes at 4^{o}C and the supernatant decanted. After two washing steps in 10 ml ice cold TBS buffer the washed pellet was gently resuspended on ice in 4 ml TE buffer with Proteinase K at 200µg/ml and 0.5%SDS and then incubated overnight at 37^{o}C in a shaker. Following this 1.5ml of a 5 M NaCl solution and 5ml of chloroform were added and the sample shaken for approximately 40 minutes using a rocking platform. The sample was then centrifuged at 3800 x g for 25minutes at 4^{o}C and the aqueous supernatant carefully removed to a new 50 ml tube using a wide bore pipette for precipitation of DNA in two volumes of 100% ethanol while gently shaking. The 100% ethanol was then replaced with 70% ethanol for further precipitation of DNA. DNA was then transferred to 1.5ml tubes and resuspended in 0.5 ml TE buffer.

Agarose gel electrophoresis was used to quantify and check the integrity of genomic DNA. 10µl of extracted DNA was mixed with an appropriate volume of loading buffer and loaded into a well on a one percent (1%) agarose gel. Gels were run at 80 Volts for 2 hours. A pUC19 DNA/HpaII standard molecular weight marker used to approximately quantify the amount of extracted DNA. DNA samples were then quantified using SmartSpec^{TM}3000 spectrophotometer, which takes absorbance readings (O.D. value) at 260, 280 and 320nm. The DNA purity was checked using the A_{260}/A_{280} ratio. A ratio of 1.8-2.0 is expected for pure DNA (indicating absence of contaminating protein).

2.4.8 Microsatellite marker choice and PCR amplification

For parentage analysis genotyping, fourteen microsatellite markers (Table 2.3) were chosen from the "goat map" website: http://dga.jouy.inra.fr/cgi-bin/lgbc/carte.pl?BASE=goat&NOM=schibler_1998**.**

Table 2.3 Characteristics of the fourteen microsatellite markers studied

Locus	Chromosome	Position (cM)	Primer (5'-3)	T_a (^{0}C)	$MgCl_2$ (mM)	Size range (bp)	Origin
			AAATGTGTTTAAGATTCCATACAGTG				
OarFCB020	2	41.0	GGAAAACCCCCATATATACCTATAC	55	2.5	119-135	Ovine
			TGTTTTGATGGAACACAGCC				
ILSTS029	3	-	TGGATTTAGACCAGGGTTGG	55	2.0	154-186	Bovine
			TCTCAGCACCTTAATTGCTGG				
SRCRSP07	6	-	TCAACACTCCAATGGTGAGCT	55	2.5	119-135	Caprine
			TCGCAAAAAGTTGGACAAGACT				
RM096	11	42.0	TTAGCAGGGTGCCTGACACTT	58	2.0	98-126	Bovine
			CATGCTATAACACTCCAGTAAG				
LSCV44	11	73.0	GCAGGTTACAGTCCATGGG	52	1.5	112-134	Ovine
			GCTTGCTACATGGAAAGTGC				
ILSTS11	14	-	CTAAAATGCAGAGCCCTACC	55	2.5	260-284	Bovine
			CAGCAGACAGCTGCAAGAGTTAGC				
TGLA53	16	-	CTTTCAGAAATAGTTTGCATTCATGCAG	55	2.5	115-143	Bovine
			ATTTGCACAAGCTAAATCTAACC				
INRA063	18	22.0	AAACCACAGAAATGCTTGGAAG	55	2.5	164-172	Bovine
			CAATCTTGCTCCCACTATGC				
INRABERN185	18	-	CTCCTAAAACACTCCCACACTA	55	2.5	256-284	Bovine
			GGACTCTACCAACTGAGCTACAAG				
SRCRSP05	21	9.0	TGAAATGAAGCTAAAGCAATGC	55	2.5	158-182	Caprine
			AAAACTGAGAAATATTCAGATGCAAC				
OarCP73	23	25.0	TAAACGTCCATCAACAGAGGAAGGG	63	4.5	154-242	Ovine
			GTATGTATTTTTCCCACCCTGC				
BM1258	23	42.0	GAGTCAGACATGACTGAGCCTG	58	2.0	102-136	Bovine
			AGCTGGGAATATAACCAAAGG				
BM1818	23	87.0	AGTGCTTTCAAGGTCCATGC	56	2.0	250-270	Bovine
			CCACTTCCCTGTATCCTCCT				
INRABERN172	26	42.0	GGTGCTCCCATTGTGTAGAC	55	2.5	232-252	Bovine

(-) indicates that the marker is not mapped on a particular chromosome

Of these fourteen markers, three were mapped as adjacent to the OLA-DRB gene or MHC (Ovine Major Histocompatibility Complex class II DR-ß) on chromosome 23 (Chr23) and two markers were assumed to be close to the region of IL1 ß (interleukin ß) and IGHML (immunoglobulin mu like) genes on chromosome 11 (Chr11). The other nine markers chosen were distributed throughout the goat genome. Optimization of PCR conditions was carried out by changing the concentrations of $MgCl_2$ (1.5-4.5mM) and 4dNTP's (62.5µM, 80.0µM and 100.0µM). Manual genotyping was performed using the conventional autoradiographic method.

PCR amplification of markers was as described by Sunduimijid (2007). Briefly reaction volumes contained ~10ng genomic DNA. After adding 1µl DNA sample to each well, the PCR mixture of 3.0µl per well was added and then overlaid with 15µl of mineral oil. The amounts of each reagent used to prepare the PCR mixture, the amount of each DNA sample used and the cycling parameters are described by Sunduimijid (2007). Final concentrations of $MgCl_2$ and 4dNTP's were adjusted according to the primers used. An MJ Research PTC-200 thermal cycler was used for running the PCR reactions. After thermal cycling, the reactions were terminated by adding 5µl of stop solution buffer and kept at -20^{o}C. Visualization of PCR products was done using gel electrophoresis and autoradiography.

2.5 Genetic and statistical analyses

2.5.1 Parentage Analysis

A detailed description of the parentage analysis is provided by Sunduimijid (2007). The effectiveness of the 14 studied loci as DNA markers for determining parentage and assigning alleged parents to individuals was based on verified Mendelian segregation of markers. The effectiveness of the microsatellite marker set was estimated using the probability of exclusion, which is defined as the probability of excluding candidate (non-parent) individuals as parent. PE_1 indicates the probability exclusions for the case when both parents are unconfirmed (neither parents are known), whereas PE_2 indicates the probability exclusions when one parent is known. The equations used for estimating PE_1 and PE_2 are as described by Jamieson and Taylor (1997).

Likelihood parentage analysis was performed using the CERVUS software package (Marshall *et al.*, 1998). CERVUS is a categorical allocation program, which assigns offspring to non-excluded parents based on maximum likelihood estimates. It calculates the following test statistics: log-likelihood ratios, and LOD and Delta (Δ) scores. The likelihood ratio is the likelihood that the candidate parent is the true parent divided by the likelihood that the candidate parent is not the true parent. The overall likelihood ratio for each candidate parent is calculated by multiplying together the likelihood ratios at each locus, whereas the LOD score is the sum of the log likelihood ratios at each locus (Marshall *et al.*, 1998). Delta (Δ) score is the difference in LOD scores between the most likely candidate parent and the second most likely candidate parent.

The parentage analysis was divided into three sequential modules: 1) the allele frequency module (module 1); 2) the simulation module (module 2); and 3) parentage analysis module (module 3). Using genotype data, module 1 calculates allele frequencies, expected and observed heterozygosities, polymorphic information content (*PIC*), probability of exclusions (PE_1) and (PE_2), goodness-of-fit Hardy Weinberg test (Chi-squared values), and null allele frequency at each locus. Then the second module uses these allele frequencies to simulate a pair of parental genotypes plus a series of random genotypes representing unrelated candidate parents of each sex. Genotypes of offspring were generally produced by Mendelian sampling of alleles. The purposes of the simulation module are 1) to test the feasibility of parentage analysis using a given set of loci and 2) to estimate critical values of the log-likelihood statistic Delta, which is used to assess the reliability of parentage assignment. Simulation parameters used in the parentage analysis were as follows: 10000 iteration cycles; 137 and 192 candidate parents for Angora and Cashmere goats, respectively; 100% of loci genotyped, 1% and 0% typing errors for preliminary and final test, respectively; pre-determined levels of relaxed and strict confidence were 80% and 95%, respectively. After simulation analysis, for each offspring the third module estimated LOD scores for each candidate parent, found the two most likely parents and then calculated the corresponding Delta (Δ) scores as for the simulation. The final assignment of this module was to evaluate the confidence of the Delta score using the appropriate Delta criteria calculated by the simulation. If the observed value of Δ scores was sufficiently large to distinguish true parents from unrelated candidate parents at pre-determined level of confidence (95%), the most likely parent with highest likelihood was accepted as the true parent with 95% confidence. The steps used in parentage assignment analysis were as follows:

Step1 (paternity assignment):
sires were genetically compared against kids

Step2: the confirmed sire were matched to each kid
kids with unresolved* sire were removed
} input file for maternity assignment

Step3 (maternity assignment):
Dams were genetically compared against kids with confirmed sire

Step4: dams were genetically compared against kids with unresolved sire

*case when there was a discrepancy between parent's and progeny genotypes at more than three loci as well as a very low LOD (<1.5).

The total number of genotypes described by parentage analysis was 428 in the Angora population and 548 in the Cashmere population (Table 2.4).

Table 2.4 The number of animals used in parentage analysis

Population	Offspring	Sires	Dams*
Angora	297	11	126
Cashmere	417	20	172

*- the number of dams, including dams that are both offspring and dams

2.5.2 Statistical and genetic analyses

Traits analysed in the analysis of genetic parameters

In total, 18 production and parasite associated traits were analysed to estimate (co) variance components, heritabilities and correlations. Parasite-associated traits analysed were worm faecal egg counts (WEC), specific anti-Trichostrongylus IgG concentration (IgG), blood eosinophil counts (EOS), lymphocyte counts (LYM), neutrophil counts (NEU), basophil counts (BASO), monocyte counts (MONO), packed cell volume (PCV), mean corpuscular volume (MCV) and mean corpuscular haemoglobin concentration (MCHC). Production traits analysed included live weight and a range fleece traits for each goat population.

Data transformation

Prior to estimation of (co)variance components, distributions of residual variance of each trait were investigated using the JMP IN 5.1 program (SAS/STATS, 2005). WEC, IgG, EOS and KEMP demonstrated skewed distributions (with high skewness and kurtosis). In order to normalize these skewed distributions, log (log(x+10)), squared ($x^{0.5}$), cubic ($x^{0.33}$), and quartic ($x^{0.25}$) transformed scales were applied for WEC, AB, EOS, and KEMP. Distributions of these traits with each transformed scale applied were assessed based on Shapiro-Wilks W Test for goodness-of-fit. The best transformation was squared for WEC and AB, cubic for EOS and quadratic for KEMP. For the estimations of genetic parameters, outliers which were more than ±5.0 standard deviations were excluded from data set.

Fixed effects

Using the "MIXED" procedure of SAS program (SAS/STATS, 1991-2005), the significance of fixed effects were tested for each trait with a sire model fitting sire as a random effect ($y = Xb + Zs + e$), where *s* and *b* are the vectors of random sire effect and fixed effects, respectively, *X* is the incidence matrix of fixed effects, and *Z* is the incidence matrix relating to records of the sire effect). The number of levels of each effect used was given in Table 2.5. Three and four years of records were collected for Angora and Cashmere goats, respectively. Age at measurement (treated as covariable) was not recorded in Cashmere goats (ie. exact birth dates were unknown). Only female kids' records were available for the Cashmere goat herd. Type of birth had two classes: singles and twins. Triplet kids (very low numbers) were classified to the "twin" class.

Table 2.5 Number of levels of main fixed effects by populations

	Number of levels of each main effect				
Population	Year of birth	Type of birth	Sex	Age	Management group
Angora_Aus	3	2	2	covariable	NA
Cashmere_Aus	4	2	NA	NA	12

NA = not applicable

Models of analysis

In order to find the best fit of model for estimating the variance components, four single trait models were examined for each trait: 1) significant fixed effects only (model 1); 2) model 1 with direct additive effect of the animal (model 2); and 3) model 2 with permanent environment effect of the dam (model 3). A model with an additive and permanent environment effect of the animal (model 4) was applied for WEC to estimate the repeatability of worm egg counts recorded at 28 and 35 days post artificial challenge (6.25 and 6.5 months of age). Models used can be presented as:

(Model 1) $y = Xb + e$

(Model 2) $y = Xb + Z_1 a + e$

(Model 3) $y = Xb + Z_1 a + Z_2 m_{pe} + e$

(Model 4) $y = Xb + Z_1 a + Wpe + e$

where y is vector of observations, b is vector of fixed effects, and a, m_{pe} and pe are vectors of additive random animal effects, maternal permanent environmental effects and random permanent environmental (non-additive genetic) effects (apply where animals have more than one record), respectively and e is a vector of residuals. X is the incidence matrix of fixed effects. Z_1, Z_2 and W are incidence matrices relating to observations to the corresponded random effects.

The expectations of variables (y) were $E(y) = Xb$ and random effects (additive random animal effects, maternal permanent environmental effects, random permanent environmental effects) and residual effects are independently distributed with means of zero ($E(a) = E(m_{pe})=E(pe)=E(e)$ = 0) and variance σ (var(a) = A σ^2_a, var(pe) = I σ^2_{pe}, var(m_{pe}) = I σ^2_{mpe}, var(e) = I σ^2_e, where A is the numerator relationship matrix and I is an identity matrix).

Phenotypic variance (σ^2_p) was computed as the sum of the variance of all the random effects fitted and the error variance.

$\sigma^2_p = \sigma^2_a + \sigma^2_e$, $\sigma^2_p = \sigma^2_a + \sigma^2_{mpe} + \sigma^2_e$, and $\sigma^2_p = \sigma^2_a + \sigma^2_{pe} + \sigma^2_e$ for model 2, 3, and 4, respectively.

Then the genetic parameters were estimated as:

heritability = $h^2 = \sigma^2_a / \sigma^2_p$

maternal permanent environmental effect = $c^2 = \sigma^2_{mpe} / \sigma^2_p$

permanent environmental effect = $pe^2 = \sigma^2_{pe} / \sigma^2_p$

repeatability = $t = (\sigma^2_a + \sigma^2_{pe}) / \sigma^2_p$

Phenotypic and genetic correlations between X and Y traits were computed using Model 2, which was the best model for most of the traits studied:

phenotypic correlation = $r_p = \sigma_{p(XY)} \Big/ \sqrt{\sigma^2_{p(X)} \sigma^2_{p(Y)}}$

genetic correlation = $r_a = \sigma_{a(XY)} \Big/ \sqrt{\sigma^2_{a(X)} \sigma^2_{a(Y)}}$

Univariate analysis was performed for estimation of heritabilities for all traits. The phenotypic and genetic correlation estimates between traits were extracted from bivariate analysis. Those univariate and bivariate analyses were developed using the ASReml software (Gilmour *et al.*, 2002). ASReml is a statistical package that fits linear mixed models using Residual Maximum Likelihood (REML) to estimate variance components with an Average Information (AI) algorithm and sparse matrix methods. The convergence was assumed when changes in the log likelihood were 0.00001. The approximate standard errors for heritability and correlations were derived from ASReml analysis as well.

Tests of random and fixed effects. The significance of fixed effects was examined by F test using ratios of adjusted mean squares (type 3 tests of fixed effects). Only those effects which were significant (P <0.05) in presence of sire effect were left in the final model. A likelihood ratio test (LRT) for the goodness-of-fit between two models was used to decide the significance of random effects after adding them to the model sequentially. The difference in log likelihood values of two compared models ($D = 2*(\ln L_1 - \ln L_2)$ after adding n random effects was tested by a χ^2_n statistic (Gilmour *et al.*, 2002). So the random effect was retained in the model only when it significantly (P <0.05) improved the likelihood ratio.

3. Immune response to worm infection in Angora and cashmere kids vaccinated with irradiated *T. colubriformis* larvae

3.1 Introduction

Use of irradiated larval vaccination (ILV) with live, radiation-attenuated third stage infective larvae has two main potential uses. Firstly it may be used to increase immunity to infection in the target population in general, as is the case with most vaccines. It may also have a role in imitating immune responses which allow differentiation of genetically resistant "responder" animals from genetically susceptible "non-responder" animals in the absence of patent infections (Windon 1996). In this sense it is a phenotypic marker for genetic resistance, one that has been shown to successfully identify sheep that on the basis of their resistance to infection with gastrointestinal nematodes (Windon and Dineen, 1981).

Use of irradiated larval vaccination (ILV) with live, radiation-attenuated third stage infective larvae to increase immunity without inducing patent infections has been the main approach adopted to date. The use of ILV has produced high levels of protection in livestock experiments for the treatment of bovine lungworm, *Dictyocaulus viviparus* (Jarret *et al.* 1957); the lung worm of sheep and goats, *Dictyocaulus fularia* (Javanovic *et al.* 1965); and the canine hook worm, *Ancylostoma caninum* (Miller 1978) and has been successfully commercialised for treating infection with bovine lungworm, *Dictyocaulus viviparus* (Jarret *et al.* 1957). Many host-parasite systems have been investigated for ILV in sheep against helminth infection. ILV *H. contortus* and *T. colubriformis* gave 95% (Benitez-Usher *et al.* 1977) and above 88% (Windon *et al.* 1984) protection, respectively in lambs not less than 6 months of age. Despite the success of these studies, commercial production has been hampered due to inability to obtain the same high level of protection in young lambs less than 7 months old. Also, a higher level of protection was obtained in the older lambs if they had not been previously exposed to infection before vaccination, something that is unlikely to be achieved in the field (Benitez-Usher *et al.* 1977). Thus ILV efficacy in sheep has been shown to increase with age. This is unfortunate as lambs and weaners are more at risk from pathogenic effects of gastrointestinal parasitism than mature sheep. Even though substantial resistance develops slowly over 3-4 months, young lambs are less able to resist parasite establishment or remove existing burdens (Emery *et al.* 1999), and are therefore primary targets for potential vaccines against gastrointestinal nematodes. Indeed these authors showed that protective immunity to *T. colubriformis* could be induced in neonatal lambs vaccinated using trickle infections with *T. colubriformis* and that this immunity was stronger than that observed in 4 month old lambs treated similarly.

However, there have been no such studies in goats, for which gastro-intestinal nematode infection is if anything, more important than in sheep. It is therefore, worthwhile investigating the possibility of inducing immune response to GIN in goats using ILV. The aim of this study was to investigate the effect of early vaccination of Angora and Cashmere kids with irradiated third stage larvae of *T. colubriformis* (TcL_3) on immune response and resistance to subsequent natural and artificial gastrointestinal parasite infection. This experiment was therefore designed to test the hypothesis that Angora and Cashmere goat kids orally vaccinated with radiation-attenuated *T. colubriformis* (*Tc*) larvae at approximately 1 month of age with a booster vaccination at 2 months of age will exhibit enhanced resistance to subsequent natural and artificial GIN challenge as evidenced by a) reduced faecal egg count (WEC); b) increased plasma levels of Tc-specific IgG; c) increased blood eosinophil count; and d) increased blood packed cell volume.

3.2 Materials and methods

The experimental animals consisted of 222 Angora mixed sex and 212 Cashmere female kids born between September and October 2000, and raised separately in the two resource herds (section 2.2). Half of the Angora and Cashmere kids selected at random were individually given oral administration of two doses of γ-irradiated (L_3) larvae of *T. colubriformis* (Vaccinated), while the other half served as controls. Vaccinated kids received the first dose of 5,000 irradiated larvae at an average age of 1 month, followed by a second (booster) dose of 14,000 of the irradiated larvae one month later. All kids were subjected to natural nematode infection up to 5 months of age. The natural infection was terminated in all the kids at the age of 5 months with moxidectin (200µg/kg, Cydectin®, Fort Dodge) and they were re-infected a week later with an oral dose of 10,000 virulent L_3 larvae of *T. colubriformis* (see section 2.3 for details). Individual faecal worm egg count (WEC) and bulk larval differentiation were determined at 3 (WEC3) and 5 (WEC5) months of age during natural challenge and at days 28 (WEC6.25) and 35 (WEC6.5) days post artificial challenge as described in section 2.4.3. Blood samples were individually collected by jugular venipuncture to determine the level of packed cell volume (PCV), specific *T. colubriformis* antibody level (Ab) and serum eosinophil counts (EOS) for each individual at natural and artificial challenge (section 2.4.4).

Statistical Analysis

The samples were analysed to determine fixed effects of importance using SPLUS statistical application (MathSoft 1999). The fixed effects considered in Angora goats were sire, birth type, sex, vaccination, age fitted as covariate and their first order interactions while in Cashmere kids the fixed effects tested were sire, birth type, vaccination and rearing management group. The first order interactions were not significant so they were dropped from the model. WEC, Ab and EOS were transformed prior to data analysis to stabilize the variances. WEC were cube root transformed ($WEC^{0.33}$), Ab were $\log_e$ transformed and in order to account for zero values, a constant of 10 was added (i.e. Log_e (Ab+10) and eosinophil counts were fourth root transformed ($EOS^{0.25}$). A significance level of $p<0.05$ is used throughout and data are generally presented as transformed least squares means (means adjusted for the other effects in the model) with standard errors.

3.3 Results

Descriptive data relating to worm infections during the experiments are summarized in Table 3.1. The data show that there was significant natural challenge, mainly with *H. contortus* on both farms, although *Teladorsagia* was the dominant parasite at 3mo in Angora goats. Artificial infection with *T. colubriformis* was successful and induced high WEC, particularly in Angora goats.

Table 3.1. Arithmetic mean faecal worm egg count (WEC) and larval differentiation data for each breed and sampling period.

Breed	Variable	Level	Natural infection		Artificial infection	
			WEC3	WEC5	WEC6.25	WEC6.5
Angora	Arithmetic WEC (epg)	All	112	1015	2785	3158
	Larval Diff. (%)	*Haemonchus spp*	9	91	5	8
		Trichostrongylus spp	15	6	95	89
		Teladorsagia sp	76	3	-	3
Cashmere	Arithmetic WEC (epg)	All	635	2039	1734	1756
	Larval Diff. (%)	*Haemonchus spp*	91	91	7	7
		Trichostrongylus spp	9	7	93	92
		Teladorsagia sp	0	2	-	1

The influence of ILV and other main effects on $WEC^{0.33}$ following statistical analysis is shown in Table 3.2 and Figure 3.1. Only the effect of ILV is discussed here as the other effects were tested over several years and are reported in later sections. ILV had no influence on $WEC^{0.33}$ at 3, 5 and 6.25 months of age in both breeds, but it significantly ($P< 0.01$) influenced $WEC^{0.33}$ at 6.5 months of age (i.e. 35 days post artificial challenge) in Angora kids, with a higher mean $WEC^{0.33}$ in vaccinated group than in the control (14.63±0.29 Vs 13.72±0.29) (Tables 3.2 and 3.3, Figure 3.1).

Table 3.2. Summary of Analysis of Variance of cube root transformed faecal egg count ($WEC^{0.33}$) in Angora and Cashmere kids measured at 2, 3, 5 and 6.5 months old in the first year (2001) of the study.

Effect	Breed	Natural infection		Artificial challenge	
		3 months	5 months	6.25 months	6.5 months
Sire	Angora	NS	*	NS	*
Birth type		*	NS	NS	NS
Sex		NS	NS	NS	NS
Irradiated larvae vaccination (ILV)		NS	NS	NS	*
Age		**	NS	NS	NS
Sire	Cashmere	NS	**	**	**
Birth type		**	NS	NS	NS
Management group		**	**	NS	*
Irradiated larvae vaccination (ILV)		NS	NS	NS	NS

* = Significant at 5% (P>0.05), ** = significant at 1% (P<0.001), NS = not significant at 5% (P>0.05) and NA = not applicable.

Table 3.3 Least squares means ± se of cube root transformed faecal egg count ($WEC^{0.33}$, eggs/g/faeces) of Angora and Cashmere goats vaccinated with irradiated L_3 larvae of *T. colubriformis*.

	Natural Challenge		Post artificial challenge	
Fixed effects	$WEC^{0.33}$ 3	$WEC^{0.33}$ 5	$WEC^{0.33}$ 6.25	$WEC^{0.33}$ 6.5
Angora				
P value of test	0.649	0.668	0.319	0.015
Overall mean	3.19±0.24 (229)	9. 13±0.26 (222)	13.38±0.26 (221)	14.26±0.22 (217)
Vaccinated	3.23±0.31 (110)	9.30±0.35 (106)	13.61±0.35(105)	14.63±0.29 (106)
Unvaccinated	3.15±0.31 (119)	9.10±0.35 (116)	13.16±0.35(116)	13.72±0.29 (111)
Cashmere				
P value of test	0.144	0.974	0583	0.748
Overall mean	6.60±0.30 (235)	10.36±0.30 (235)	11.78±0.14 (232)	11.90±0.17 (224)
Vaccinated	6.34±0.30 (116)	10.36±0.36 (120)	11.85±0.19 (115)	11.86±0.21 (112)
Unvaccinated	6.86±0.41 (119)	10.37±0.39 (115)	11.71±0.20 (117)	11.95±0.23 (112)

$WEC^{0.33}$ 3, $WEC^{0.33}$ 5, $WEC^{0.33}$ 6.25, $WEC^{0.33}$ 6.5 = cube root transformed faecal egg count measured in kids at 3, 5, 6.25 and 6.5 months of age respectively, figure in parenthesis represents number of kids observed for each tested class. $WEC^{0.33}$ at 6.25 and 6.5 months represent 28 and 35 days post artificial challenge measurements.

The effect of ILV on PCV, Ab and EOS at 2, 3, 5 and 6.25 months in Angora goats is shown in Table 3.4 and for Cashmere goats in Table 3.5 and for both in Figure 3.1. In Angora goats ILV had no effect ($P<0.05$) on PCV and $EOS^{0.25}$ during natural or artificial challenge. Generally the vaccinated kids had higher mean PCV and $EOS^{0.25}$ counts than the unvaccinated kids at all times of measurement. These means appeared to decrease with age during natural challenge. However, there was a significant increase in specific Log_eAb of the vaccinated Angora goats at 5 and 6.5 months old.

Specific Log_eAb increased after vaccination, rising from 5.58 at 3 months to 6.98 at 35 days post artificial challenge. In cashmere goats vaccination of kids with irradiated larvae had no significant influence ($P>0.05$) on any of the blood parameters at any times of measurement. While eosinophil

counts in Angora goats showed a marked increase following artificial challenge at 5 months, no such increase was observed in Cashmere goats in which eosinophil counts remained high throughout.

WEC 4 weeks after artificial challenge was negatively correlated with daily change in liveweight during the period in both Angora (P<0.001) and Cashmere (P=0.034) goat kids (Figure 3.2). In both cases growth was slowed by about 5g/d through the period for every additional 1000 epg 4 weeks after challenge.

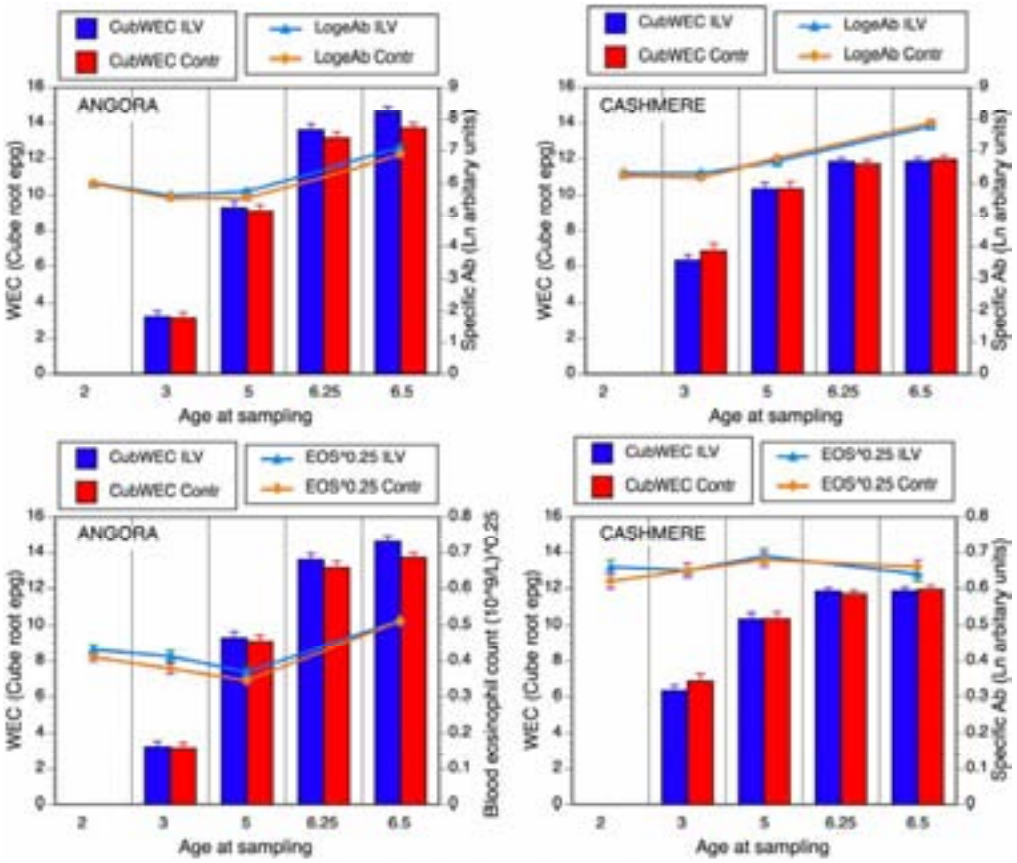

Figure 3.1 Association of key variables over time in Angora (left panels) and Cashmere (Right panels) goats following vaccination or no vaccination with ILV at 1 and 2 months of age. Least squares means and standard errors from Tables 3.2-3.5.

Table 3.4 Least squares means ± se of packed cell volume (PCV, %), log transformed specific antibody level (Log_eAb) and fourth root transformed blood eosinophil count ($EOS^{0.25}$) at natural challenge (2, 3 and 5 months of age) and after artificial challenge (6.5 months of age) of Angora goat kids vaccinated with irradiated L_3 larvae of *T. colubriformis*.

	Age (months)			
Traits	2	3	5	6.5
PCV (%)				
P value of test	0.131	0.477	0.174	0.430
Overall mean	27.90±0.24 (222)	24.41±0.20 (219)	20.72±0.18 (215)	20.92±0.18 (215)
Vaccinated	28.22±0.35 (112)	24.46±0.30 (111)	20.86±0.25 (110)	21.11±0.25 (110)
Unvaccinated	27.59±0.34 (110)	24.18±0.29 (108)	20.62±0.24 (105)	20.85±0.25 (105)
Log_eAb (Ab units)				
P value of test	0.803	0.392	0.028	0.034
Overall mean	6.01±0.03 (222)	5.58±0.03 (219)	5.64±0.05 (215)	6.98±0.05 (215)
Vaccinated	6.02±0.05 (112)	5.62±0.05 (111)	5.75±0.07 (110)	7.10±0.07 (110)
Unvaccinated	6.00± 0.05(110)	5.53±0.04 (108)	5.54±0.07 (105)	6.87±0.07 (105)
P value of test				
$P(f \geq F \mid H_0)$	0.438	0.099	0.314	0.899
Overall mean	0.421±0.001(222)	0.397±0.012(219)	0.358±0.012(215)	0.514±0.012(215)
Vaccinated	0.433±0.012(112)	0.414±0.016(111)	0.369±0.016(110)	0.515±0.016(110)
Unvaccinated	0.411±0.012(110)	0.379±0.015(108)	0.345±0.016(105)	0.513±0.016(105)

PCV = Packed cell volume; Log_eAB(10+Ab) = logarithmic transformed antibody; $EOS^{0.25}$ = fourth root transformed eosinophil count, figure in parenthesis represents number of kids observed for each tested class. Measurements at 6.5 months represent 35 days post artificial challenge measurements.

Table 3.5 Least squares means ± se of packed cell volume (PCV,%), log transformed specific antibody level (Log_eAb) and fourth root transformed blood eosinophil count ($EOS^{0.25}$) at natural challenge (2, 3 and 5 months of age) and after artificial challenge (6.5 months of age) in Cashmere goat kids vaccinated with irradiated L_3 larvae of *Trichostrongylus colubriformis*.

	Age (months)			
Traits	2	3	5	6.5
PCV (%)				
P value of test	0.244	0.4748	0.4147	0.3367
Overall mean	29.23±0.20 (244)	26.23±0.19 (244)	26.98±0.21 (243)	26.32±0.19 (240)
Vaccinated	29.00±0.27 (124)	26.10±0.27 (124)	26.81±0.30 (123)	26.14±0.27 (123)
Unvaccinated	29.47±0.28 (120)	26.37±0.27 (120)	27.16±0.30 (120)	26.51±0.27 (117)
Log_eAb (Ab units)				
P value of test	0.3536	0.0578	0.2710	0.2238
Overall mean	6.29±0.05 (244)	6.26±0.04 (244)	6.72±0.04 (243)	7.83±0.05 (240)
Vaccinated	6.33±0.06 (124)	6.33±0.05 (124)	6.67±0.06 (123)	7.78±0.06 (123)
Unvaccinated	6.24±0.06 (120)	6.19±0.06 (120)	6.77±0.06 (120)	7.89±0.07 (117)
$EOS^{0.25}$ (x 10^9/L)				
P value of test	0.1875	0.9331	0.7683	0.5198
Overall mean	0.64±0.01 (244)	0.65±0.01 (244)	0.69±0.02 (243)	0.65±0.01 (240)
Vaccinated	0.66±0.02 (124)	0.65±0.02 (124)	0.69±0.02 (123)	0.64±0.02 (123)
Unvaccinated	0.62±0.20 (120)	0.65±0.02 (120)	0.68±0.02 (120)	0.66±0.02 (117)

PCV = Packed cell volume; Log_eAB = logarithmic transformed antibody; $EOS^{0.25}$ = fourth root transformed eosinophil count, figure in parenthesis represents number of kids observed for each tested class. Measurements at 6.5 months represent 35 days post artificial challenge measurements.

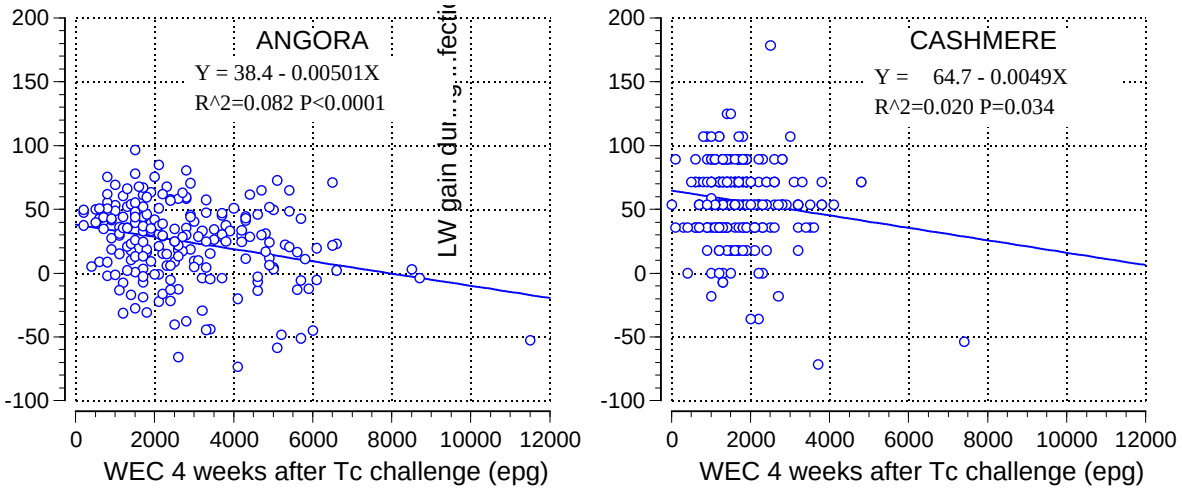

Figure 3.2 Association between WEC 4 weeks after challenge with 10,000 larvae of *T. colubriformis* and daily change in liveweight (g) over the 4 week challenge period in Angora (Right panel) and Cashmere (left panel) kids challenged at 5 months of age.

3.4 Discussion

The use of irradiated *T. colubriformis* larvae in this study failed to confer resistance to natural infection with *H. contortus* in either Angora and Cashmere goat kids as evidenced by the non-significant difference in WEC between the immunised and non-immunised kids at 3 and 5 months of age. This might have been due to a number reasons: i) exposure of the kids to a different nematode species (*H. contortus*) at from that which they were vaccinated against (*T. colubriformis*); ii) the kids were incapable of mounting a protective immune response to the parasite at the age at which they were immunised; iii) interference with an antigenic stimulation by colostrum-acquired antibodies and iv) the use of single dose, followed by a booster dose of ILV may not be provide sufficient antigenic stimulation. Each of these is discussed in turn.

As discussed in section 1.2.3 the immune response to exposure to each worm genus is specific for that genus, although the capacity of the host to mount an immunological response appears to be similar across worm species. However, although the natural challenge following ILV was predominantly *H. contortus*, infections with *Trichostrongylus spp* were present in both herds at both 3 and 5 months indicating that some level of challenge with the homologous worm genus after vaccination. It is possible that this level of infection was below that required to stimulate or maintain an immune response (Walkden-Brown and Maslen, 2002), and if this was the case, the 3 month gap between booster vaccination and artificial challenge would be sufficient to ensure a marked decline in immunity. Barnes and Dobson (1993) showed that in lambs which had developed immunity to *T. colubriformis* over a 6 week period, the immunity was labile and mostly lost within 12 weeks in the absence of ongoing challenge.

The early age of vaccination is potentially a cause of vaccination failure despite the success at inducing immunity in neonatal lambs reported by Emery *et al.* (1999). This may be due to a failure of the neonatal immune system or maternal antibody interference with the vaccinating antigens as has been suggested by (Urquhart *et al.* 1966). Our finding is in agreement with other reports of failure to induce effective immunity with irradiated *H. contortus* vaccines earlier than 6 months of age in lambs (Windon 1991). Numerous trials of ILV on nematode parasites of sheep, including *H. contortus*, have shown that efficacy (based on WEC) in young animals (less than 6months) is poor, but was consistently high in older animals (Gray 1997); (Mulligan *et al.* 1961). Irradiated *H. contortus* larvae were reported to induce less protection in 2-month-old Suffolk/Greyface lambs than in adults, again suggesting the involvement of an age- or bodyweight-related factor (Smith and Angus 1980). However, there are conflicting reports on the effect of ILV on WEC in lambs depending on the worm species, method and the experimental conditions. There are number of reports of positive effects of irradiated *Trichostrongylus spp* vaccination described for lambs vaccinated under controlled pen

conditions (Windon *et al.* 1984), 85-91% reduction in worm count in 3 month old lambs trickle infected with *T. colubriformis* for 6 weeks (Emery *et al.* 1999) and a partial protection at 3 months in lambs trickled infected at 1 month of age (McClure *et al.* 1998). Although there was a failure of protection in the present experiment, there was evidence that the immunization had worked to some extent because it resulted in a significant rise in the level of circulating IgG directed against *T. colubriformis* (Log_eAb) after Angora kids were challenged with this parasite at 5 months of age (Table 3.4). There was a strong trend in the same direction in Cashmere goats at 3 months of age.

The use of two ILV challenges a month apart may not have provided sufficient antigenic stimulation to develop protective immunity. In most studies in which immunity to worms is induced, intensive multiple infections are required (Barnes and Dobson, 1993; Balic *et al.*, 2000) with both the duration of challenge and the frequency of challenge important. This would be particularly true in the case of ILV where patent infections do not follow a challenge. In the study of Emery *et al.* (1999) in which immunity was successfully induced in neonatal lambs, the lambs were trickle immunised 3 times weekly for 6 weeks from birth.

The significant elevation of WEC following later challenge with *T. colubriformis* in Angora goats in this study was quite unexpected and contrary to other reported studies where WEC has been reduced or unchanged following ILV. Possibly some form of immunological tolerance may have been induced by the vaccination regimen.

3.4.1 Summary

The results did not support our hypothesis, with ILV having no effect on WEC in Cashmere goats, and increasing, rather than decreasing WEC in Angora goats 35 days after artificial challenge with *T. colubriformis*. However, some level of immune response was observed in Angora at 5 and 6.5 months old. The reason for the anomalous WEC response to ILV in Angoras is unclear. The overall lack of a marked response to vaccination in both breeds may reflect the mode of application (bolus rather than trickle), lack of responsiveness in neonatal goats or the presence of concurrent challenge with other worm species.

4. Pedigree verification and parentage analysis

4.1 Introduction

The second phase of the project involved pedigree verification using DNA tests. While mothering up and pedigree recording of the Angora goats occurred soon after birth there had been some recorded instances of bucks escaping and moving to other mating groups on this property. Cashmere goats were pedigreed by mothering up at marking time, which is potentially subject to greater error than mothering up at birth. However there were fewer instances of recorded buck ingress into mating groups on this property.

Traditionally, pedigree verification in livestock species has been performed using blood groups and protein polymorphisms but this has now been largely supplanted by molecular techniques based on microsatellite DNA markers. A large number of highly polymorphic microsatellites are now characterized and mapped in goats (Schibler *et al.*, 1998). The accuracy of DNA-based verification is much greater than for blood markers because of greater degree of variation (many alleles) and availability of a virtually unlimited supply of markers. It is important to use a highly informative set of markers with easily scorable alleles for parentage inference. Highly informative MS markers allow parentage analysis with a high degree of confidence. Prior to the use of the MS markers in parentage analysis, an evaluation of the power of a panel of the studied markers in parentage testing is recommended in order to minimise the possible confounding effects of pedigree errors. The power of MS markers in pedigree verification is evaluated using the probability of parental exclusion. For example the probability of exclusion for 22 MS markers was found extremely high (>0.9999) in domestic cattle and goats (Heyen *et al.*, 1997; Luikart *et al.*, 1999; Schnabel *et al.*, 2000). The more the number of markers used, the higher (close to 1.0) the probability of exclusion. A likelihood-based testing procedure (as implemented in the CERVUS program) often has been widely utilized to identify true parent with high statistical confidence among the many candidate parents (Schnabel *et al.*, 2000; Isberg *et al.*, 2004).

Errors in breeding records are common and range between 4.85%-15.5% in sheep and 2%-22% in cattle (Barnett *et al.*, 1999; Visscher *et al.*, 2002; Al-Atiyat, 2004). Incorrect pedigree records, especially errors in sire identification, could slow down genetic progress and lead to a loss in genetic gain (Visscher *et al.*, 2002). Pedigree errors also influence correct estimation of genetic parameters. The resolution of pedigree disputes based on genotype data allows us to obtain accurate estimates of heritability and genetic correlations, which are used as prime tools for developing selection programs in livestock.

A discrepancy between a putative parent and offspring arises when one or a few loci contain mismatches. Beside genotyping errors, mutations and null alleles which are also the result of mutation could be invoked to explain such mismatches. It is well known that mutation rates in many MS markers are high enough (e.g. $1x10^{-4}$ per locus) to occasionally cause errors in parentage testing (Luikart *et al.*, 1999). Null alleles (non amplifying alleles) have been reported in many organisms such as humans and plants. The presence of null alleles could be due to poor primer annealing, mutations in one or both flanking primers, PCR failure and population genetic phenomena such as a Wahlund effect (excess of homozygosity observed after two subpopulations with different allele frequencies are lumped together) or inbreeding (Dakin and Avise, 2004). Finding and quantification of frequencies of mutation and null alleles are generally accomplished by conducting controlled parentage studies.
The objectives of this section of the project were as follows:

- to evaluate 14 MS markers for their effectiveness in parentage analysis in the two goat breeds
- to quantify the level of pedigree errors in sire and dam identification in Australian Angora and Cashmere goat herds using DNA-based parentage analysis.
- to determine the presence of null alleles or mutations.

4.2 Materials and methods

Details of DNA sampling and processing, microsatellite marker selection and PCR amplification and parentage analysis methods are provided in sections 2.4.7, 2.48 and 2.51 respectively. The CERVUS program (Marshall *et al.*, 1998) was utilized to evaluate the effectiveness of both markers and infer parent-offspring assignment in the Angora and Cashmere resource herds using their microsatellite genotypic data with 428 and 548 and animals fully genotyped respectively.

4.3 Results

4.3.1 Effectiveness of MS markers

Deviations from Hardy-Weinberg equilibrium (HWE) due to linked loci and non-random association between loci could bias the calculation of genotype frequencies and exclusion probabilities from allele frequencies. The *OarCP73* locus in the Cashmere and *SRCRSP07* locus in the Angora populations demonstrated significant departure from HWE. Therefore, there were two violations of the assumptions affecting the expectation of loci being in HWE and in linkage equilibrium, which were taken into considerations when calculating the probabilities of parental exclusion.

4.3.2 Probability of parental exclusion

The exclusion probabilities for each population were determined based on MS genotypes from 428 and 548 animals from the Angora and Cashmere populations, respectively. Table 4.1 shows the number of alleles, *PIC* and the exclusion probabilities (*PE1* and *PE2*) for each locus, as computed by CERVUS. Exclusion probability one (*PE1*) assumes that genotypes are known for an offspring and an alleged parent (one parent genotyped), but genotypes not available for a confirmed parent (one parent missing). Exclusion probability two (*PE2*) assumes that genotypes are known for the offspring, one confirmed parent, and one alleged parent (both parents genotyped). Therefore, *PE2* is always higher than *PE1* (Figure 4.1).

The extent of exclusion probabilities (*PE1* and *PE2*) for each locus was different in the two populations (Table 4.1). For both populations, the lowest *PE* values were observed at the *SRCRSP07* and *INRABERN185* loci. Angoras had the highest *PE* at *BM1258*, while Cashmeres had the highest *PE* at the *SRCRSP05* locus. As can be seen from Table 4.1 that the values of *PE1* and *PE2* for each locus increase with an increase of *PIC* values. *PIC* and *PE1* or *PE2* had a strong positive correlation (Table 4.2). Therefore, loci with a high *PIC* give high *PE* values and the loci with small *PIC* give low *PE* values (Table 4.1). Consequently the power of *PE* depends heavily on the level of *PIC* at each locus.

Table 4.1 The number of alleles, *PIC* and exclusion probabilities for each locus by population

	Angora					Cashmere			
Locus[a]	Allele No.	*PIC*	*PE1*[b]	*PE2*[c]	Locus[a]	Allele No.	*PIC*	*PE1*[b]	*PE2*[c]
BM1258	9	0.840	0.550	0.712	*SRCRSP05*	11	0.834	0.541	0.705
OarCP73	13	0.827	0.527	0.693	*BM1258*	10	0.821	0.515	0.683
RM096	10	0.782	0.451	0.628	*TGLA53*	10	0.783	0.451	0.629
TGLA53	7	0.768	0.423	0.601	*OarCP73*	13	0.775	0.441	0.617
BM1818	9	0.758	0.408	0.586	*RM096*	9	0.761	0.421	0.601
LSCV44	9	0.730	0.376	0.554	*LSCV44*	11	0.708	0.351	0.533
ILSTS11	5	0.726	0.364	0.543	*ILSTS11*	8	0.699	0.337	0.517
ILSTS029	8	0.720	0.362	0.544	*OarFCB020*	6	0.699	0.334	0.510
SRCRSP05	9	0.712	0.357	0.540	*BM1818*	8	0.637	0.275	0.463
INRABERN172	8	0.711	0.348	0.524	*INRABERN172*	7	0.610	0.246	0.411
INRA063	4	0.653	0.280	0.453	*ILSTS029*	13	0.603	0.241	0.418
OarFCB020	7	0.537	0.188	0.353	*INRA063*	5	0.600	0.235	0.404
SRCRSP07	4	0.507	0.168	0.302	*INRABERN185*	7	0.516	0.168	0.333
INRABERN185	3	0.403	0.101	0.233	*SRCRSP07*	5	0.405	0.107	0.237

[a] Loci for each population in decreasing order of power of of *PE*. * *PE1* = probability of exclusion for each locus when neither parent is known and [†] *PE2* = probability exclusion for each locus given one parent is already known.

Table 4.2 Correlation coefficients between *PE1*, *PE2*, and *PIC* in two populations

Correlation between	Angora_Aus	Cashmere_Aus
PIC and *PE1*	0.98	0.98
PIC and *PE2*	0.99	0.99

Total exclusion probabilities (*PEt*) for a given set of loci were computed using CERVUS. Figure 4.1 shows the trend of total probability of exclusion (*PEt1* and *PEt2*) and mean *PIC* for MS marker panels based on increasing number of loci. The total exclusion probabilities (*PEt1* and *PEt2*) increased with each extra marker genotyped until approximately 100% was approached (Figure 4.1). However, the rate of increase in *PEt* values depended on *PE* or *PIC* values of each extra marker. For example, when we ordered the loci in descending order of *PE* or *PIC* values for each locus, the total exclusion probabilities (*PEt*) increased rapidly from a high base, but mean *PIC* decreased gradually from a high initial value (Figure 4.1.A). When loci were included in ascending order of *PE* or *PIC* values, the total exclusion probabilities increased sharply from a low base and the mean *PIC* rose gradually with each extra marker genotyped (Figure 4.1.B).

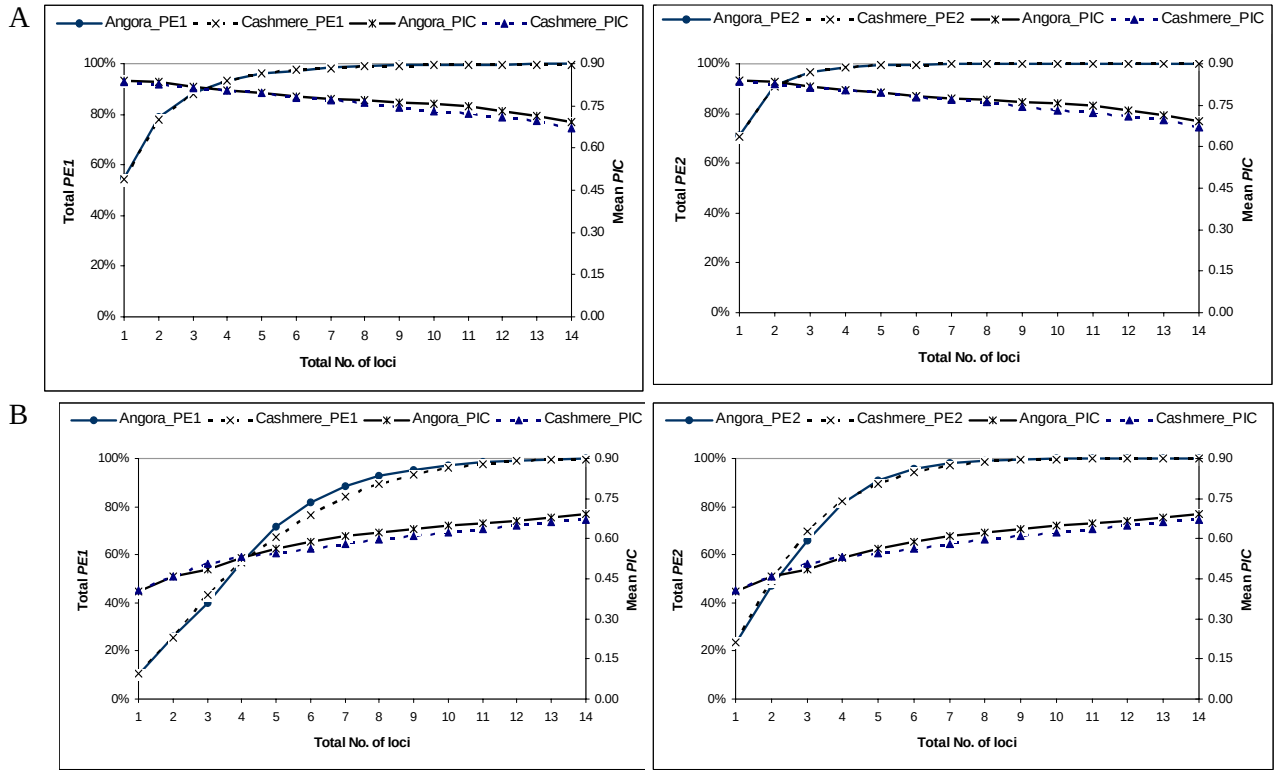

Figure 4.1 The relationship between exclusion probabilities (*PE1* and *PE2*), polymorphic information content (*PIC*) and total number of MS loci used for genotyping. Figure A represents the relationship with loci ordered in descending order of PE1 and PE2 values. Figure B represents the relationship with loci ordered in ascending order of *PE1* and *PE2* values.
- Total No. of loci = a given set of loci utilized for parentage testing
- Total *PE1* and *PE2* = the total probabilities of exclusion when all loci are combined (total exclusionary power)
- Mean *PIC* = average polymorphic information content for a given set of loci

In the first case (loci in descending order of *PE* or *PIC*) the increase in total *PE1 (PEt1)* becomes negligible after eight markers (with *PIC*=0.763), providing a *PEt1* value of 99.3%, whereas using *PEt2*, it becomes negligible after using the first five markers (with *PIC*=0.795) providing a *PEt2* value of 99.4%. In the second case (loci in ascending order of *PE* or *PIC*), twelve markers (with *PIC*=0.668) and five markers (*PIC*=0.634) respectively were adequate to exclude the incorrect parent with total exclusionary power of 0.991 (*PEt1*) and 0.996 (*PEt2*). The trend of total probability of exclusion (*PEt1* and *PEt2*) and mean *PIC* for the two populations was very similar (Figure 4.1).
The total exclusion probabilities, *PEt1* and *PEt2*, using all fourteen loci (mean *PIC*=0.7) for each population were 0.997 and 0.9999, respectively (Table 4.3). Thus if the alleged parent is not the true parent, the 14 markers will exclude the alleged parent with *PEt1* of 0.997 and *PEt2* of 0.9999. As noted at 4.3.1, *SRCRSP07* deviated significantly from HWE, and linkage disequilibrium was found between the loci *LSCV44*, *OarCP73*, and *BM1818*. In order to avoid any potential bias in estimation of *PE*s at the studied MS panel due to significant deviations from HWE and presence of LD, the total exclusion probabilities (*PEt1* and *PEt2*) were recomputed, excluding or including *SRCRSP07*, *LSCV44*, *OarCP73*, and *BM1818* loci (Table 4.3). The *PEt1* and *PEt2* were virtually unaltered after excluding *SRCRSP07* in both populations. Even after removing all four loci (*SRCRSP07*, *LSCV44*, *OarCP73*, and *BM1818*), the *PEt1* and *PEt2* declined only slightly to 98.72% indicating that the parentage assignment using the remaining 10 loci was still highly effective in both populations.

Table 4.3 Total exclusion probabilities (*PEt*) excluding various loci that were a potential source of inaccuracy.

	Angora_Aus		Cashmere_Aus	
No. of loci	*PEt1*	*PEt2*	*PEt1*	*PEt2*
All loci	0.99816	0.99998	0.99733	0.99997
13 loci[1]	0.99777	0.99997	0.99701	0.99996
11 loci[2]	0.98950	0.99964	0.98985	0.99966
10 loci[3]	0.98724	0.99947	0.98864	0.99955

[1]Excluding *SRCRSP07*; [2]Excluding *LSCV44*, *OarCP73*, and *BM1818*; [3]Excluding *SRCRSP07*, *LSCV44*, *OarCP73*, and *BM1818*.

4.3.3 Parent-offspring genotype mismatches

Following the initial stage of parentage assignment several mismatches between parent and offspring were identified. Repeat genotypings were performed and after correcting for genotyping errors, 33 mismatches still remained. Of these 33 mismatches, 7 were accounted for by apparent mutations at 4 loci in Angoras and 3 loci in Cashmeres (Table 4.4).

Table 4.4 Mutations observed in studied MS markers in the two populations.

Population	Locus	Repeat motif[a]	Offspring genotype[b]	Sire genotype	Dam genotype
Angora	*BM1818*	$(GT)_n$	254/260	258/260	258/260
	SRCRSP05	$(AT)_n(GT)_n$	170/174	170/170	170/176
	INRABERN172	dinucleotide	242/244	238/242	236/236
	TGLA53	$(GT)_n$	119/121	119/119	119/127
Cashmere	*RM096*	$(CA)_n$	102/110	102/106	116/120
	BM1818	$(GT)_n$	254/254	254/254	256/260
	TGLA53	$(GT)_n$	123/127	121/127	121/139

[a]From the references in literature. [b]Underlined number represents the mutant alleles;

The remaining 26 parent-offspring genotype mismatches were found at only one locus, *SRCRSP07*, in the Angora population and there is convincing evidence that these are due to the presence of null alleles that fail to amplify in the PCR reaction. The null alleles observed in the Angora population may be the consequence of an old mutation with a single origin (starting from a common ancestor) several generations back. Based on our observations, the observed null alleles seemed to be only paternally transmitted (Table 4.5). This appearance may be due to the fact that sires have many progeny through whom the null alleles are easily recognized as mismatches of sire-progeny genotype pairs. Dams generally have only one progeny and therefore, if a dam is heterozygous for a null allele at a particular locus, the chance to recognize null heterozygotes (123/null or 129/null) through its sole progeny is very small and she will be scored as a homozygous dam (123/123 or 129/129) instead of a null heterozygous dam (123/null or 129/null). The CERVUS software program used an iterative likelihood approach to estimate the null allele frequency. The null allele frequency at *SRCRSP07* was 0.07 in the Angoras. As the frequency of null alleles was low, we retained *SRCRSP07* in subsequent analysis.

4.3.4 Success rate of parentage analysis

After excluding the thirty-three cases of unmatched genotypes due to mutations or null alleles, the feasibility of parentage analysis using the fourteen markers was examined by simulation through CERVUS. The simulation results showed the success rate of parentage analysis using the fourteen loci was 100% at a confidence level of 95% with no unresolved cases. This success rate is calculated on the assumption that there were no genotyping errors (error rate = 0%) and all putative parents were genotyped.

Tables 4.5 and Table 4.6 summarize the parentage analysis in Angora and Cashmere resource herds by year. Paternity assignment analysis was performed under the assumption that neither parent is known.

After two rounds of parentage analysis (including and then excluding 33 genotypes), 97% (289/297) and 96% (402/417) of the offspring were unambiguously assigned parentage to the correct sire in the Angora and Cashmere herds, respectively. In total, 8 offspring in Angoras and 15 offspring in the Cashmeres were not assigned parentage to any sire after the second round because some sires were not available for genotyping.

Table 4.5 Summary of parentage analysis for Angora goats

Year	Paternity assignment				Kids with dams' DNA	Maternity assignment			
	No. of kids tested	No. of candidate sires	No. of kids assigned to sire (%)*[a]	not resolved (%)*[a]		No. of kids tested	No. of candidate dams	No. of kids assigned to dam (%)*[b]	not resolved (%)*[b]
2000	76	3	70 (92%)	6 (8%)	23	20	20	18 (90%)	2 (10%)
2001	113	6	111 (98%)	2 (2%)	69	68	64	65 (96%)	3 (4%)
2002	108	6	108 (100%)	0 (0%)	91	91	89	83 (91%)	8 (9%)
Total	**297**	**11**	**289 (97%)**	**8 (3%)**	**183**	**179**	**126**	**166 (93%)**	**13 (7%)**

(%)*[a] indicates the success rate of parentage analysis when neither parent is known, whereas (%)*[b] shows the success rate when one parent is known.

Table 4.6 Summary of parentage analysis for Cashmere goats

Year	Paternity assignment				Kids with dams' DNA	Maternity assignment			
	No. of kids tested	No. of candidate sires	No. of kids assigned to sire (%)*[a]	not resolved (%)*[a]		No. of kids tested	No. of candidate dams	No. of kids assigned to dam (%)*[b]	not resolved (%)*[b]
2000	33	2	32 (97%)	1 (3%)	-	-	-	-	-
2001	149	5	139 (93%)	10 (7%)	30	24	29	20 (83%)	4 (17%)
2002	73	9	71 (97%)	2 (3%)	26	25	26	23 (92%)	2 (8%)
2003	162	8	160 (99%)	2 (1%)	150	148	131	133 (90%)	15 (10%)
Total	**417**	**20**	**402 (96%)**	**15 (4%)**	**206**	**197**	**172**	**177 (90%)**	**20 (10%)**

(%)*[a] indicates the success rate of parentage analysis when neither parent is known, whereas (%)*[b] shows the success rate when one parent is known.

Of all kids genotyped, only 62% (187/297) and 50% (206/417) had their dam's DNA information available in Angora and Cashmere goat herds, respectively. The maternity assignment is the case where a confirmed sire-offspring pair was assigned against a putative dam (one parent is known). The results from maternity assignment analysis revealed that 93% (166/179) and 90% (177/197) of offspring were unambiguously assigned parentage to the correct dam in Angora and Cashmere studs, respectively (Tables 4.5 and 4.6). In total, 13 Angora and 20 Cashmere offspring were not assigned to any dams because of absence of potential dams' genotype data. Additionally, 4 of 8 Angora kids and 9 of 15 Cashmere kids, whose sire was unresolved, were assigned parentage to the correct dam using the case where neither parent is known.

If we look at the number of unresolved paternity and maternity assignments by year, the highest proportion was observed in 2000 and 2001. Collection of DNA samples started in 2002 and by this time many dams as well as some sires were slaughtered or sold and therefore the DNA samples were unavailable for these animals. Hence, even though a highly effective panel of MS loci was used, the rate of unresolved cases of parentage assignment depended on a proportion of genotyped parents.

4.3.5 Pedigree recording errors

The results of DNA-based parentage analyses a significant error rate in breeding records, being 13.80% and 8.39% for sires and 9.84% and 14.56% for dams in the Angora and Cashmere populations, respectively (Table 4.7). Consequently the results of parentage analysis rather than breeding records were utilized in subsequent quantitative genetic analyses in both populations.

Table 4.7 Number and percentage of kids with incorrect sire and dam pedigrees

Population	No. of kids	Sire breeding record		Percentage of kids with incorrect sire pedigree (%)	No. of kids	Dam breeding record		Percentage of kids with incorrect dam pedigree (%)
		correct	incorrect			correct	incorrect	
Angora	297	256	41	13.80	183	165	18	9.84
Cashmere	417	382	35	8.39	206	176	30	14.56
Total	**714**	**638**	**76**	**10.64**	**389**	**341**	**48**	**12.34**

In Angora goats, the number of errors in breeding records was higher for sires than that for dams, whereas it was lower for sires than that for dams in the Cashmere resource herd (Table 4.7).

4.4 Discussion

The high probability of parental exclusion (*PEt*) confirmed the effectiveness of molecular markers for pedigree verification. As our results showed, the panel of fourteen MS loci had great power to infer correct parentage. The total probability of excluding a wrongly assigned sire (*PEt1*) for the 14 loci was close to 99.7% and the total probability of excluding a wrongly assigned dam (*PEt2*) was nearly 99.99%. This is similar to the power of two multiplex systems consisting of 11 markers each for goats (Luikart *et al.*, 1999). Finding the minimal number of loci which will provide sufficient power of parentage assignment is important for practical implementation. Our data clearly indicated that the required minimal number of MS markers in pedigree analysis depends on the *PIC* of each MS marker. Obviously, the markers with high *PIC* are more useful within a multiplex panel for parentage testing than the others. A set of fewer MS markers with high *PIC* would give the same result as that of more markers with low *PIC*. Therefore choosing fewer MS markers with high *PIC* reduces the cost of DNA-based parentage analysis. In cases where neither parent is known, at least eight markers with mean *PIC*=0.763 or twelve markers with mean *PIC*=0.668 were required to exclude the incorrect parent with 99.1% accuracy. A similar finding was reported in three Australian Merino sheep populations by Al-Atiyat (2004), where the minimal number of required MS markers was eight (*PE*=99.6%). On the other hand, if one parent is already known, our study has shown that only five loci with *PIC*=0.795 can have sufficient power (99.4%) to exclude the putative second parent.

An assumption for correct estimation of probability of exclusion was that loci should be in HWE and in linkage disequilibrium. Any violations of this assumption could cause a bias of the probability of parental exclusion. However, small deviations from HWE at individual loci will not necessarily lead to significant bias (Marshall *et al.*, 1998). Therefore, the deviation from HWE for *OarCP73* ($P = 0.02$) in the Cashmere population was ignored. The highly significant deviation on the *SRCRSP07* locus was considered when we estimated *PEt*. However, almost no change in *PEt* values was observed in the both populations when excluding *SRCRSP07* (Table 4.3). *PEt* were also recomputed after removal of loci with major deviation from HWE or which were in linkage disequilibrium. Even removing all four loci causing these violations, the remaining set of MS markers had adequate exclusionary power to determine parentage.

The *SRCRSP07* locus also presented a significant heterozygote deficiency in both Angora and Cashmere populations. A significant heterozygote deficiency in *SRCRSP07* has also been reported other goat populations (Luikart *et al.*, 1999). A direct examination of genotypes in parent-offspring pairs revealed that a highly significant heterozygote deficit at *SRCRSP07* in Angoras resulted from null alleles. Therefore the heterozygote deficits at *SRCRSP07* reported as a typical phenomenon in some goat breeds could be due to the presence of null alleles. Null alleles are nonamplifying alleles due to mutations at PCR priming sites (Ibarguchi *et al.*, 2004). Generally, the flanking regions of repeats (priming sites) have been found to be neutral and conserved across some species because the mutation rates of flanking DNA are extremely low compared with microsatellite mutation rates. Brohede and Ellegren (1999) observed that the first nucleotide positions flanking repeats had significantly higher mutation rates than that in sequences further away. The mutation that occurred at the PCR priming site (and thus did not produce any PCR product) of *SRCRSP07* might be old. One could hypothesize that the old mutation originated from a single source spread across several generations of some goat populations, in our case the Angora resource population. The null allele

frequency at *SRCRSP07* in Angoras was small and estimated as 7%. According to a survey by Dakin and Avise (2004) from 233 published articles in which authors reported the suspected presence of one or more microsatellite null alleles, the frequency of null alleles was usually less than 20%. Of these 233 published papers, 13% reported that the frequency of null alleles ranged from 5% to 10%. If the estimated frequency of null alleles at *SRCRSP07* was 0.07, it is expected that 3 null homozygotes and 56 null heterozygotes should be observed among the 428 genotyped animals in the Angora population. However no null homozygotes (null/null or no PCR product) were observed. This may be a chance finding or suggest that null homozygotes suffer a selective disadvantage during the juvenile stage as Van Treuren (1998) found in studies of locus *20H7* in a bird species. In total, thirty-two null heterozygotes were observed which was also lower than expected number of null heterozygotes. This suggests that some dams who appeared to be homozygous (123/123, 127/127 or 129/129) at *SRCRSP07* may have been null heterozygote dams (123/null, 127/null or 129/null). However, it is very hard to demonstrate the existence of null heterozygote dams unless the dam has many offspring. In the future, avoiding use of *SRCRSP07* marker in parentage and population genetics analyses is recommended because inclusion of this locus will result in incorrect estimation of allele frequency and it will cause false exclusions when null heterozygotes are scored incorrectly as non-null homozygotes.

The overall estimate of pedigree error rates in both resource herds was 10% for sire and 12% for dams. This is within range of pedigree errors observed in sheep (4.8-15.5%) and in cattle (2-22%) (Barnett *et al.*, 1999; Visscher *et al.*, 2002; Al-Atiyat, 2004). The error rates of sire and dam pedigree were different in the two goat herds higher accuracy for dam pedigree in the Angora herd, and for sire pedigree in the Cashmere herd (Table 4.7). This finding probably reflects the management practices on the two farms. The Angora goat farm did not have fencing of sufficient quality to maintain complete separation of males during mating and the assignment of kid to dam was mostly done within 24 hr of birth. On the other hand, the Cashmere goat farm had high quality fences ensuring good segregation of males and sire pedigree, but the dam pedigree recording was performed when kids were 2-3 months old, with consequently lower accuracy. Collecting dam pedigree information at older ages using “mothering up” techniques based on proximity to certain does or suckling from a particular doe is risky because some kids at older ages manage to suckle any does or some does allow others’ kids suckle them. Actually the error rate of maternal pedigree of 14.6% in the cashmere goats is surprisingly low given this method of determination. On the other hand in the Angoras a significant level of error (9.4%) was still observed following mothering up in the first hours or days after birth. This might be due to some does failing to keep their litter together, kid stealing, and kid separation or the sheer numbers of does and kids intermingling during peak kidding time. Therefore, accurate pedigree information using DNA-based parentage testing is recommended, where economically feasible, in breeding programs in order to reduce the management costs of pedigree recording and to ensure more accurate estimates of progeny EBVs and rate of genetic gain.

4.4.1 Summary

- The fourteen MS markers evaluated in this study provide a very high level of power (*PE* >99.7%) for both parentage testing and individual identification. The extent of *PE* depends on *PIC* and number of alleles for each marker. In order to get high *PEt* using few MS markers use of MS markers with high *PIC* is required. Using the studied loci, the minimum number of MS markers for accurate determination of parentage was 12 when neither parent is known (*PE1*) and 10 when one parent is known (*PE2*).
- Of fourteen MS markers, one locus, *SRCRSP07*, had null alleles present in the heterozygous state. This null allele was revealed by mismatches of genotypes of parent-offspring pairs. The highly significant deviation from HWE and significant heterozygote deficiency was also observed at this locus. The total *PE* value for a panel excluding *SRCRSP07* marker in both populations was still powerful for parentage testing and this marker should not be included in panels used for parentage analysis. A panel of 8-12 of the most informative markers used in this study is a suitable panel for parentage analysis (8 if one parent is known, 12 if neither parent is known).
- The overall pedigree error rate of 12.7% was within the range reported for other species. Differences in pedigree error rate between sire and dam assignment between the two resource herds were readily explained by differences in management practices on the two properties.

5. Quantitative genetic analysis of parasite-associated traits

5.1 Introduction

As detailed in Section 1.2 gastrointestinal nematodiasis (GIN) is a major problem in small ruminants worldwide, one that has been exacerbated by the widespread development of anthelmintic resistance to all major classes of broad spectrum anthelmintic. An alternative to the current chemo-centric control methods is integrated parasite control methods based on a range of chemical and non-chemical approaches. One such alternative approach is breeding for nematode resistance, and approach that is has significant uptake in the sheep industry. One of the major objectives of this project was to determine the feasibility of such an approach in the Australian fibre goat industries, something that necessitates the estimation of key genetic parameters for parasite resistance traits including their association with production traits. That is the main purpose of this section of the report.

Currently worm burdens can only be measured by sacrificing animals and conducting total worm counts on their gastrointestinal contents. This is an impractical measure for use in genetic improvement programs so correlated phenotypic markers for worm burden need to be used instead. Faecal worm egg count (WEC) is a disease-associated trait that reflects worms burdens reasonably accurately and is therefore the most widely used marker for GIN in studies on gastrointestinal nematode infections of ruminants. Animals are mainly identified as parasite resistant based on low WEC. In sheep, WEC has been found to be a heritable trait, with heritability estimates varying from low (0.12) to high (0.63) (Eady *et al.*, 1994; Woolaston and Piper, 1996; Baker *et al.*, 2003) although most accurate estimates fall between 0.2 and 0.3. Heritability estimates for WEC in goats range from low (0.04) to medium (0.37) (Woolaston *et al.*, 1992; Morris *et al.*, 1997; Jackson, 2000; Baker *et al.*, 2001; Mandonnet *et al.*, 2001; Vagenas *et al.*, 2002). However, to date there are no parameter estimates for this trait in Australian fibre-producing goats.

The use of WEC as a marker for genetic resistance to GIN has some drawbacks. WEC requires faecal collection from individual animals which is difficult and sometimes unsuccessful, requires a significant patent infection for differences between animals to be expressed, has a relatively low repeatability, a skewed distribution with a high proportion of zero counts, is not amenable to high volume automated processing of samples and requires culture of faeces for a week after collection to determine the species of GIN present. A blood-borne indicator of the immune response to GIN infection could provide an effective alternative selection criterion for resistance to GIN, overcoming most of the limitations listed above. Indeed the efficacy of selection for resistance to infection with *Trichostrongylus colubriformis* in sheep based on circulating specific IgG responses to infection has been demonstrated in sheep (Windon, 1996). Circulating eosinophil count has been investigated for this purpose (Woolaston *et al.*, 1996). Thus class of potential markers for resistance to worm infection is based upon measures of the immune response to worm infection.

Another class of potential markers is that associated with the pathology caused by infection. This might include indications of diarrhoea (eg. dag score) for the scour worms (*Trichostrongylus* and *Teladorsagia* spp.) or measures of anaemia (eg. red cell count and packed cell volume) for infection with hematophagous parasites such as *Haemonchus contortus*. These measures may reflect either the resistance of the host to worm infection (successful limitation of level of infection) or the resilience of the host in the face of infection (successful limitation of the consequences of infection) or some combination of these two traits.

In order to predict genetic progress in breeding program, it is necessary to use the most suitable genetic parameters, ideally those estimated from data from the owner's own herd data or from herds of a similar genetic background in a similar environment. In addition to estimates of heritability of given

traits it is necessary to know the genetic and phenotypic associations between traits to ensure that genetic progress can be optimised and unexpected responses to selection for correlated traits are minimized. Because, genetic variation in genetic resistance to gastrointestinal nematodes in goats has been reported between breeds (Baker *et al.*, 2001) as well as between animals within breeds (Vlassoff *et al.*, 1999) the estimation and understanding genetic and phenotypic parameters of traits related to this is a prime concern of any breeding program aiming to improve resistance to GIN.
The objectives of this section of the report are:

- to report the level of infection, immune responsiveness to, and pathogenic consequences of, nematode infections in Angora and Cashmere goats after natural (mainly *H. contortus*) and artificial (*T. colubriformis*) challenge. The parasite-associated traits used to measure this are faecal worm egg counts (WEC), specific anti-nematode antibody (IgG), circulating leukocyte counts (eosinophils, lymphocytes, neutrophils, basophils and neutrophils) and erythrocyte counts (packed cell volume, mean corpuscular volume, and mean corpuscular haemoglobin concentrate)
- to test the significance of main fixed effects (eg. year of birth, sex, birth type) for these parasite-associated traits during natural and artificial challenge
- to estimate genetic and phenotypic parameters for parasite-associated traits at 3, 5, 6.25 and 6.5 months of age after natural (3 and 5 months) and induced (artificial) nematode infection (6.25 and 6.5 months).
- to identify at which age and challenge type it is best to make measurements of parasite-associated traits.

5.2 Materials and methods

Kids of the two resource goat populations (Australian Angora and Cashmere) were exposed to natural challenge via pasture exposure up to the age of 5 months after which they were treated with anthelmintics and challenged with a fixed infection of *T. colubriformis* (artificial challenge). Data were recorded at 3 months of age, 5 months of age and 28 and 35 days post challenge at 5 months of age (6.25 and 6.5 month measurements). Full details are provided in sections 2.3.2 and 2.4.1. Estimates of genetic parameters for each variable were carried out using pedigree data corrected based on the results from parentage analysis. Details of the data transformations used and analysis are presented in section 2.5.2 and the number of records available is presented in Table 5.1.

Table 5.1 Numbers of records, number of animals, sires and dams, number of animals with sire and dam information by breed

	Angora	Cashmere
No. of records	608	796
No. of animals in pedigree	934	1210
No. of sires	14	29
No. of dams*	338	532
No. of kids with sire information	593	778
No. of kids with dam information	594	769

* Including dams which were also kids

Although data for these two breeds was not fitted together in the one analysis since they were managed separately, comparisons between the two populations are sometimes made on the assumption that major differences between two populations are more likely to be due to breed differences than environmental effects, because the farms they were raised on were in the same district and shared many common features including artificial challenge with a fixed dose of *T. colubriformis*.

5.3 Results

5.3.1 Faecal worm egg counts

Unadjusted mean arithmetic WEC by breed and year is shown in Figure 5.1. Natural challenge was significant for the first kid drops on the project (2000, 2001) but the dry springs and summers for the next two kid drops (2002 and 2003) led to low natural challenge rates with negligible counts. Artificial challenge with 10,000 T. colubriformis larvae/kid was successful in each year with the resultant WECs being higher in the later years following low levels of natural challenge, and also being higher in Angora than Cashmere goats in each year when both were challenged.

Larval differentiation (Table 5.2) indicated that in year 1 (2000 drop kids) natural challenge was predominantly with *H. contortus* (apart from at 3 months in Angora kids), while in year 2 mixed infections with *H. contortus* and *T. colubriformis* predominated. In years 3 and 4 natural infections were very low on both farms and insufficient larvae were cultured for reliable determination of genus. Following artificial challenge the predominant genus was *Trichostrongylus* in all years. The relatively lower level of infection with *H. contortus* in Angora goats at 3 months may be due to host influences, or the higher altitude of the Angora farm resulting in cooler spring temperatures and a slower rise in *Haemonchus* numbers. The free-living stages of *H. contortus* are very cold susceptible.

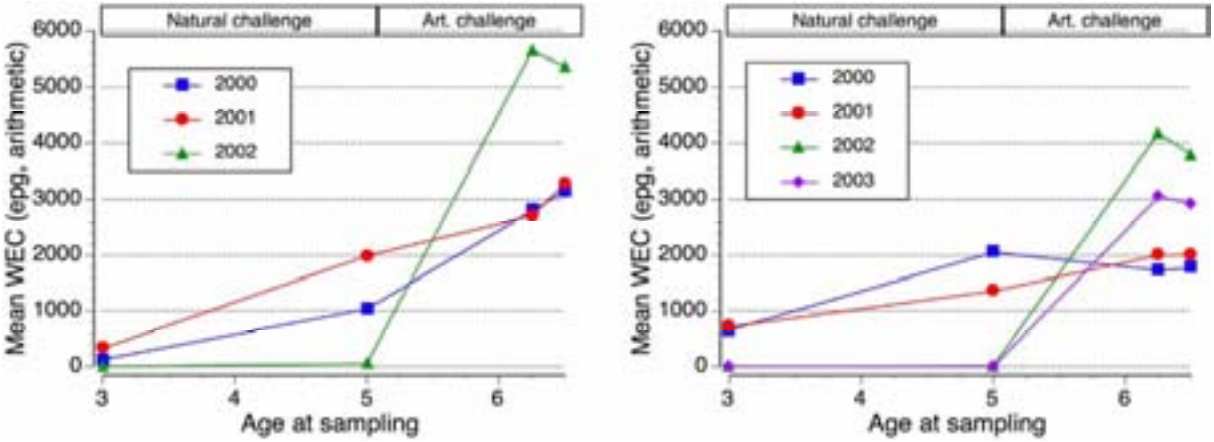

Figure 5.1 Arithmetic mean faecal worm egg counts (WEC, epg) by age at sampling in goat kids on both resource herds. At 3 and 5 months kids were subject to natural challenge while samples at 6.25 and 6.5 months of age reflect WEC following artificial challenge with 10,000 L3 of *T. colubriformis* at 5.25 months of age.

Table 5.2 The proportion (%) of different parasitic nematode genera in faecal samples of Angora and Cashmere kids during natural challenge (3 and 5 months of age) and 28 and 35 days after artificial infection with *Trichostrongylus colubriformis* (6.25 and 6.5 months of age).

Worm genus	Angora				Cashmere			
	WEC_N3m	WEC_N5m	WEC_A28d	WEC_A35d	WEC_N3m	WEC_N5m	WEC_A28d	WEC_A35d
Year 1								
Haemonchus spp.	9	91	5	8	91	91	7	7
Trichostrongylus spp.	15	6	95	89	9	7	93	92
Ostertagia spp.	76	3	-	3	0	2	-	1
Year 2								
Haemonchus spp.	32	50	3	3	52	44	1	2
Trichostrongylus spp.	54	40	90	89	41	50	94	95
Ostertagia spp.	14	10	7	8	7	6	5	3
Year 3								
Haemonchus spp.	-	66	0	0	-	-	0	0
Trichostrongylus spp.	-	18	100	100	-	-	100	100
Ostertagia spp.	-	16	0	0	-	-	0	0

- Insufficient eggs

The numbers of records, range, mean, standard error, skewness, and kurtosis (after transformation) of $WEC^{0.5}$ at various ages (3, 5, 6.25 and 6.5 months) in Angora and Cashmere goats are shown in Table 5.3. The square root transformation was applied to normalise the skewed distribution of WEC. Square root transformation dramatically reduced kurtosis for WEC and skewness for WEC at 6.25 and 6.5, but less so for WEC at 3 and 5 months because of the many zero values of WEC in these months. Mean WEC from natural challenge generally increased between 3 and 5 months of age in both populations. Mean WEC following artificial infection with *T. colubriformis* was almost two fold higher than WEC arising from natural infection from the pasture, indicating that the artificial challenge was more effective at inducing infection due to the large dose of infective larvae given in a single bonus. Except at 3 months, Cashmere kids had lower WEC than Angora kids.

Table 5.3 Number of observations, means and standard deviations (SD) of square-root-transformed faecal worm egg counts (WEC, $epg^{0.5}$) at 3 and 5 months of age and 28 and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 and 6.5 months of age) in Angora and Cashmere goats

Breed and age	No. of records	Range	Mean	SD	Skewness	Kurtosis
Angora						
3 mo	594	0-63	7.6	9.8	1.39	2.61
5 mo	588	0-95	26.0	19.8	0.32	-0.54
6.25 mo	561	0-114	57.1	18.9	0.09	-0.09
6.5 mo	572	0-117	59.4	17.2	0.19	0.56
Cashmere						
3 mo	776	0-89	14.1	16.0	1.09	1.10
5 mo	777	0-117	23.6	23.7	1.02	1.07
6.25 mo	758	0-96	46.6	15.1	-0.01	0.22
6.5 mo	758	0-93	46.4	13.86	0.04	0.30

Faecal worm egg counts: Fixed effects

The significance of main effects and their first order interactions in Angora and Cashmere goats are shown in Table 5.4. Generally, birth type, sex and exact age fitted as a covariate had no significant influence on WEC at all ages in both populations, whereas year of birth had a significant effect (P<0.001). For both breeds at 3 months and Angoras at 5 months kids born in 2001 had higher faecal egg counts (WEC) than those born in 2000, 2002, and 2003 (Table 5.5). This variation was associated with differences in rainfall during these years with increased infectivity associated with higher rainfall. At 6.25 and 6.5 months of age, kids of both breeds born in 2002 had higher WEC than kids born in other years.

Table 5.4 Significance of main fixed effects and interactions for WEC at 3 and 5 months of age and 28 and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 and 6.5 months of age) in Angora and Cashmere goats

	Fixed effects							
	Angora				Cashmere			
Variable	Birth type	Year	Sex	Age	Birth type	Year	Paddock	Paddock:Year
WEC_N3m	NS	***	NS	NS	NS	***	***	***
WEC_N5m	*	***	NS	NS	NS	***	***	***
WEC_A28d	NS	***	NS	NS	NS	***	*	NS
WEC_A35d	NS	***	NS	**	NS	***	***	NS

*** = P<0.001; ** = P<0.01; * = P< 0.05; NS = not significant

Back-transformed least squares means of WEC increased with age (Table 5.5). Overall, kids at 5 months of age had more than two fold greater WEC than kids at 3 months. After artificial challenge with *T. colubriformis* L_3 WEC at 6.25 and 6.5 months were much higher than the values for natural infection in both populations. This confirms the efficacy of the artificial challenge in each breed and in each year. In contrast to the first two years, the majority of kids in 2002/2003 and 2003/2004 (year 3 and 4) had zero WEC at natural infections at 3 and 5 month of age, demonstrating that dependence of natural challenge on favourable climatic conditions.

Table 5.5 Least squares means of WEC of Angora and Cashmere kids by year of birth. Values are back transformed from the square root scale.

Angora					Cashmere				
Year	3 mo	5 mo	6.25 mo	6.5 mo	Year	3 mo	5 mo	6.25 mo	6.5 mo
2000/2001	55	873	2459	2802	2000/2001	349	1222	1669	1750
2001/2002	226	1800	2515	3074	2001/2002	473	962	1853	2037
2002/2003	0	23	5529	5386	2002/2003	0	0	4132	3907
					2003/2004	13	18	2640	2649

The paddock in which kids were reared from birth to weaning in the different management groups significantly influenced $WEC^{0.5}$ at all studied ages in Cashmere kids but the effect across ages was inconsistent. For example the lowest WEC at 3, 5, 6.25 and 6.5 months respectively was found in kids grazed in Nscrubby, Nscrubby, Peach tree and Back Scotchmans Paddocks. The highest $WEC^{0.5}$ at 3, 5, 6.25 and 6.5 months respectively was observed for kids grazed in East fall, Bottom Box, Highlands, and Nscrubby Paddocks. These data suggest that low natural exposure in Nscrubby in the first 5 months of life resulted in low levels of immunity and consequently kids reared in this paddock exhibited higher susceptibility to infection (and thus higher WEC) when artificially challenged at 5.25 months of age.

Faecal egg counts: Genetic parameter estimates

In order to find the best fitted model for estimating genetic parameters for WEC, three different models were examined as described in Section 2.5.2. Model 2 (model with animal additive effect) gave the best fit for $WEC^{0.5}$ at almost all studied ages in both populations and so was used throughout (Table 5.6).

Table 5.6 $WEC^{0.5}$ at 3 and 5 months of age and 28 and 35 days and following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 and 6.5 months) in Angora and Cashmere goats: Log likelihood values for models with different random effects. Decreases in log likelihood indicate an improvement in the model fit.

Angora				Cashmere			
$WEC^{0.5}$ at	1	2	3	$WEC^{0.5}$ at	1	2	3
3 months	**-1537.67**	-1537.54	-1537.54	3 months	-2160.02	**-2159.25**	-2158.48
5 months	-1772.90	**-1769.45**	1769.41	5 months	-2330.04	**-2323.37**	-2322.54
6.25 months	**-1802.53**	-1802.00	-1802.00	6.25 months	-2297.48	**-2295.46**	-2295.46
6.5 months	-1834.78	**-1832.36**	-1832.36	6.5 months	-2195.42	**-2185.45**	-2185.45

Model 1: Significant fixed effects (SFE) only
Model 2: SFE + additive effect of the animal (a)
Model 3: SFE + a + permanent environment effect of the animal's dam (m_{pe})

The variance components were estimated using REML with the animal model (model 2). Additive (σ^2_a) and phenotypic (σ^2_p) variance components, and h^2 estimates of $WEC^{0.5}$ in Angora and Cashmere goat populations are summarized in Table 5.7. Heritability estimates for $WEC^{0.5}$ were weak to moderate, ranging from 0.02 and 0.22. The highest estimates were observed at 5 and 6.5 months of age in both populations. The h^2 estimates were higher for Cashmere goats than for Angora goats. In Cashmere goats when the permanent environment effect of the animal's dam was fitted, the heritability of $WEC^{0.5}$ at 3 and 5 months of age dropped to 0.04 and 0.17, respectively.

Fitting the permanent environmental effects of the animal (pe) allowed an estimation of the within-artificial infection repeatability of WEC (at 28 and 35 days post artificial challenge). Estimates of repeatability between two infections with *T. colubriformis* were high (0.40) in Angora goats and moderate (0.27) in Cashmere goats (Table 5.8). For Angora goats, the animal permanent environment had a large effect (0.36) compared with that for Cashmere goats (0.12).

Table 5.7 Estimates of variance components of square root transformed faecal egg counts ($WEC^{0.5}$) from kids at 3 and 5 months of age and 28 and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 and 6.5 months of age) in Angora and Cashmere goat breeds.

Breed and component	Natural infection at		Artificial challenge at	
	3 mo	5 mo	6.25 mo	6.5 mo
Angora				
σ^2_a	0.13	2.47	0.85	2.70
σ^2_e	6.25	13.24	23.30	20.14
σ^2_p	6.38	15.71	24.15	22.84
h^2	0.02	0.16	0.04	0.12
s.e.(h^2)	0.05	0.09	0.05	0.08
Cashmere				
σ^2_a	0.64	3.49	1.46	2.96
σ^2_e	10.56	14.15	15.11	10.51
σ^2_p	11.20	17.64	16.57	13.47
h^2	0.06	0.20	0.09	0.22
s.e.(h^2)	0.06	0.09	0.06	0.08

$\sigma^2_p = \sigma^2_a + \sigma^2_e$, $h^2 = \sigma^2_a / \sigma^2_p$; σ^2_p – phenotypic variance; σ^2_a – additive variance; σ^2_e – residual variance; h^2 –estimate of heritability; s.e. (h^2) – standard error of heritability

Table 5.8 Genetic parameters for $WEC^{0.5}$ estimated based on two repeated measurements at 6.25 and 6.5 months of age (28 and 35 days after artificial challenge with *T. colubriformis*) in Angora and Cashmere goats

Breed	σ^2_a	σ^2_{pe}	σ^2_e	σ^2_p	h^2(s.e.)	pe^2 (s.e.)	r_e (s.e.)
Angora_Aus	8.35	0.98	13.94	23.27	0.04 (0.04)	0.36(0.05)	0.40 (0.04)
Cashmere_Aus	2.20	1.87	11.20	15.27	0.14 (0.05)	0.12(0.05)	0.27 (0.04)

h^2=heritability; pe^2=permanent environmental effect; r_e = repeatability
Model 4: SFE + a+ permanent environmental effect of the animal (pe)

In both goat populations, moderate and positive relationships were found between WEC measured within each (natural or artificial) infection cycle (Table 5.9). However, correlations between infection cycles in either breed were close to zero.

Table 5.9 Phenotypic correlations$_{s.e.}$ between square root transformed faecal egg counts ($WEC^{0.5}$) in Angora (above diagonal) and Cashmere (below diagonal) goats

Age	3 months	5 months	6.25 months	6.5 months
3 months		$0.31_{0.04}$	$0.09_{0.04}$	$-0.01_{0.04}$
5 months	$0.33_{0.03}$		$0.05_{0.04}$	$0.13_{0.04}$
6.25 months	$-0.03_{0.04}$	$0.02_{0.04}$		$0.41_{0.04}$
6.5 months	$0.09_{0.04}$	$0.08_{0.04}$	$0.29_{0.03}$	

5.3.2 Circulating specific anti-nematode IgG concentrations

Data summary

Values for specific IgG were highly variable with a skewed distribution. After applying square root transformation the skewness and kurtosis was significantly reduced in both populations. As with WEC, circulating concentrations of specific IgG rose with increase in age and exposure to worms (Table 5.10, Figure 5.10).

Table 5.10 Basic statistics for square-root-transformed specific antibody concentration (IgG, $units^{0.5}$) at 3 and 5 months of age and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 months of age) in Angora and Cashmere goats

Breed and age	No. of kids	Mean	SD	Range	Skewness	Kurtosis
Angora						
3 mo	557	14.87	4.35	0-32	0.44	0.56
5 mo	569	17.52	6.36	0-42	0.66	1.33
6.25 mo	564	34.01	12.62	0-34	0.64	0.57
Cashmere						
3 mo	755	26.01	13.82	5-82	1.35	1.64
5 mo	765	27.82	13.86	0-85	0.82	1.20
6.25 mo	754	54.03	19.08	0-129	1.00	1.87

Compared with IgG antibody levels after natural challenge (at 3 and 5 months of age), two fold higher concentrations of IgG were observed after artificial challenge with *T. colubriformis*, showing a response to challenge infection in primed animals. Even though kids in both populations were challenged with the same dose of *T. colubriformis* L_3 larva, the production of IgG in Cashmere goats was higher than that in Angora goats (Table 5.10, Figure 5.2). This is consistent with the greater ability of Cashmere goats to limit infections following artificial challenge.

Specific Antibody: Fixed effects

The significance of main effects and their interactions for $IgG^{0.5}$ at 3, 5, and 6.25 months of age in Angora goats and Cashmere goats are given in Table 5.11. With the exception of an effect of birth type on $IgG^{0.5}$ at 3 months of age in Cashmere goats (higher levels in single born kids), birth type and sex did not influence concentration of $IgG^{0.5}$ in either population. Year of birth had a significant effect on $IgG^{0.5}$ concentration at 3 and 5 months of age, but not on $IgG^{0.5}$ at 6.5 months in Angora goats.

Table 5.11 Specific antibody levels (IgG $units^{0.5}$) associated with natural infection at 3 (N3m) and 5 (N5m) months of age and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (A28d; 6.25 months of age) in Angora and Cashmere goats: Significance of main fixed effects and covariates.

Angora				Cashmere			
Fixed effect	IgG_N3m	IgG_N5m	IgG_A2d8	Fixed effect	IgG_N3m	IgG_N5m	IgG_A28d
Birth type	NS	NS	NS	Birth type	*	NS	NS
Sex	NS	NS	NS	Year	***	***	*
Year	***	***	NS	Paddock	**	***	**
Age within sampling period	NS	NS	NS	Paddock:Year	NS	***	***

*** = P<0.001; ** = P<0.01; * = P< 0.05; NS = not significant

Table 5.12 and Figure 5.2 show the effects of year at each age of sampling for both sample populations. For both breeds at 5 months kids born in 2001 had higher concentration of specific antibody ($IgG^{0.5}$) than those born in 2000, 2002, and 2003. Significantly elevated concentrations of $IgG^{0.5}$ were observed for 3-month old Cashmere kids born in 2003. The reason for this is unknown. For Cashmere kids, paddock had a great effect on $IgG^{0.5}$ at all sampling times (Table 5.10). Much of this effect at the 3 month and 5 month samplings could be explained by differences in WEC reflecting differences in worm challenge within paddocks (Figure 5.3).

Table 5.12 Least squares means of $IgG^{0.5}$of Angora and Cashmere kids by year of birth. Values are back transformed from the square root scale.

Angora					Cashmere		
Year	3 mo	5 mo	6.25 mo	Year	3 mo	5 mo	6.25 mo
2000/2001	268	306	1253	2000/2001	529	861	2725
2001/2002	230	384	1050	2001/2002	431	1338	2921
2002/2003	154	221	1193	2002/2003	170	483	3108
				2003/2004	2157	242	2491

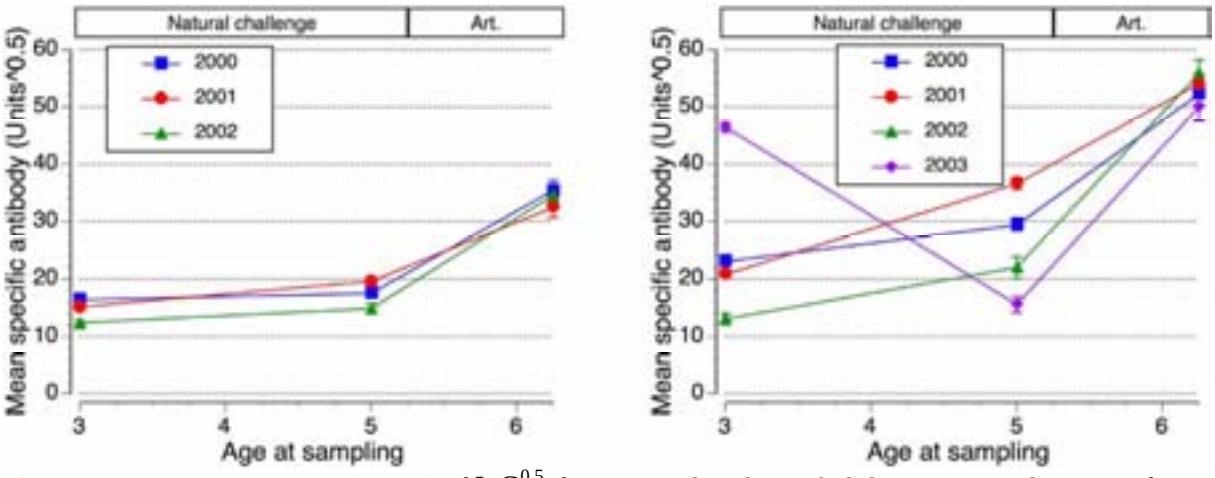

Figure 5.2 Least squares means (±SEM) of $IgG^{0.5}$of Angora and Cashmere kids by age at sampling year of birth. by age at sampling in goat kids on both resource herds. At 3 and 5 months kids were subject to natural challenge while samples at 6.25 months of age reflect antibody levels 4 weeks after artificial challenge with 10,000 L3 of *T. colubriformis* at 5.25 months of age.

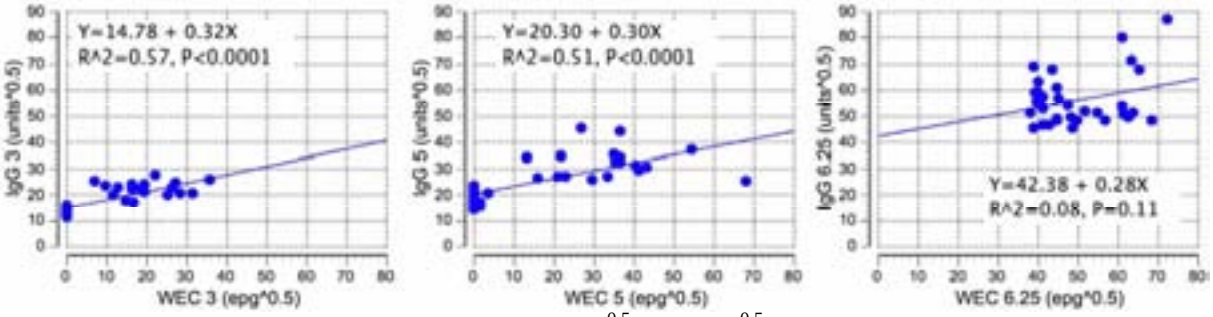

Figure 5.3 Association between paddock means for $IgG^{0.5}$ and $WEC^{0.5}$ for Cashmere kids in each year of sampling for the 3, 5 and 6.25 sampling times. Each point represents one paddock in one year. Curves are linear regressions.

Specific Antibody: Parameter estimates

The log likelihood values of three different models are given in Table 5.13. After fitting the animal additive effect, the log likelihood decreased for $IgG^{0.5}$ at all ages except at 3 months of age in Cashmere goats, where the animal effect or heritability was non-significant. Maternal non-additive genetic (m_{pe}) effects were negligible or non significant, except at 3 months in Angora goats and 5 months in Cashmere goats. For $IgG^{0.5}$ at 3 months in Angora goats and $IgG^{0.5}$ at 5 months in Cashmere goats, however, the permanent environment effect of the dam had a large effect on log likelihood values as well as on heritability estimates.

Table 5.13 Specific antibody ($IgG^{0.5}$) at 3 and 5 months of age and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 months of age) in Angora and Cashmere goats: Log likelihood values for models with different random effects

Angora				Cashmere			
$IgG^{0.5}$ at	1	2	3	$IgG^{0.5}$ at	1	2	3
3 months	- 1058.11	- 1056.35	**1054.37**	3 months	**- 1947.21**	- 1947.04	- 1947.04
5 months	1309.70	**- 1305.88**	- 1305.88	5 months	- 2159.06	- 2156.24	**- 2151.45**
6.25 months	- 1712.13	**- 1693.65**	- 1693.65	6.25 months	- 2502.03	**- 2500.21**	- 2499.97

Model 1: Significant fixed effects (SFE) only
Model 2: SFE + additive effect of the animal (a)
Model 3: SFE + a + permanent environment effect of the animal's dam (m_{pe})

Specific $IgG^{0.5}$ at the different sampling ages was weakly to moderately heritable (Table 5.14).

Table 5.14 Estimates of variance components of square root transformed specific antibody concentration ($IgG^{0.5}$) from kids at 3 and 5 months of age and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 months of age) in Angora and Cashmere goats

Breed and age	σ^2_a	σ^2_e	σ^2_p	h^2	s.e.(h^2)
Angora					
3 months	0.25	1.40	1.65	0.15	0.11
5 months	0.51	3.18	3.69	0.14	0.08
6.25 months	6.76	9.38	16.14	0.42	0.13
Cashmere					
3 months	0.14	6.43	6.57	0.02	0.04
5 months	1.93	10.10	12.03	0.16	0.09
6.25 months	3.09	30.23	33.32	0.09	0.07

$\sigma^2_p = \sigma^2_a + \sigma^2_e$, $h^2 = \sigma^2_a / \sigma^2_p$; σ^2_p – phenotypic variance; σ^2_a – additive variance; σ^2_e – residual variance; h^2 – estimate of heritability; s.e. (h^2) – standard error of heritability

The h^2 estimates for specific IgG in Angora goats were low (0.14-0.15) during natural infection at 3 and 5 months of age, but it was significantly elevated (0.42) at 4 weeks later (at 6.25 moths of age) after artificial infection with *T. colubriformis*. For $IgG^{0.5}$ at 3 months of age in Angora goats, including the permanent environmental effect of the dam significantly affected the heritability value, reducing it from 0.15 to 0.05. On the other hand, the estimates of heritability for Cashmere goats were generally low (0.02-0.16). The highest estimate of h^2 (0.16) for Cashmeres was observed at 5 months of age after natural infection. However, this heritability estimate reduced from 0.16 to 0.06 after fitting the permanent environment effect of the dam into model. Generally, the direct heritability values estimated using model 3 (fitting the permanent environmental effect of the dam) rose with increase of age in both populations.

5.3.3 Leukocyte (white blood cell) traits

Leukocyte traits: Means and fixed effects

Blood concentrations of the five main white cell families (eosinophil (EOS), lymphocyte (LYM), neutrophil (NEU), basophil (BASO), and monocyte (MONO) counts) were analysed for both populations (Table 5.15). Higher numbers of circulating eosinophils were observed in Cashmere than Angora kids at all ages. For Angora kids EOS did not increase significantly during natural infections with *H. contortus*, but a significant eosinophil response was observed after artificial challenge with *T. colubriformis* (Table 5.15, Figure 5.4). In Cashmere kids EOS were high throughout, with little evidence of a response to infection. Angora goats tended to have higher concentrations of lymphocytes, basophils and monocytes at all ages. Basophil and monocyte concentrations increased sharply after artificial infection in both breeds while neutrophil concentrations in both populations declined over time. Lymphocyte concentrations increased over time in Angora goats, but decreased in Cashmere goats.

Birth type had a significant effect on NEU and MONO, with 3 and 6.25 month-old twin Angora kids having lower concentrations of NEU and MONO (Table 5.13). Paddock and paddock by year of birth interactions were significant for some traits in Cashmere goats. Year of birth significantly influenced all white blood cell variables for Angora kids and some white cell variables for Cashmere kids (Table 5.15, Figure 5.4).

Leukocyte traits: Estimates of heritabilities and correlations

The best fitting model for the majority of white blood cell traits was the animal model (Model 2). Estimates of heritability (h^2) and genetic and phenotypic correlations between white blood cell variables in Angora and Cashmere populations are summarised in Table 5.16.

Table 5.15 Leukocyte traits at 3 and 5 months of age and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 months of age) in Angora and Cashmere goats: Number of records, means and significance of main fixed effects and covariables

	Angora						Cashmere				
Age and effects	$EOS^{0.33}$	LYM	NEU	BASO	MONO	Age and effects	$EOS^{0.33}$	LYM	NEU	BASO	MONO
3 months											
No. of records	589	589	584	592	592	No. of records	783	785	781	785	784
Mean ± s.e.	0.33±0.04	5.40±0.13	5.49±0.09	0.64±0.01	1.90±0.04	Mean ± s.e.	0.51±0.01	6.21±0.12	5.67±0.09	0.58±0.01	1.73±0.03
Fixed effect						Fixed effect					
Birth type	NS	NS	*	NS	*	Birth type	NS	NS	NS	NS	NS
Year	***	***	***	***	***	Year	**	NS	***	***	*
Sex	*	NS	*	NS	NS	Paddock	***	**	***	***	NS
Age at records	NS	NS	NS	NS	NS	Paddock:Year	***	**	***	NS	*
5 months											
No. of records	586	587	587	589	586	No. of records	787	786	782	787	784
Mean ± s.e.	0.34±0.01	7.09±0.17	5.33±0.08	0.62±0.01	1.60±0.02	Mean±s.e.	0.57±0.01	5.81±0.11	5.43±0.07	0.56±0.01	1.47±0.02
Fixed effect						Fixed effect					
Birth type	NS	NS	NS	NS	*	Birth type	NS	NS	NS	NS	NS
Year	***	***	***	***	***	Year	NS	NS	NS	***	***
Sex	NS	NS	NS	NS	NS	Paddock	**	NS	**	NS	NS
Age at records	NS	NS	NS	NS	NS	Paddock:Year	***	***	***	NS	NS
6.25 months											
No. of records	575	575	575	576	575	No. of records	777	781	772	779	778
Mean ± s.e.	0.43±0.01	6.68±0.16	5.11±0.08	0.66±0.01	1.96±0.03	Mean ± s.e.	0.55±0.01	5.71±0.10	4.14±0.05	0.65±0.01	1.85±0.02
Fixed effect						Fixed effect					
Birth type	NS	NS	**	NS	NS	Birth type	NS	NS	NS	NS	NS
Year	NS	***	*	***	***	Year	***	NS	***	NS	NS
Sex	NS	*	**	NS	NS	Paddock	***	NS	***	NS	NS
Age at records	NS	NS	NS	NS	***	Paddock:Year	NS	NS	NS	NS	NS

$EOS^{0.33}$ (eosinophils, $x10^9$/L); BASO (basophils, $x10^9$/L); NEU (neutrophils, $x10^9$/L); LYM (lymphocytes, $x10^9$/L); MONO (monocytes, $x10^9$/L) *** = P<0.001; ** = P<0.01; * = P< 0.05; NS = not significant

Table 5.16 Estimates of genetic parameters and their standard errors for leukocyte traits at 3 and 5 months of age and 35 days following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 months of age) in Angora and Cashmere goats. Heritabilities$_{s.e.}$ (denoted by bold number) are on the diagonal, phenotypic correlations$_{s.e.}$ are above the diagonal and genetic correlations$_{s.e.}$ are below the diagonal.

	Angora					Cashmere				
Age and traits	$EOS^{0.33}$	LYM	NEU	BASO	MONO	$EOS^{0.33}$	LYM	NEU	BASO	MONO
3 months										
$EOS^{0.33}$	$\mathbf{0.06_{0.07}}$	$0.17_{0.04}$	$-0.01_{0.04}$	$-0.03_{0.04}$	$0.00_{0.04}$	$\mathbf{0.43_{0.09}}$	$0.12_{0.04}$	$0.00_{0.04}$	$0.13_{0.04}$	$0.06_{0.04}$
LYM	$0.27_{0.54}$	$\mathbf{0.31_{0.12}}$	$0.02_{0.05}$	$-0.16_{0.04}$	$-0.04_{0.05}$	$0.05_{0.20}$	$\mathbf{0.31_{0.09}}$	$-0.17_{0.04}$	$-0.22_{0.04}$	$-0.19_{0.04}$
NEU	$0.49_{0.47}$	$0.48_{0.22}$	$\mathbf{0.51_{0.12}}$	$0.19_{0.04}$	$0.20_{0.04}$	$-0.01_{0.30}$	$0.25_{0.30}$	$\mathbf{0.10_{0.07}}$	$0.27_{0.03}$	$0.29_{0.03}$
BASO	$0.04_{0.85}$	$-0.46_{0.42}$	$0.37_{0.39}$	$\mathbf{0.05_{0.07}}$	$0.67_{0.02}$	$0.72_{0.22}$	$0.37_{0.31}$	$0.29_{0.42}$	$\mathbf{0.14_{0.07}}$	$0.70_{0.02}$
MONO	$0.16_{0.47}$	$0.25_{0.28}$	$0.36_{0.22}$	$0.95_{0.48}$	$\mathbf{0.34_{0.12}}$	$0.41_{0.30}$	$0.56_{0.36}$	$0.19_{0.51}$	$0.63_{0.25}$	$\mathbf{0.09_{0.06}}$
5 months										
$EOS^{0.33}$	$\mathbf{0.26_{0.11}}$	$0.26_{0.04}$	$0.01_{0.05}$	$-0.07_{0.04}$	$0.03_{0.05}$	$\mathbf{0.38_{0.09}}$	$0.08_{0.04}$	$0.01_{0.04}$	$0.15_{0.04}$	$0.14_{0.04}$
LYM	$0.67_{0.21}$	$\mathbf{0.55_{0.13}}$	$0.04_{0.05}$	$-0.01_{0.05}$	$0.01_{0.05}$	$0.39_{0.19}$	$\mathbf{0.40_{0.10}}$	$-0.12_{0.04}$	$-0.13_{0.04}$	$-0.13_{0.04}$
NEU	$0.66_{0.23}$	$0.90_{0.14}$	$\mathbf{0.71_{0.12}}$	$0.16_{0.04}$	$0.17_{0.05}$	$0.06_{0.36}$	$0.05_{0.36}$	$\mathbf{0.06_{0.05}}$	$0.12_{0.04}$	$0.13_{0.04}$
BASO	$-0.14_{0.44}$	$0.13_{0.38}$	$0.37_{0.28}$	$\mathbf{0.09_{0.08}}$	$0.62_{0.03}$	$0.24_{0.24}$	$-0.04_{0.24}$	$-0.40_{0.43}$	$\mathbf{0.19_{0.07}}$	$0.64_{0.02}$
MONO	$0.21_{0.31}$	$0.06_{0.24}$	$0.22_{0.20}$	$0.91_{0.09}$	$\mathbf{0.37_{0.14}}$	$0.23_{0.24}$	$-0.26_{0.21}$	$-0.20_{0.39}$	$0.60_{0.18}$	$\mathbf{0.20_{0.07}}$
6.25 months										
$EOS^{0.33}$	$\mathbf{0.28_{0.13}}$	$0.15_{0.05}$	$0.03_{0.05}$	$0.03_{0.04}$	$0.17_{0.05}$	$\mathbf{0.34_{0.09}}$	$0.08_{0.04}$	$0.17_{0.04}$	$0.15_{0.04}$	$0.16_{0.04}$
LYM	$0.34_{0.26}$	$\mathbf{0.42_{0.13}}$	$0.01_{0.05}$	$-0.05_{0.05}$	$0.07_{0.05}$	$0.35_{0.18}$	$\mathbf{0.41_{0.09}}$	$-0.09_{0.04}$	$-0.23_{0.04}$	$-0.09_{0.04}$
NEU	$0.57_{0.27}$	$0.63_{0.22}$	$\mathbf{0.60_{0.12}}$	$0.14_{0.04}$	$0.13_{0.05}$	$0.27_{0.23}$	$0.01_{0.24}$	$\mathbf{0.19_{0.08}}$	$0.21_{0.04}$	$0.21_{0.04}$
BASO	$-0.52_{0.39}$	$0.04_{0.36}$	$-0.04_{0.31}$	$\mathbf{0.12_{0.09}}$	$0.55_{0.03}$	$0.35_{0.22}$	$-0.07_{0.21}$	$-0.12_{0.30}$	$\mathbf{0.21_{0.07}}$	$0.62_{0.02}$
MONO	$0.50_{0.27}$	$0.62_{0.19}$	$-0.12_{0.21}$	$0.35_{0.35}$	$\mathbf{0.32_{0.13}}$	$0.47_{0.18}$	$0.05_{0.19}$	$0.21_{0.25}$	$0.61_{0.16}$	$\mathbf{0.36_{0.09}}$

Angora and Cashmere goat populations behaved quite differently. In Angora goats, NEU, LYM and MONO traits were most heritable (0.31-0.71) among the white blood cell variables and the h^2 estimates were also higher than those in Cashmere goats. In Cashmere goats, $EOS^{0.33}$ and LYM traits were the most heritable, with moderate estimates (0.30-0.48). With increase of age, the h^2 estimates of $EOS^{0.33}$ increased in Angora goats, but decreased in Cashmere goats. Estimates of h^2 for BASO ranged from 0.05-0.21, indicating that this trait is not highly heritable.

EOS had positive genetic correlations with LYM and NEU, but their phenotypic correlations were low, almost not significant. Positive high genetic correlation existed between LYM and NEU for Angoras, but for Cashmeres no genetic correlations between them were observed. The phenotypic correlation coefficients showed no relationship between LYM and NEU for Angoras and negative weak relationship for Cashmeres. The highest positive genetic and phenotypic correlations were found between BASO and MONO in both populations. Generally, genetic and phenotypic correlations between white cell variables were quite different and inconsistent through all ages in both populations. Furthermore, it should be noted that standard errors of genetic correlations were very high due to the limited size of the data set.

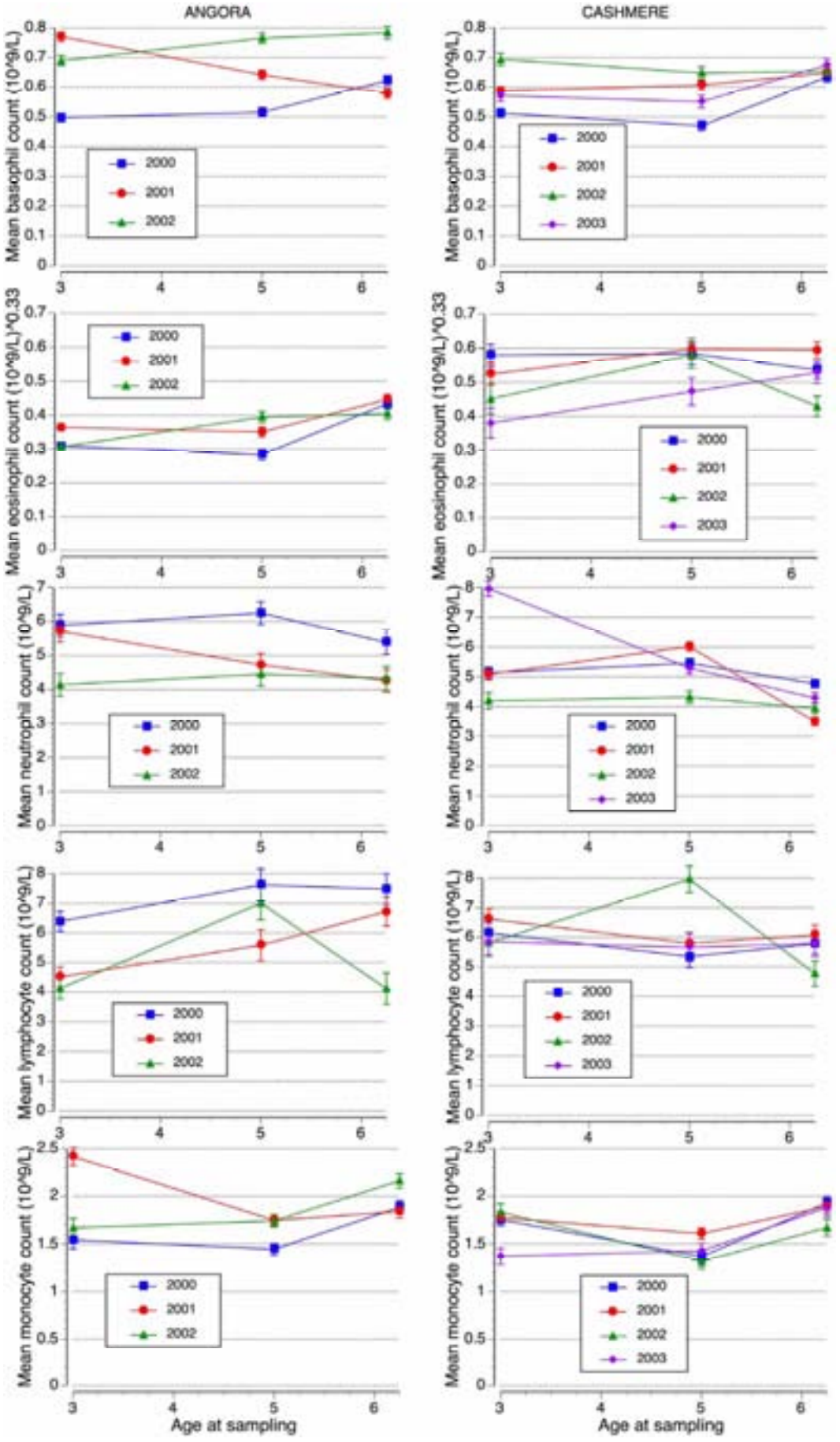
ANGORA
CASHMERE
Mean basophil count (10^9/L)
Mean eosinophil count (10^9/L)^0.33
Mean neutrophil count (10^9/L)
Mean lymphocyte count (10^9/L)
Mean monocyte count (10^9/L)
2000
2001
2002
2003
Age at sampling

Figure 5.4 Least squares mean (±SEM) count of circulating BAS, EOS, NEUT LYM and MONO of Angora (left panel) and Cashmere (Right panel) kids by age at sampling year of birth. At 3 and 5 months kids were subject to natural challenge while samples at 6.25 months of age reflect antibody levels 4 weeks after artificial challenge with 10,000 L3 of *T. colubriformi*s at 5.25 months of age.

5.3.4 Erythrocyte (red blood cell) traits

Erythrocyte traits: Means and fixed effects

Pathological consequences of infection with blood sucking parasites such as *Haemonchus contortus* can be assessed by change in level of red blood cell variables (packed cell volume (PCV), mean corpuscular volume (MCV), and mean corpuscular hemoglobin concentration (MCHC)). The number of records, means and significance of main fixed effects of red blood cell variables after natural and artificial challenge infection are shown in Table 5.17. Compared with Angora kids, at all sampling times Cashmere kids demonstrated consistently higher PCV and MCV and lower MCHC. For Angora kids PCV and MCV were significantly reduced after natural challenge at 5 months of age compared to 3 months of age, and there was little change in those variables following artificial challenge with *T. colubriformis*, which is not a blood sucking parasite.

For Angora goats, sex had a significant effect on all three variables, with female kids having higher level of PCV and MCV, but lower MCHC at all studied ages (Table 5.17). For both populations, birth type had a significant influence on PCV and MCV at 3 months of age, with twin-born kids demonstrating lower PCV and MCV. However, the birth type effect generally disappeared with an increase in age. Year of birth had significant effects in both breeds and for all variables but not consistently. Year of birth dramatically influenced all these variables in 5- and 6.25-month old Angora kids and PCV and MCV in 3- and 5-month old Cashmere kids. Compared with 2000 and 2002, Angora kids born in 2001 had lower PCV, MCV, and MCHC at all ages excluding PCV at 3 months of age. Similar to Angora kids, 3- and 5 month-old Cashmere kids born in 2001 had a lower level of PCV and MCV. This reflects the higher prevalence of *H. contortus* during natural infections in year 2001/2002.An effect of paddock and significant interaction between paddock and year of birth was observed only for MCV for 5-month old Cashmere kids.

Table 5.17 Erythrocyte traits at 3 and 5 months of age and 4 weeks following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 months of age) in Angora and Cashmere goats: Number of records, means and significance of main fixed effects and covariables.

Angora				Cashmere			
Age and effects	PCV	MCV	MCHC	Age and effects	PCV	MCV	MCHC
3 months							
No. of records	592	591	592	No. of records	785	783	784
Mean ± s.e.	24.4±0.1	19.0±0.0	46.8±0.2	Mean ± s.e.	26.2±0.1	19.2±0.0	42.7±0.2
Fixed effect				Fixed effect			
Birth type	***	***	NS	Birth type	***	***	*
Year	NS	NS	**	Year	*	***	NS
Sex	***	***	**	Paddock	*	**	NS
Age at records	***	***	*	Paddock:Year	NS	NS	NS
5 months							
No. of records	589	587	587	No. of records	785	785	786
Mean ± s.e.	21.2±0.1	17.8±0.0	48.2±0.3	Mean±s.e.	26.6±0.1	19.1±0.0	44.4±0.2
Fixed effect				Fixed effect			
Birth type	NS	NS	NS	Birth type	*	***	NS
Year	***	***	***	Year	*	*	NS
Sex	***	***	***	Paddock	NS	***	NS
Age at records	*	NS	NS	Paddock:Year	NS	**	NS
6.25 months							
No. of records	576	575	576	No. of records	778	777	779
Mean ± s.e.	21.2±0.1	17.8±0.0	46.2±0.3	Mean ± s.e.	26.2±0.1	19.0±0.0	46.1±0.2
Fixed effect				Fixed effect			
Birth type	NS	NS	NS	Birth type	NS	NS	NS
Year	*	***	**	Year	NS	**	*
Sex	***	***	***	Paddock	NS	NS	NS
Age at records	NS	NS	NS	Paddock:Year	NS	NS	NS

PCV (packed cell volume, %); MCV (mean corpuscular volume, fL); MCHC (mean corpuscular haemoglobin concentrate, g/dL)

*** = P<0.001; ** = P<0.01; * = P< 0.05; NS = not significant

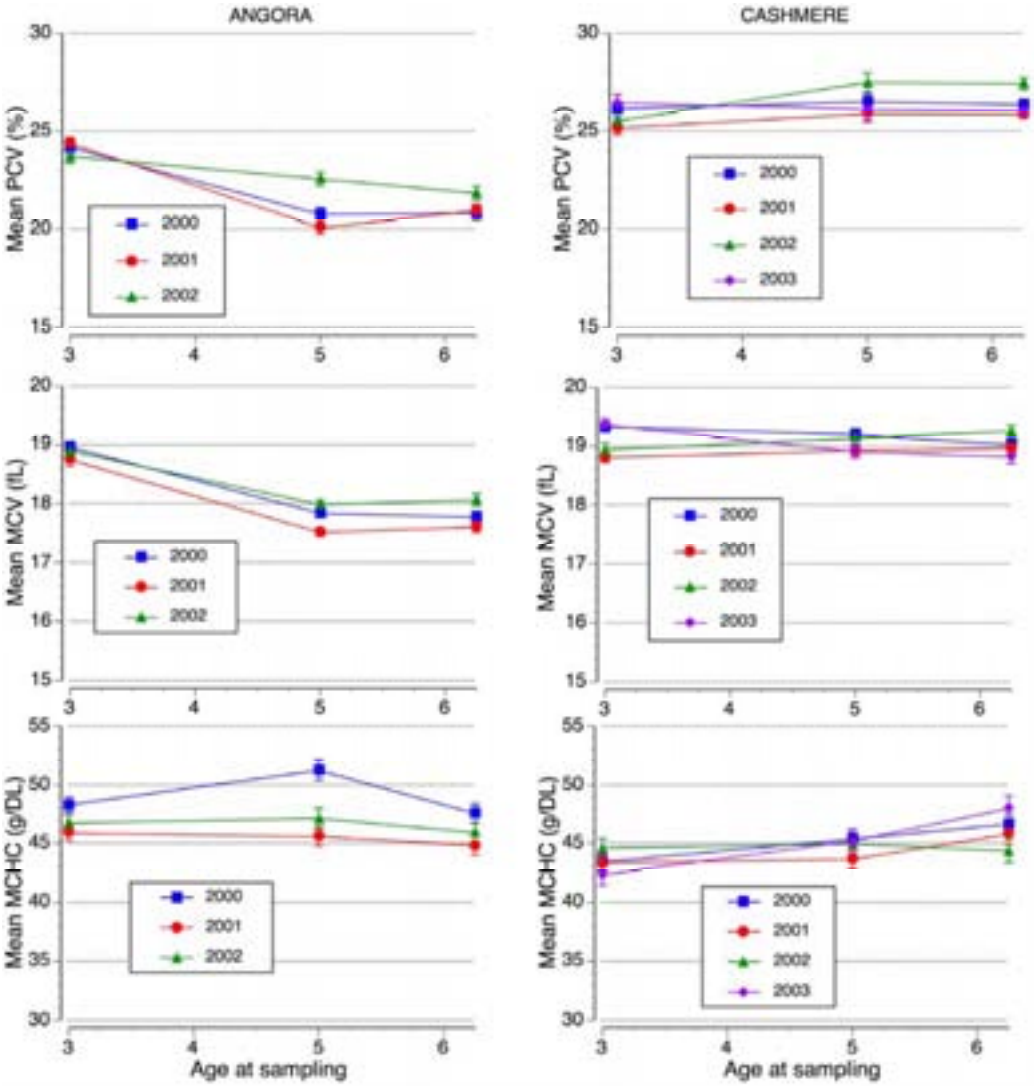

Figure 5.5 Least squares means (±SEM) of circulating PCV, MCV and MCHC of Angora (left panel) and Cashmere (Right panel) kids by age at sampling year of birth. At 3 and 5 months kids were subject to natural challenge while samples at 6.25 months of age reflect antibody levels 4 weeks after artificial challenge with 10,000 L3 of *T. colubriformis* at 5.25 months of age.

Erythrocyte traits: Estimates of heritabilities and correlations

The animal model was the best fit for red blood cell variables. Estimates of heritability (h^2) of and genetic and phenotypic correlations between red blood cell traits in Angora and Cashmere populations are reported in Table 5.18. All red blood cell traits were moderately to highly heritable, ranging from 0.40 to 0.97. The heritability estimates for Cashmere goats were higher than those for Angora goats. The estimates of heritability of red blood cell traits for Cashmere goats at 3 months of age were generally lower compared with that at other two ages (5 and 6.25 months). For both populations, the h^2 estimates significantly increased at the age of 5 months old and then it dropped slightly at 6.25 months of age.

At all ages, positive and strong consistent genetic (0.88-0.99) and phenotypic (0.77-0.92) correlations between PCV and MCV were observed in both goat populations (Table 5.18). MCHC had high negative (genetic and phenotypic) correlations with the other two variables. This means kids with high PCV (measure of red blood cell numbers, expressed as a percentage of the total volume of blood) and high MCV (average volume of a single red blood cell) tend to have low MCHC (average concentration of haemoglobin in red blood cells) and MCHC will reduce rapidly with selection for high PCV and MCH.

Table 5.18 Estimates of genetic parameters for erythrocyte traits at 3 and 5 months of age and 4 weeks following artificial challenge with *T. colubriformis* at 5.25 months of age (6.25 months of age) in Angora and Cashmere goats. Heritabilities$_{s.e.}$ (denoted by bold number) are on the diagonal, phenotypic correlations$_{s.e.}$ are above the diagonal and genetic correlations$_{s.e.}$ are below the diagonal.

	Angora			Cashmere		
Age and traits	PCV	MCV	MCHC	PCV	MCV	MCHC
3 months						
PCV	**0.49$_{0.15}$**	0.89$_{0.01}$	-0.72$_{0.02}$	**0.73$_{0.10}$**	0.89$_{0.01}$	-0.78$_{0.02}$
MCV	0.92$_{0.04}$	**0.48$_{0.13}$**	-0.55$_{0.03}$	0.99$_{0.01}$	**0.49$_{0.11}$**	-0.68$_{0.02}$
MCHC	-0.79$_{0.09}$	-0.64$_{0.13}$	**0.51$_{0.14}$**	-0.92$_{0.03}$	-0.99$_{0.00}$	**0.75$_{0.09}$**
5 months						
PCV	**0.62$_{0.14}$**	0.92$_{0.01}$	-0.77$_{0.02}$	**0.97$_{0.07}$**	0.90$_{0.01}$	-0.80$_{0.01}$
MCV	0.95$_{0.02}$	**0.52$_{0.15}$**	-0.60$_{0.03}$	0.98$_{0.01}$	**0.86$_{0.09}$**	-0.64$_{0.03}$
MCHC	-0.85$_{0.06}$	-0.70$_{0.12}$	**0.51$_{0.13}$**	-0.90$_{0.02}$	-0.82$_{0.05}$	**0.98$_{0.07}$**
6.25 months						
PCV	**0.59$_{0.14}$**	0.77$_{0.02}$	-0.64$_{0.03}$	**0.93$_{0.08}$**	0.86$_{0.01}$	-0.80$_{0.02}$
MCV	0.88$_{0.06}$	**0.46$_{0.13}$**	-0.64$_{0.03}$	0.95$_{0.02}$	**0.79$_{0.08}$**	-0.51$_{0.03}$
MCHC	-0.80$_{0.09}$	-0.88$_{0.08}$	**0.45$_{0.12}$**	-0.92$_{0.03}$	-0.72$_{0.07}$	**0.87$_{0.08}$**

Phenotypic correlations between WEC and other parasite-associated traits

Figures 5.6 and 5.7 show the phenotypic relationships between faecal worm egg counts (WEC$^{0.5}$) at 3, 5, 6.25, and 6.5 and other parasite-associated traits at 3, 5 and 6.25 months of ages in Angora and Cashmere goats. In Angora goats at 3 and 5 months of age, WEC$^{0.5}$ was negatively and weakly associated with PCV, LYM, and EOS$^{0.33}$, but was positively associated with NEU and IgG$^{0.5}$ (Figure 5.1). A similar trend occurred with Cashmere kids, but the strength of the associations was weaker. PCV of five month-old Cashmere kids showed weak and positive correlations with WEC. There were no significant correlations (close to zero) between WEC$^{0.5}$ and EOS$^{0.33}$ at five months of ages in both populations.

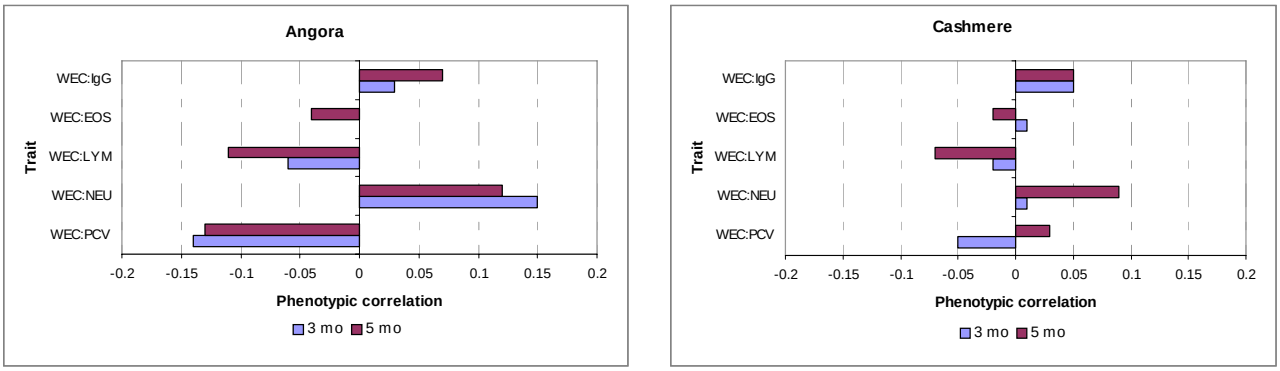

Figure 5.6 Phenotypic correlations between faecal worm egg counts (WEC) and blood traits at 3 and 5 months of age in Angora and Cashmere goats

For both goat populations, weak but negative relationships existed between WEC$^{0.5}$ at 6.25 and 6.5 months of age and IgG$^{0.5}$, EOS$^{0.33}$, and PCV at 6.25 months of age (Figure 5.7). No significant relationships were observed between WEC$^{0.5}$ and LYM at these ages. For Angora kids, WEC$^{0.5}$ was positively but not significantly correlated with NEU. A non-significant negative association existed between WEC$^{0.5}$ and NEU in Cashmere goats.

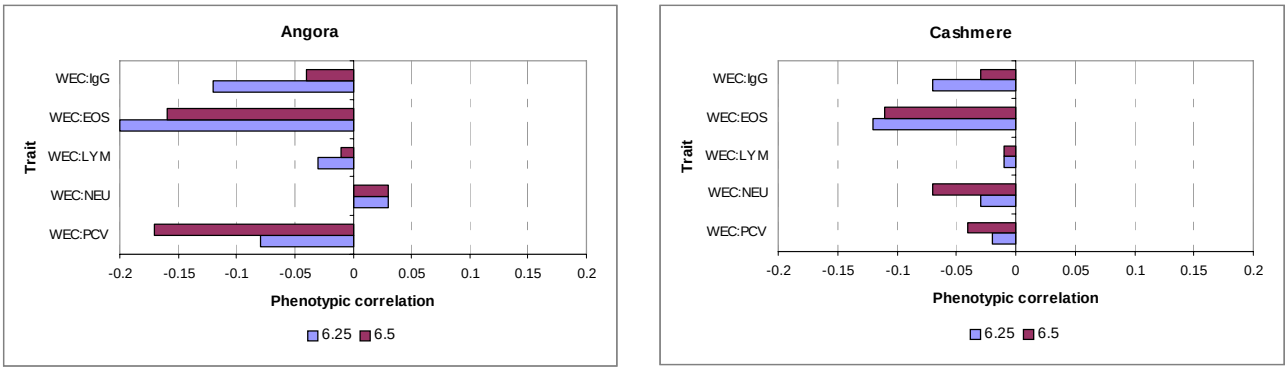

Figure 5.7 Phenotypic correlations between blood cell traits at 6.25 and WEC at 6.25 and 6.5 months of ages in Angora and Cashmere goats

5.4 Discussion

Overview of challenge and immunity. The low levels of natural challenge of kids from the 2002 and 2003 drops indicates the risks associated with reliance on natural challenge to obtain estimates of resistance. In retrospect it would have been better to have done bulk WEC counts (WEC on a mixture of fresh faeces from 20-30 kids collected off the ground) to establish levels of infection prior to individual animal WECs. Nevertheless there was good natural challenge in the first 2 years of the project and the artificial challenge was effective in each year of the experiment. The variation in natural challenge provided good insights into the development of immunity in the goat kids. In the first two years when there was good natural challenge and a chance for the kids to develop immunity, WEC following the standard challenge dose of 10,000 L3 of *T. colubriformis* was considerably lower than that seen in later years when kids were challenged without significant early challenge. While differences in the viability of the challenge L3 can't be completely ruled out as a cause of this observation, differences in immunity due to differences in exposure prior to challenge is the most likely cause. This is supported by the strong negative association between paddock mean WEC at 3 and 5 months, and mean WEC at 6.25 and 6.5 months (Figure 5.8, below).

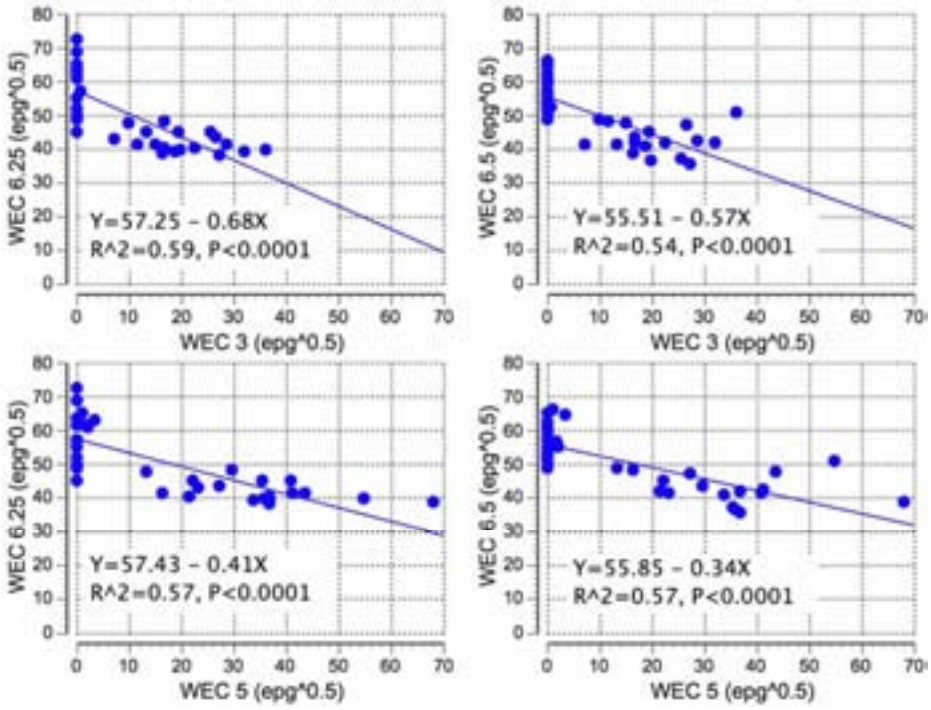

Figure 5.8 Association between paddock means for $WEC^{0.5}$ under natural challenge (3 and 5 months) and artificial challenge (6.25 and 6.5 months) for Cashmere kids in each year of sampling. Each point represents one paddock in one year. Curves are linear regressions.

Indeed at an individual animal level there was a highly significant negative correlation between WEC at 3 and 5 months of age, and WEC following artificial challenge (6.25 and 6.5 months) in both Angora (r= -0.22 to -0.39) and Cashmere goats (r= -0.27 to -0.37). It is also supported by the strong positive association between paddock mean WEC and specific IgG at 3 and 5 months (Figure 5.3). This suggests that goat kids are able to develop significant resistance to infection before the age of 6 months provided that they have sufficient challenge beforehand. This suggests that parasite control methods that rely on maintaining some parasite burden in the host are more likely to be successful than those that suppress worm infections totally. Tactical worm control based upon close monitoring of WEC and treatment intervention only when the WEC exceeds a given threshold based on worm species involved, animal condition, feed availability and climatic conditions is likely to maintain immunity in the population and help regulate infections. Such an approach has been successfully used in sheep (Kahn *et al.*, 2006; Scrivener *et al.*, 2006). The negative association between early WEC and WEC following artificial challenge is not evident in the phenotypic correlations for these WEC estimates (Table 5.9). The reason for this is that the phenotypic correlations examine associations between traits after adjustment for other effects in the model. For example it would test the relationship between WEC3 and WEC6.25 within paddocks and years. However, since much of the "effect" of paddocks and years is mediated by differences in WEC, adjusting for these sources of information is inadvertently obscuring the overall relationship between early and later WEC counts.

Heritability estimates

In both populations, heritability estimates for WEC were low to moderate (range: 0.02-0.22), which falls within the rage of estimates reported in literature for goats (See Section 1.3, Table 1.2). These tend to be somewhat lower than estimates in sheep and cast doubt on the practicality of breeding for resistance to GIN based on WEC despite the presence of genetic variation in resistance. However heritability estimates above 0.3 have been derived from studies in Greole goats (Mandonnet *et al.*, 2001), and Scottish Cashmere goats (Jackson, 2000; Vagenas *et al.*, 2002). Our study differed from others in some unexpected areas. Firstly the heritability for WEC in neither Angora nor Cashmere goats increased with age and secondly the heritability of the mean of two measures (6.25 and 6.5 months) was not higher than the higher of the two estimates based on a single measurement. The reason for these differences is not clear. Unlike WEC, the heritability of several parasite-associated traits was moderate to high. The heritability of specific antibody response was generally low but reached 0.42 for Angora goat 4 weeks after artificial challenge. Eosinophil counts at 5 or 6.25 months had heritability estimates ranging from 0.26-0.38 in both breeds and several other leucocyte traits were also moderately to highly heritable. Red cell variables tended to be highly heritable, for example PCV at 5 and 6.25 months had heritability estimates ranging from 0.59-0.97. The heritability estimates for PCV in this study are considerably higher than other published estimates (0.10-0.33 as shown in Table 1.2).

Reports on studies of genetic correlations between WEC and leucocyte and antibody variables are scarce. In this study, due to a small data set and a high proportion of zero counts for WEC, the estimation of genetic correlations between WEC and IgG or EOS was non-estimable. In 6-month old Romney lambs, a low negative genetic correlation (-0.21) was found between WEC and *T. colubriformis* antibodies (Douch *et al.*, 1995). Also, an increased level of circulating eosinophils correlated with a declining WEC in Romney lambs challenged twice a week with 5,000 *T. colubriformis* L_3 larvae (Buddle *et al.*, 1992). These results indicate that selection for high IgG and/or EOS would result in a slow decrease in WEC.

The high heritability estimates for PCV suggest that it could be used as an alternative marker in breeding program for resilience to *Haemonchus contortus*. It is less likely to be useful as a marker for resistance as very low genetic correlations were found between PCV and WEC in other goat studies (Baker *et al.*, 2001; Mandonnet *et al.*, 2001). Phenotypic correlations between these variables in the

present study were weakly negative. Given the highly heritable nature of PCV (one extreme animal had PCV values of 39.5, 40.0 and 40.2 at 3, 5 and 6.25 months respectively) rapid progress could be made by selecting for this variable. However the consequences for the circulatory system of marked increases in the cellular component of blood would need to be considered.

Means and phenotypic correlations

In both populations, much higher WEC and levels of circulating specific IgG were observed after artificial challenge with *T. colubriformis* than after natural challenge infection. This was particularly true in years 3 and 4, where, due to dry weather conditions, the level of natural challenge was extremely low and the majority of goats had zero egg counts. Artificial challenge is therefore a more effective way to induce infection than natural challenge although it is unlikely to be practical as an industry practice. However in the present study this did not result in significantly improved heritability estimates for WEC or eosinophil counts. It did result in improved heritability estimates for specific antibody in Angora, but not in Cashmere goats. Given the costs and risks associated with artificial challenge it is likely to remain only a research tool with estimation of parasite-associated traits in the field limited to natural infection. The use of bulk WEC monitoring tests prior to individual animal WEC measurements to ensure that a threshold WEC has been achieved (say 500 epg) should be mandatory prior to the expense of determining individual animal WEC.

Generation and expression of resistance to larval challenge depends on the mode and duration of challenge infections and the presence or absence of an adult worm population (Balic *et al.*, 2000). Key effector mechanisms include an increase in eosinophil counts (cell mediated immunity) and specific antibody directed against the nematode parasite (antibody-mediated or humoral immunity). In young goats (3 and 5 months of age) the association between WEC and specific IgG was positive suggesting that kids exposed to higher worm challenge developed humoral immunity at a greater rate than those exposed to lower levels of challenge. However following fixed challenge at 5 months of age, the relationship was stronger and negative, indicating that goats with higher levels of circulating antibody were more resistant to infection. Both of these findings are consistent with our understanding of the immune response to nematode infection (Balic *et al.*, 2000). Similarly, eosinophil counts were poorly related to WEC in young goats under natural challenge because counts could reflect either a positive relationship between exposure to worms and development of an eosinophil response, or a negative relationship between eosinophil counts and WEC in infected goats. The situation was clearer following artificial challenge with a fixed dose of larvae, with a clear negative association between WEC and eosinophil counts evident in both breeds.

The other white cell counts and the red cell variables are more likely to be associated with the pathology induced by the parasites, than a direct immune response to them. The positive association between WEC and neutrophil counts in young goats at 3 and 5 months of age may reflect damage to the gastrointestinal epithelium, particularly by *Haemonchus contortus*, and the subsequent inflammatory response to the damage. This association was not as evident following artificial infection with the less invasive *Trichostrongylus colubriformis*. This highlights that *H. contortus* predominated in faeces of kids up to 5 months of age, particularly the Cashmere kids. On the other hand, following artificial challenge with *T. colubriformis* the challenge species dominated, despite ongoing natural challenge from the pasture, including with *H. contortus*. The negative relationship between WEC and PCV was an expected phenotypic relationship for a region where *H. contortus* is a significant parasite, because it is a haematophagous (blood sucking) parasite. That the relationship was stronger in Angora goats suggests that they have less ability to cope with parasitic infections than Cashmere goats, something supported by other findings in the experiment (see below).

It is disappointing that genetic correlations between WEC and the other parasite indicator traits were not estimable as information on this in the literature is scant. As can be seen in section 1.5.4 and from Table 1.8 genetic correlations between WEC and PCV are invariably negative in other goat studies those between PCV and liveweight are invariably positive.

The study could not formally test breed differences between Angora and Cashmere goats because the two breeds were on separate farms with subtly different management and environmental conditions. Nevertheless, there were some apparent differences between the two breeds that warrant further investigation in properly designed studies. Several lines of evidence point to greater susceptibility to gastrointestinal nematode infection of Angora goats. Despite generally lower WEC at 3 months of age, Angoras had higher WEC in two of 3 years by 5 months of age. This however may reflect different levels of challenge in the two environments. What is most interesting is the response of the two breeds to challenge with the same dose of infective larvae of *T. colubriformis*. By 28 days after infection Angora goats had mean WECs 38% higher than those of Cashmere goats and by day 35 post-infection the difference had increased to 49% (Table 5.5). As well as having lower WEC than Angoras, Cashmere goats had considerably higher concentrations of both specific antibody and eosinophils in blood. By day 35 post-infection plasma specific antibody concentrations (back transformed means from Table 5.10) in Cashmere goats were 2.5 fold higher than those of Angora goats. However it should be noted that this was a consistent difference between the breeds at all ages sampled, and the proportional increase in antibody levels following artificial challenge was similar between the breeds (3.8 fold increase). The situation was similar with EOS counts being higher in Cashmere than Angora goats at all ages, particularly at 5 months of age (67% higher) just prior to artificial challenge. Resistant sheep show higher numbers of circulating eosinophils after both vaccination and challenge with 20,000 *T. colubriformis* L_3 larva than susceptible sheep (Dawkins *et al.*, 1989), showing that level of eosinophils is associated with resistance status in this species. The combination of lower WEC following a fixed-dose parasite challenge and higher specific antibody and EOS in Cashmere compared to Angora goats is strongly suggestive of a better ability to mount an effective immune response to infection in the former. This is further supported by the reduction in PCV in Angora but not Cashmere goats between 3 and 5 months of age in response to natural infection with *H. contortus*. The reasons for this apparent breed difference are speculative but are likely to involve the greater partitioning of amino acids towards fibre production in the Angora relative to Cashmere goats. Resistance to gastrointestinal nematode infection in intimately associated with protein metabolism of the host and availability of amino acids (MacRae, 1993).

5.4.1 Summary

- Natural challenge was an unreliable source of infection in years 3 and 4 of the study while artificial challenge was successful in every year.
- Goats exposed to infection early in life (up to 5 months) mounted more effective immune responses to infection which led to reduced WEC following artificial challenge at 5.22 months of age.
- In both Angora and Cashmere goats, estimates of heritability of WEC and IgG were low. The highest heritabilities for WEC and IgG were at 5 and 6.5 months of age. The estimates of heritability and genetic and phenotypic correlations between these traits varied with age at sampling and breed. These results suggest that selection for low WEC would result in slow genetic progress in resistance to GIN.
- Year of birth had a great impact on WEC, IgG, and haematological variables at 3, 5 and 6.5 months of age. Sex and birth type had no effect on WEC, IgG and the majority of leukocyte traits, however, significant effects were observed for erythrocyte traits at 3 months of age in both populations. In Cashmere goats the effect of Paddock was important particularly for WEC, IgG and leukocyte traits.
- Angora goats appeared to be more susceptible to GIN than Cashmere goats as assessed by a number of variables. However as breed and location were fully confounded in the present study an effect of location and management cannot be categorically ruled out.
- Heritability estimates for blood cell measurements, both white and red cells, had moderate to high heritability and rapid progress could be made if selecting for these traits. However genetic correlations with other traits were non-estimable so correlated responses in other traits (eg. WEC) would be difficult to predict.

6. Quantitative genetic analysis of production traits and association with parasite-associated traits

6.1 Introduction

The main objective of this project was to investigate parasite-associated traits in fibre goats as there have been many investigations into the genetics of production traits in Angora and Cashmere goats as detailed in Section 1.4 and Tables 1.3 and 1.4. However it is important to assess the association between parasite traits and production traits, so a range of production variables was also measured in the current project. If parasite resistance is chosen one of the objectives of a breeding program, the selection should also take into account any correlated effects on genetic improvement of the production traits. The genetic and phenotypic relationships between parasite and production traits are less frequently reported in goats than in sheep. In Scottish crossbred cashmere-producing goats, genetic correlations between WECs and production traits were found to be slightly positive, but not significantly different from zero (Vagenas *et al.*, 2002). Our data allows estimation of genetic parameters for production traits, which would be used for developing breeding programs that aim to improve fibre traits as well as parasite resistance traits to improve parasite resistance.

There is also merit in having another set of estimates of genetic parameters for fibre goat production variables. Although genetic parameters are extensively studied in farm animals, ideally estimates should be based on data from the particular population of interest because genetic variation for production traits (such as fleece and live weights) could be different for different subpopulations. Prior knowledge of genetic and phenotypic parameters is required for designing and implementing effective breeding programs in a particular herd, because the rate of genetic gain, development of selection indices and optimisation of selection are heavily dependent on the genetic and phenotypic parameters.

The aims of this part of the project were therefore as follows:

- to test the significance of fixed effects for production traits
- to estimate the heritability of, and the genetic and phenotypic correlations between, production traits in Australian Angora and Cashmere goats
- to estimate phenotypic correlations between production traits and parasite-associated traits (WEC, IgG, leukocyte and erythrocyte counts) in both goat populations

6.2 Results

6.2.1 Analysis of live weights

Live weight: Data summary

Summary statistics for live weights are shown in Table 6.1. The mean live weights increased with age in both populations. Based on raw phenotypic means, Cashmere goats were heavier than Angora goats at all ages except at 3 months of age. However, between breed comparisons are confounded with location since the animals of these two breeds were raised in two different environments.

Table 6.1 Number of observations, means and standard deviations (SD) of various weights for Angora and Cashmere goat breeds

Live weight (kg) and age	Angora			Cashmere		
	No. of kids	Mean	SD	No. of kids	Mean	SD
3 mo	600	13.13	2.88	781	13.94	2.96
5 mo	589	16.19	3.23	775	20.10	3.34
6 mo	579	16.80	3.29	776	21.62	3.29
10 mo				295	24.51	2.92
12 mo	549	19.19	4.26			
18 mo	499	26.10	6.58			
22 mo				277	31.04	4.60

Live weight: Main fixed effects

Table 6.2 and 6.3 show the significance of main fixed effects and the least squares means for live weight at different ages for Angora and Cashmere goats. In both goat populations, birth type had a significant effect on live weights at early ages (from 3 to 10 months of age). Kids born and reared as twins were lighter than kids born single (Table 6.2 and 6.3). Year significantly influenced live weights in both populations (Figure 6.1). Age (days) and sex in the Angora goat herd and paddock in the Cashmere goat herd had strong effects on live weights. Female Angora goats grew more slowly than male goats. A significant interaction was observed between year and paddock in Cashmere goats.

Table 6.2 Live weight of Angora goats: Significance of main fixed effects and covariables and their least squares means

Effect	Wt3	Wt5	Wt6	Wt12	Wt18
Birth type	P<0.001	P<0.001	P<0.001	P=0.134	P=0.113
Single	13.5±0.1	16.5±0.2	17.1±0.2	19.6±0.3	25.7±0.5
Twin	11.6±0.2	14.8±0.3	15.8±0.3	19.1±0.4	26.5±0.6
Year	P<0.001	P=0.012	P=0.007	P<0.001	P<0.001
2000/2001	13.0±0.2	16.5±0.3	17.3±0.3	17.2±0.4	25.2±0.6
2001/2002	13.8±0.2	15.4±0.3	16.0±0.3	18.2±0.4	20.4±0.6
2002/2003	10.8±0.3	15.1±0.3	16.0±0.4	22.6±0.4	32.6±0.7
Sex	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001
Female	12.0±0.2	15.1±0.2	15.8±0.3	18.5±0.3	25.0±0.5
Male	13.0±0.2	16.3±0.2	17.01±0.3	20.2±0.3	27.0±0.5
True age	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001

Table 6.3 Live weight of Cashmere goats: Significance of main fixed effects and their least squares means

Effect	Wt3	Wt5	Wt6	Wt10	Wt22
Birth type	P<0.001	P<0.001	P<0.001	P<0.001	P=0.374
Single	14.0±0.1	20.4±0.2	21.6±0.2	25.0±0.3	31.4±0.4
Twin	12.9±0.2	19.1±0.2	20.5±0.2	23.6±0.4	30.9±0.6
Year	P<0.001	P<0.001	P<0.001	P=0.008	P<0.001
2000/2001	11.9±0.2	18.4±0.2	19.9±0.2	24.8±0.3	33.2±0.5
2001/2002	14.2±0.2	19.3±0.2	21.6±0.2	23.8±0.3	NA
2002/2003	11.1±0.3	18.3±0.3	18.9±0.4	25.7±0.6	NA
2003/2004	16.6±0.3	23.1±0.3	23.9±0.3	24.9±0.5	29.1±0.7
Paddock (Pdk)	P<0.001	P<0.001	P<0.001	P=0.092	P=0.768
Year:Pdk	P<0.001	P<0.001	P=0.019	P=0.378	P=0.396

NA = not available

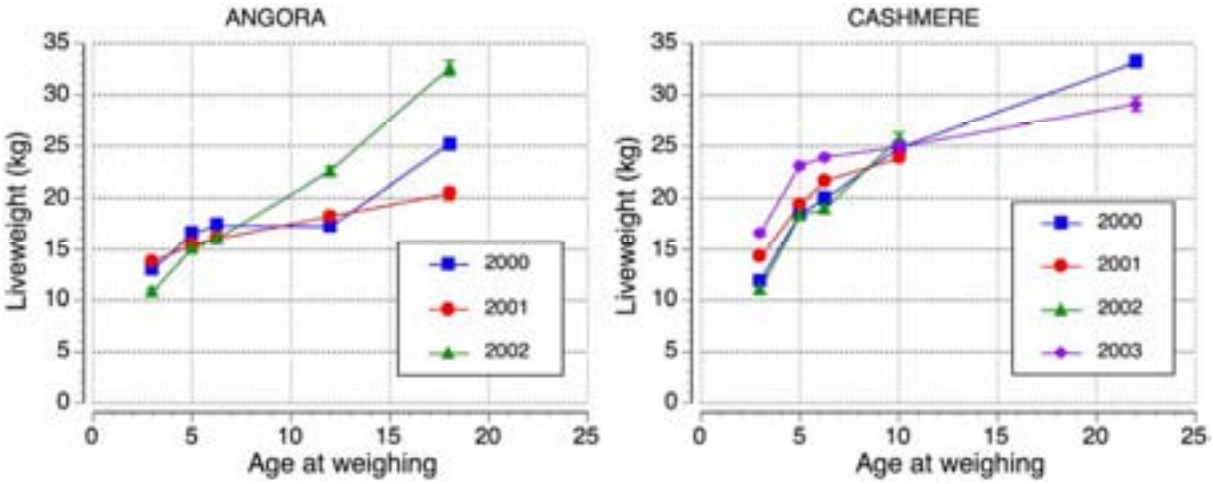

Figure 6.1 Least squares means (±SEM) for live weight of Angora and Cashmere goats by year and age.

Live weights: Random effects and heritability estimates

The log likelihood values used to decide the appropriate model for various live weights are given in Table 6.4 and the heritability estimates are in Table 6.5.

Table 6.4 Live weights (LWT): Log likelihood values for models 1-3 with different random effects fitted. The maximum log likelihood for each variable is bolded.

Angora LWT	Model 1	Model 2	Model 3	Cashmere LWT	Model 1	Model 2	Model 3
3 months	-846.945	-841.899	**-833.697**	3 months	-1017.74	-1013.38	**-1010.05**
5 months	-939.622	-932.128	**-928.260**	5 months	-1145.49	**-1144.65**	-1144.23
6 months	-928.969	-918.563	**-915.002**	6 months	-1179.05	**-1176.81**	-1176.19
12 months	-922.683	**-905.131**	-904.921	12 months	-452.350	**-451.498**	-451.498
18 months	-1023.99	**-1009.61**	-1009.61	18 months	-527.514	**-526.135**	-526.135

Model 1: Significant fixed effects (SFE) only
Model 2: SFE + additive effect of the animal (a)
Model 3: SFE + a + permanent environment effect of the animal's dam (m_{pe})

Model 3 with direct additive and permanent environment effect was the model of best fit (P >0.05) for live weight at 3, 5 and 6 months of age in Angora goats and for live weights at 3 months in Cashmere goats. For live weights at 12 and 18 months in Angora goats and live weights at 5, 6, 12 and 22 months in Cashmere goats, model 2 was the best fit and fitting the permanent maternal environment effect (model 3) did not significantly improve the fit of model.

Table 6.5 Estimates of genetic parameters and their standard errors for live weight at various ages

	Model 3		Model 2
Breed and age	h^2_3±s.e.	c^2±s.e.	h^2_2±s.e.
Angora			
3 months			0.57±0.15
5 months			0.59±0.15
6 months			0.52±0.14
12 months			0.55±0.15
18 months			0.58±0.16
Cashmere			
3 months	0.14±0.08	0.19±0.07	0.20±0.09
5 months	0.06±0.06	0.07±0.07	0.07±0.06
6 months	0.09±0.07	0.07±0.07	0.11±0.07
10 months	0.15±0.14	0.00±0.00	0.15±0.14
22 months	0.17±0.14	0.00±0.16	0.17±0.14

h^2_3 = direct h^2 _model 3; c^2= maternal environmental effect as proportion of total variance;
h^2_2= direct h^2 _model 2; s.e.= standard error

Using model 2, live weights in Angora goats were highly heritable (range: 0.52-0.59) compared with estimates in Cashmere goats (range: 0.07-0.20). However, the standard errors of heritability estimates in both populations were high due to the small size of the data set. The permanent maternal environment effect in the Cashmere population decreased with increase of age (Table 6.5). For live weight at 10 and 22 in Cashmere goats, the permanent maternal environment effect was negligible and the heritability estimates were not affected by fitting the permanent maternal environment effect. Even though model 3 with direct additive and permanent environment effect was the best fit of model in Angora goats at 3, 5 and 6 months of age, results in the estimates of variance components using model 3 appeared to be aberrant (majority of Angora dams had only one offspring) and therefore, they are not shown here.

6.2.2 Analysis of fleece traits

Fleece traits for Angora goats: Data summary and main fixed effects

The data summary and the significance of main fixed effects for fleece traits in Angora goats are reported in Table 6.6 and the least squares means of fleece traits for different years at three shearing time are shown in Figure 6.2. An effect of birth type on fleece traits was observed for the first shearing, but not for the second and third shearings. Sex affected greasy fleece weights (GFW) and mean fibre curvature (CURVE) at all three shearing times with heavier GFW for males. Year of birth had a strong influence on almost all fleece traits (Table 6.6). From Figure 6.1, the goats born in 2002 demonstrated the highest GFW, broadest and straightest fibres, and the highest percentage of KEMP and MED, particularly at the second and third shearings. From this figure, ignoring any other possible confounding effects it also can be seen the difference in fleece traits between years as well as seasons. For example, spring-GFW (second shearing) recorded in 2002 was significantly lower than that recorded in 2003, whereas autumn-GFW (third shearing) recorded in 2002 was significantly higher than that in 2003. However, it is hard to interpret whether this difference was due to environmental (e.g. climate) or genetic effects.

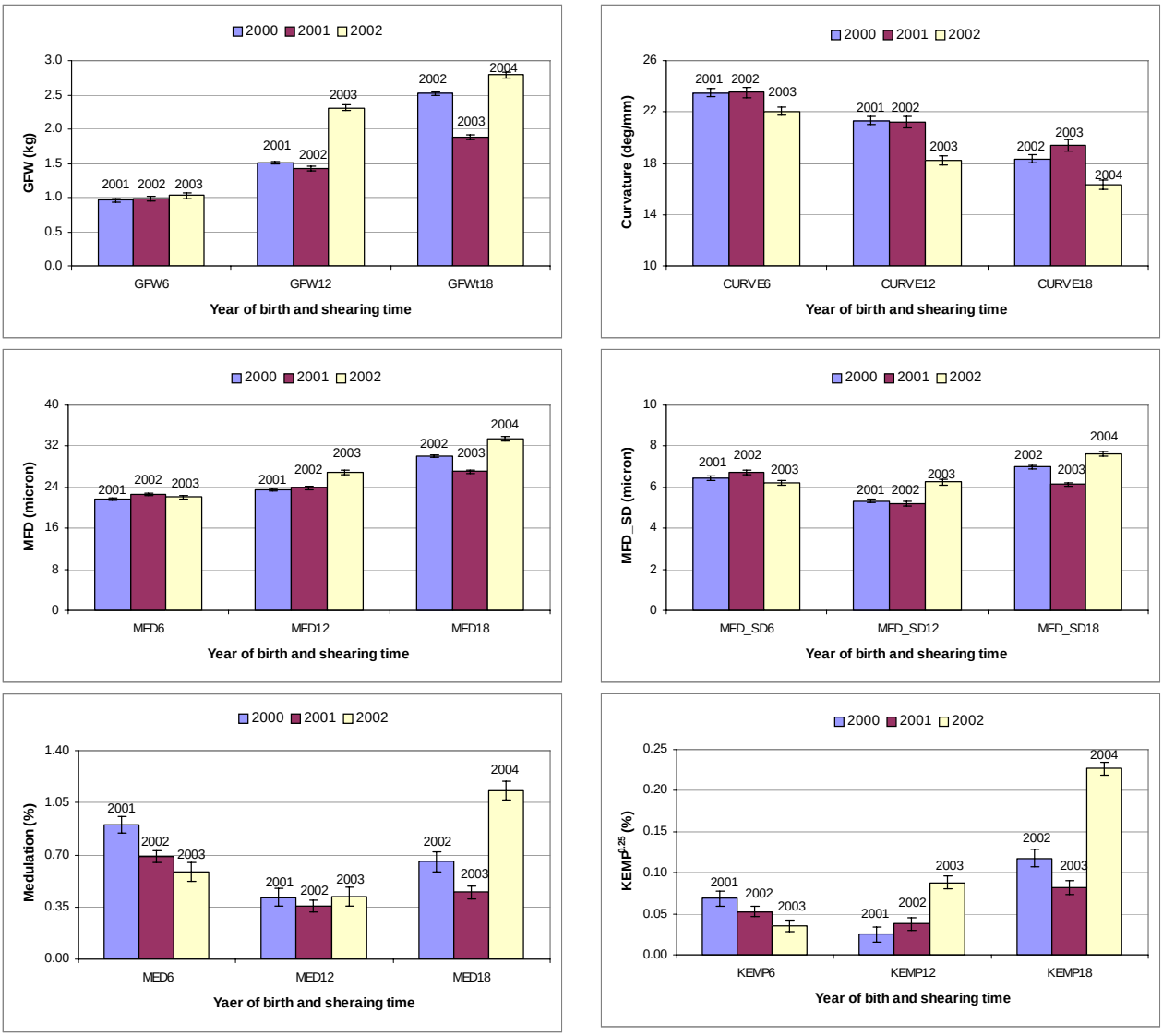

Figure 6.2 Least squares means (±SEM) of fleece traits their standard errors at three shearing times (6, 12 and 18 months) in Angora goats born in 2000, 2001 and 2002. Numbers above bar indicate the year when each shearing was performed.

Table 6.6 Data summary and significance of main fixed effects for fleece traits at 6, 12 and 18 months of age in Angora goats

Trait	Unit	No. of records	Mean±SD	Fixed effect			
				Birth type	Year	Sex	Age
6 months							
GFW	kg	577	1.02±0.22	P<0.001	P=0.032	P<0.001	P<0.001
MFD	µm	585	22.4±1.7	P<0.001	P<0.001	P=0.236	P=0.017
MFD_SD	µm	585	6.6±0.7	P<0.001	P<0.001	P=0.020	P<0.001
CURVE	deg/mm	584	23.1±2.6	P=0.794	P<0.001	P<0.001	P=0.159
MED	%	586	0.76±0.46	P=0.030	P<0.001	P=0.013	P=0.462
KEMP	$\sqrt[4]{\%}$	586*	0.06±0.09	P=0.061	P=0.037	P=0.018	P=0.292
12 months							
GFW	kg	546	1.73±0.49	P=0.096	P<0.001	P<0.001	P<0.001
MFD	µm	548	24.7±2.5	P=0.187	P<0.001	P=0.026	P=0.093
MFD_SD	µm	548	5.6± 0.9	P=0.178	P<0.001	P=0.607	P=0.347
CURVE	deg/mm	548	20.5±3.9	P=0.350	P<0.001	P=0.003	P=0.004
MED	%	545	0.39± 0.26	P=0.999	P=0.264	P=0.028	P=0.004
KEMP	$\sqrt[4]{\%}$	548	0.05± 0.07	P=0.394	P<0.001	P=0.823	P=0.125
18 months							
GFW	kg	508	2.43±0.55	P=0.810	P<0.001	P<0.001	P<0.001
MFD	µm	502	30.3± 3.5	P=0.550	P<0.001	P=0.132	P=0.192
MFD_SD	µm	502	6.9± 1.2	P=0.029	P<0.001	P=0.947	P=0.163
CURVE	deg/mm	503	18.1± 2.8	P=0.037	P<0.001	P=0.020	P=0.340
MED	%	502	0.75± 0.46	P=0.337	P<0.001	P=0.118	P=0.684
KEMP	$\sqrt[4]{\%}$	504	0.14±0.10	P=0.125	P<0.001	P=0.985	P=0.702

GFW = greasy fleece weight; MFD = mean fibre diameter; MFD_SD = standard deviation of mean fibre diameter; CURVE = mean fibre curvature; MED = medullation; * - 393 (67%), 391 (71%), 146 (30%) of total records were “zero” values for KEMP at 6, 12, and 18 months of age, respectively.

Table 6.7 Least squares means (±SEM) for fleece traits in Angora goats; Effects of sex and birthtype.

Variable	Effect	Level	Shearing age (1st 3 shearings)		
			6 months	12 months	18 months
GFW (kg)	Sex	F	0.96±0.01[a]	1.71±0.03[a]	2.32±0.03[a]
		M	1.02±0.01[b]	1.79±0.03[b]	2.48±0.03[b]
	Birth type	Single	1.05±0.01[a]	1.77±0.03	2.40±0.03
		Twin	0.92±0.02[b]	1.73±0.03	2.39±0.04
MFD (µm)	Sex	F	22.18±0.17	24.97±0.27[a]	30.34±0.32
		M	22.03±0.16	24.59±0.27[b]	30.01±0.32
	Birth type	Single	22.57±0.15[a]	24.90±0.26	30.10±0.30
		Twin	21.64±0.18[b]	24.66±0.29	30.25±0.35
MFD-SD (µm)	Sex	F	6.52±0.08[a]	5.57±0.08	6.92±0.10
		M	6.40±0.07[b]	5.60±0.08	6.91±0.09
	Birth type	Single	6.61±0.07[a]	5.64±0.07	6.79±0.08a
		Twin	6.31±0.08[b]	5.53±0.09	7.03±0.11b
CURVE (deg/mm)	Sex	F	22.50±0.24[a]	17.75±0.27[a]	19.83±0.36[a]
		M	23.55±0.24[b]	18.26±0.27[b]	20.70±0.35[b]
	Birth type	Single	22.99±0.22	18.27±0.25	20.42±0.32[a]
		Twin	23.05±0.27	17.74±0.30	20.11±0.40[b]
MED (%)	Sex	F	0.77±0.05[a]	0.42±0.03[a]	0.77±0.05
		M	0.68±0.05[b]	0.37±0.03[b]	0.72±0.05
	Birth type	Single	0.77±0.04[a]	0.40±0.03	0.73±0.05
		Twin	0.68±0.05[b]	0.40±0.04	0.76±0.06
KEMP ($\sqrt[4]{\%}$)	Sex	F	0.061±0.008[a]	0.050±0.005	0.142±0.006
		M	0.044±0.007[b]	0.051±0.005	0.142±0.005
	Birth type	Single	0.060±0.006	0.047±0.004	0.136±0.004
		Twin	0.044±0.009	0.053±0.007	0.148±0.007

[ab]Means with different superscripts within shearing time and effect level indicate significant differences. Refer to Table 6.6 for exact P values.

Fleece traits for Cashmere goats: Data summary and main fixed effects

The data summary and the significance of main fixed effects tested for fleece traits in Cashmere goats are given in Table 6.8 and the least squares means of fleece traits at two shearing times for different years is shown in Figure 6.3. There was no difference in fleece traits between singles and twins. Paddock and interaction between paddock and year of birth affected greasy fleece weights as well as cashmere weights. Year difference at recording had a strong influence on fleece traits at both shearing times. Like Angora goats, Cashmere goats born in 2002 demonstrated higher fleece and down weights and down yield compared goats born in the other years.

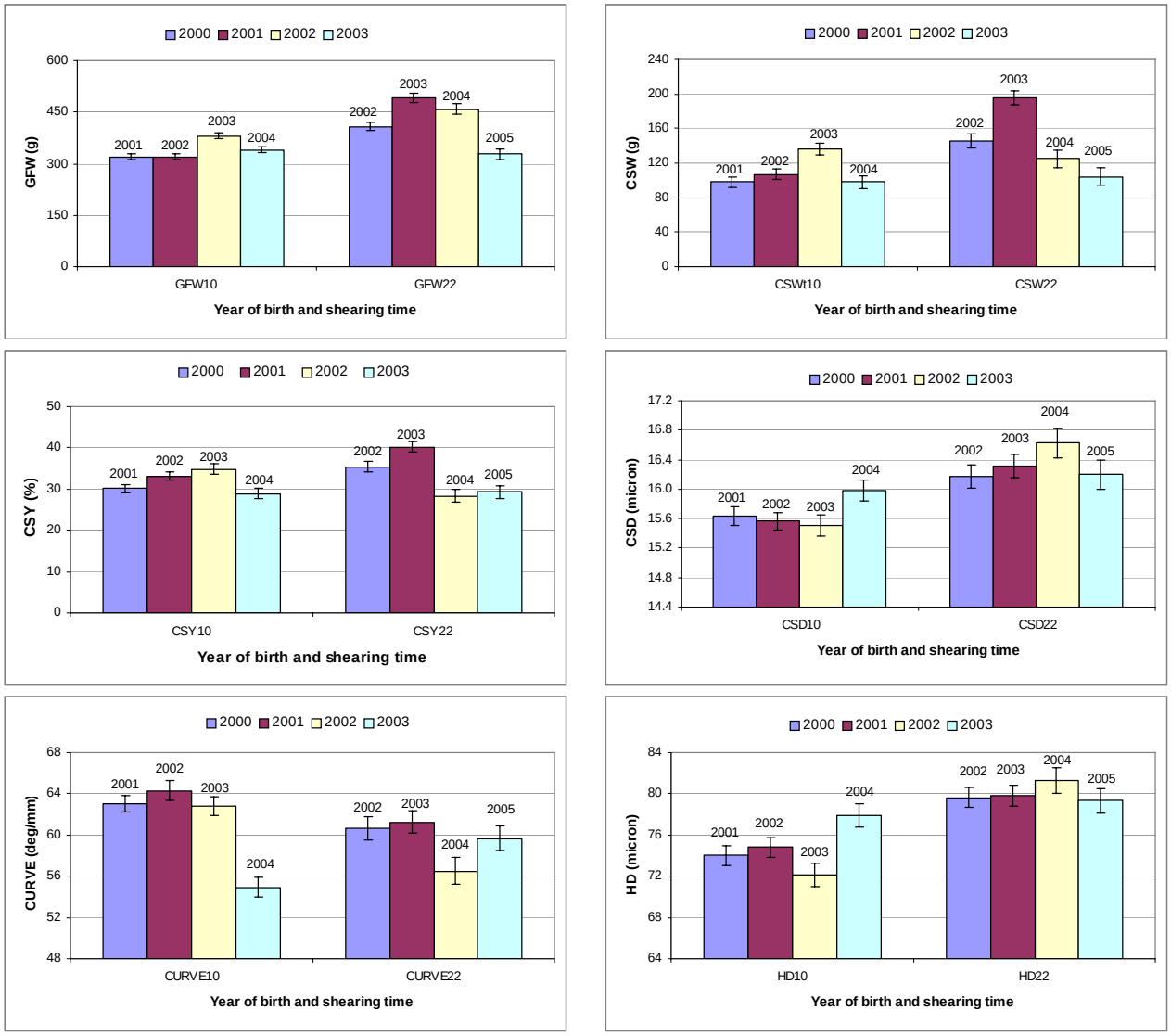

Figure 6.3 Least squares means of fleece traits and their standard errors at two shearing times (10 and 22 months) in Cashmere goats born in 2000, 2001, 2002 and 2003; numbers above bar indicate the year when each shearing was performed

Table 6.8 Data summary and significance of main fixed effects for fleece traits at 10 and 22 months of age in Cashmere goats

Trait	Unit	No. of records	Mean±SD	Fixed effect			
				Birth type	Year	Paddock	Year:Pdk
10 months							
GFW	g	533	332±61	P=0.563	P<0.001	P=0.071	P<0.001
CSW	g	485	105±37	P=0.787	P<0.001	P=0.510	P=0.002
CSY	%	503	31±7	P=0.776	P<0.001	P=0.816	P=0.329
CSD	µm	503	15.8± 0.9	P=0.858	P=0.062	P=0.190	P=0.067
CURVE	deg/mm	450	60.0± 7.1	P=0.623	P<0.001	P=0.149	P=0.120
HD	µm	503	75. 9±7.1	P=0.993	P=0.001	P=0.056	P=0.881
22 months							
GFW	g	460	407±101	P=0.614	P<0.001	P=0.516	P<0.001
CSW	g	441	141±62	P=0.672	P<0.001	P=0.563	P=0.003
CSY	%	461	34±10	P=0.655	P<0.001	P=0.963	P=0.003
CSD	µm	459	16.3± 1.1	P=0.657	P=0.210	P=0.524	P=0.007
CURVE	deg/mm	412	59.3± 7.2	P=0.342	P=0.006	P=0.264	P=0.089
HD	µm	461	79.9±6.7	P=0.289	P=0.581	P=0.418	P=0.229

GFW = greasy fleece weight; CSW = cashmere weight; CSY = cashmere yield; CURVE = mean fibre curvature; HD = hair diameter

Fleece traits for Angora goats: Estimates of genetic parameters

A log likelihood ratio test revealed that model 2 fitting only additive direct effects was the best fit for all fleece traits (P >0.05) with the exception of greasy fleece weight (GFW) at 6 months of age (Table 6.9). Adding the permanent environmental effect of dam increased the log likelihood for GFW at the first shearing, but not significantly at the second and third shearings (Table 6.9).

Table 6.9 Log likelihood values for models with different random effects for fleece traits in Angora goats. The maximum log likelihood for each variable is bolded.

	Characteristic					
Model	GFW	MFD	MFD_SD	CURVE	%MED	%KEMP
	6 months					
1	674.695	-576.078	-84.5307	-820.382	165.021	1099.58
2	681.880	**-557.082**	**-61.8851**	**-815.178**	**179.400**	**1103.07**
3	**685.029**	-556.223	-61.8851	-815.178	179.400	1103.07
	12 months					
1	412.211	-690.900	-184.123	-966.654	448.742	1156.38
2	**424.672**	**-651.620**	**-171.437**	**-950.490**	**475.819**	**1157.13**
3	425.185	-651.620	-171.437	-950.348	475.819	1157.13
	18 months					
1	195.001	-729.259	-288.740	-716.723	216.613	1025.47
2	**202.147**	**-698.889**	**-284.351**	**-702.777**	**237.810**	**1025.60**
3	203.388	-698.889	-284.351	-702.777	237.810	1025.60

Model 1: Significant fixed effects (SFE) only
Model 2: SFE + additive effect of the animal (a)
Model 3: SFE + a + permanent environment effect of the animal's dam (m_{pe})

Univariate heritabilities with standard errors (based on model 2) and genetic and phenotypic correlations between production traits at three shearing times for Angora goats are shown in Table 6.10. Because of the small size of the data set, the standard errors were high for both estimates of heritability and genetic correlations. At all three shearing times, heritability values for all traits with the exception of %KEMP are moderate to high. With increase of age, heritability values of LWT, GFW, CURVE and %MED increased and heritability values of MFD_SD and %KEMP decreased. For MFD, the estimate of heritability at 12 months of age was highest.

Table 6.10 Estimates of genetic parameters for fleece traits in Angora goats. Heritability estimates are on the diagonal) while phenotypic correlations are above the diagonal and genetic correlations are below the diagonal. Standard errors are show in subscript.

Age and variables	LWT	GFW	MFD	MFD_SD	CURVE	%MED	%KEMP$^{0.25}$
6 months							
LWT	**$0.52_{0.14}$**	$0.66_{0.03}$	$0.55_{0.03}$	$0.25_{0.05}$	$-0.37_{0.04}$	$-0.01_{0.05}$	$0.19_{0.05}$
GFW	$0.39_{0.23}$	**$0.38_{0.14}$**	$0.52_{0.04}$	$0.20_{0.04}$	$-0.35_{0.04}$	$0.07_{0.05}$	$0.13_{0.04}$
MFD	$0.65_{0.15}$	$0.27_{0.24}$	**$0.43_{0.13}$**	$0.57_{0.03}$	$-0.50_{0.03}$	$0.05_{0.05}$	$0.20_{0.04}$
MFD_SD	$0.12_{0.25}$	$0.17_{0.26}$	$0.91_{0.09}$	**$0.42_{0.13}$**	$-0.16_{0.05}$	$0.18_{0.05}$	$0.33_{0.04}$
CURVE	$-0.34_{0.28}$	$0.11_{0.42}$	$-0.60_{0.18}$	$-0.29_{0.28}$	**$0.18_{0.09}$**	$0.07_{0.05}$	$-0.05_{0.04}$
%MED	$0.07_{0.26}$	$0.46_{0.25}$	$-0.11_{0.24}$	$0.08_{0.26}$	$0.43_{0..28}$	**$0.40_{0.13}$**	$0.20_{0.04}$
%KEMP$^{0.25}$	-	-	-	-	-	-	**$0.14_{0.09}$**
12 months							
LWT	**$0.55_{0.15}$**	$0.58_{0.04}$	$0.48_{0.04}$	$0.13_{0.05}$	$-0.27_{0.05}$	$0.22_{0.05}$	$0.16_{0.04}$
GFW	$0.55_{0.16}$	**$0.52_{0.15}$**	$0.58_{0.03}$	$0.21_{0.05}$	$-0.27_{0.05}$	$0.19_{0.05}$	$0.30_{0.04}$
MFD	$0.71_{0.12}$	$0.64_{0.12}$	**$0.63_{0.13}$**	$0.32_{0.04}$	$-0.53_{0.04}$	$0.20_{0.05}$	$0.36_{0.04}$
MFD_SD	$-0.11_{0.23}$	$0.28_{0.24}$	$0.59_{0.18}$	**$0.36_{0.13}$**	$0.15_{0.05}$	$0.08_{0.05}$	$0.28_{0.04}$
CURVE	$-0.51_{0.20}$	$-0.14_{0.24}$	$-0.70_{0.14}$	$0.05_{0.28}$	**$0.42_{0.14}$**	$-0.03_{0.05}$	$-0.18_{0.04}$
%MED	$0.38_{0.20}$	$0.41_{0.22}$	$0.18_{0.20}$	$-0.25_{0.24}$	$0.08_{0.24}$	**$0.47_{0.12}$**	$0.16_{0.05}$
%KEMP$^{0.25}$	-	-	-	-	-	-	**$0.06_{0.06}$**
18 months							
LWT	**$0.58_{0.16}$**	$0.61_{0.04}$	$0.50_{0.04}$	$0.21_{0.05}$	$-0.22_{0.05}$	$0.21_{0.05}$	$0.30_{0.04}$
GFW	$0.45_{0.19}$	**$0.57_{0.16}$**	$0.60_{0.04}$	$0.15_{0.05}$	$-0.29_{0.05}$	$0.20_{0.05}$	$0.31_{0.04}$
MFD	$0.50_{0.17}$	$0.47_{0.17}$	**$0.55_{0.14}$**	$0.33_{0.04}$	$-0.49_{0.04}$	$0.27_{0.05}$	$0.42_{0.04}$
MFD_SD	$0.17_{0.33}$	$0.03_{0.34}$	$0.19_{0.29}$	**$0.17_{0.09}$**	$-0.05_{0.05}$	$0.30_{0.05}$	$0.40_{0.04}$
CURVE	$-0.11_{0.25}$	$-0.28_{0.23}$	$-0.69_{0.13}$	$-0.21_{0.33}$	**$0.44_{0.14}$**	$-0.03_{0.05}$	$-0.23_{0.04}$
%MED	$0.10_{0.23}$	$0.09_{0.22}$	$0.11_{0.21}$	$0.12_{0.30}$	$0.30_{0.24}$	**$0.53_{0.13}$**	$0.31_{0.04}$
%KEMP$^{0.25}$	-	-	-	-	-	-	**$0.02_{0.06}$**

(-) – correlations were higher than one and/or standard errors were extremely high due to that "zero" percentages of kemp (%KEMP$^{0.25}$) were recorded for majority of goats.

Live weight had high and positive correlations with greasy fleece weight and fibre diameter, indicating that selection for greasy weight will increase live weight as well. Strong genetic and phenotypic (unfavourable) relationships were observed between greasy fleece weight and fibre diameter. These indicate that diameter will increase rapidly with selection for high greasy fleece weight. Mean fibre curvature had moderate, but negative phenotypic and genetic correlations with other traits. This means that animals with higher degrees of fibre curvature have lower LWT, GFW, MFD, and %KEMP, and fibre curvature will decline with selection for high fleece weight. Percentage of kemp and medullation had weak to moderate, but consistently positive associations with fleece traits except mean fibre curvature. Percentages of medullation rose with increase of percentages of kemp.

Fleece traits for Cashmere goats: Estimates of genetic parameters

A log likelihood test revealed that model 2 with only additive direct effects provided the best fit (*P* >0.05) for all fleece traits in Cashmere goats (Table 6.11). For all fleece traits, Model 3, which included the permanent environmental effect of the dam, was found to fit the data no better than the models without it.

Table 6.11 Log likelihood values for models with different random effects for fleece traits in Cashmere goats. The maximum log likelihood for each variable is bolded.

	Characteristic					
Model	GFW	CSW	CSY	CSD	CURVE	HD
	10 months					
1	- 2307.70	- 1865.41	1057.68	- 210.124	- 1032.56	- 1222.23
2	**- 2299.07**	**- 1844.01**	**1089.33**	**- 189.305**	**- 1020.88**	**- 1204.68**
3	- 2299.05	- 1844.01	1089.33	- 189.305	- 1020.88	- 1204.68
	22 months					
1	- 2118.33	- 1824.57	803.511	- 274.931	- 1005.74	- 1110.67
2	**- 2109.65**	**- 1807.03**	**823.368**	**- 249.785**	**- 999.189**	**- 1093.34**
3	- 2109.65	- 1807.03	823.368	- 249.785	- 999.189	- 1093.34

Model 1: Significant fixed effects (SFE) only
Model 2: SFE + additive effect of the animal (a)
Model 3: SFE + a + permanent environment effect of the animal's dam (m_{pe})

Univariate heritabilities, genetic and phenotypic correlations between production traits at the first two shearing time are shown in Table 6.12.

Table 6.12 Estimates of genetic parameters for fleece traits in Cashmere goats. Heritability estimates are on the diagonal) while phenotypic correlations are above the diagonal and genetic correlations are below the diagonal. Standard errors are show in subscript.

Age and variables	LWT	GFW	CSW	CSY	CSD	CURVE	HD
10 months							
LWT	$\mathbf{0.15_{0.14}}$	$0.21_{0.07}$	$0.07_{0.08}$	$-0.08_{0.07}$	$0.11_{0.07}$	$-0.13_{0.08}$	$0.33_{0.06}$
GFW	-	$\mathbf{0.43_{0.12}}$	$0.75_{0.02}$	$0.33_{0.05}$	$0.29_{0.05}$	$-0.24_{0.05}$	$-0.14_{0.05}$
CSW	-	$0.76_{0.09}$	$\mathbf{0.56_{0.13}}$	$0.85_{0.02}$	$0.47_{0.04}$	$-0.33_{0.05}$	$-0.27_{0.05}$
CSY	-	$0.48_{0.16}$	$0.93_{0.03}$	$\mathbf{0.75_{0.12}}$	$0.44_{0.04}$	$-0.32_{0.05}$	$-0.32_{0.05}$
CSD	-	$0.09_{0.19}$	$0.44_{0.14}$	$0.59_{0.11}$	$\mathbf{0.74_{0.12}}$	$-0.63_{0.04}$	$0.22_{0.05}$
CURVE	-	$-0.12_{0.22}$	$-0.37_{0.19}$	$-0.39_{0.17}$	$-0.54_{0.13}$	$\mathbf{0.48_{0.14}}$	$-0.01_{0.06}$
HD	-	$-0.36_{0.20}$	$-0.42_{0.17}$	$-0.36_{0.16}$	$0.27_{0.17}$	$0.02_{0.22}$	$\mathbf{0.53_{0.12}}$
22 months							
LWT	$\mathbf{0.17_{0.14}}$	$0.39_{0.06}$	$0.29_{0.06}$	$0.15_{0.07}$	$0.26_{0.06}$	$-0.24_{0.06}$	$0.27_{0.06}$
GFW	-	$\mathbf{0.38_{0.13}}$	$0.67_{0.03}$	$0.22_{0.05}$	$0.37_{0.05}$	$-0.27_{0.05}$	$-0.01_{0.05}$
CSW	-	$0.70_{0.13}$	$\mathbf{0.50_{0.13}}$	$0.82_{0.02}$	$0.58_{0.04}$	$-0.35_{0.05}$	$-0.12_{0.05}$
CSY	-	$0.44_{0.21}$	$0.91_{0.05}$	$\mathbf{0.54_{0.13}}$	$0.57_{0.04}$	$-0.27_{0.05}$	$-0.23_{0.05}$
CSD	-	$0.29_{0.18}$	$0.66_{0.12}$	$0.73_{0.10}$	$\mathbf{0.78_{0.13}}$	$-0.61_{0.04}$	$0.18_{0.05}$
CURVE	-	$-0.42_{0.26}$	$-0.61_{0.20}$	$-0.52_{0.22}$	$-0.74_{0.11}$	$\mathbf{0.32_{0.13}}$	$0.03_{0.05}$
HD	-	$-0.37_{0.24}$	$-0.35_{0.22}$	$-0.25_{0.20}$	$0.07_{0.18}$	$0.37_{0.26}$	$\mathbf{0.50_{0.12}}$

(-) – genetic correlations were higher than one and/or standard errors were extremely high due to few records, 295 and 277, for live weights at 10 and 22 months of age, respectively.

Fleece traits were moderate to highly heritable, ranging from 0.32 to 0.78. Greasy fleece weight had high and positive (0.70 at 10 months 0.76 at 22 months) genetic correlations with cashmere weight, indicating that greasy weight can be exploited as an indicator trait of cashmere weight. Strong and unfavourable genetic and phenotypic correlations existed between cashmere weight or yield and cashmere diameter. As observed in Angora goats, curvature of fibre had a moderate to high, but consistently negative correlations with other fleece traits. Animals with highly curved fibres would produce fibres of fine diameter. Because of the small number of records for live weights at shearing, the genetic correlations between live weight and fleece weights were not estimable.

Phenotypic correlations between parasite resistance and production traits

Figure 6.4 shows the phenotypic correlations between live weight and parasite-associated traits in Angora and Cashmere goats from 3 to 6 months of age. The range of phenotypic correlations between live weights and parasite-associated traits was between -0.30 and +0.42 in Angora, and between -0.17 and +0.29 in Cashmere goats. WEC and NEU were mostly negatively correlated with live weights, indicating that kids that were infected and had elevated values of NEU tended to be lighter. The estimates of correlation coefficients in Angora goats were two or more fold higher compared with those in Cashmere goats. EOS, LYM, and PCV were positively associated with live weights. Correlation coefficients between LWT and PCV in Angora goats decreased with age, while in Cashmere goats they increased with age.

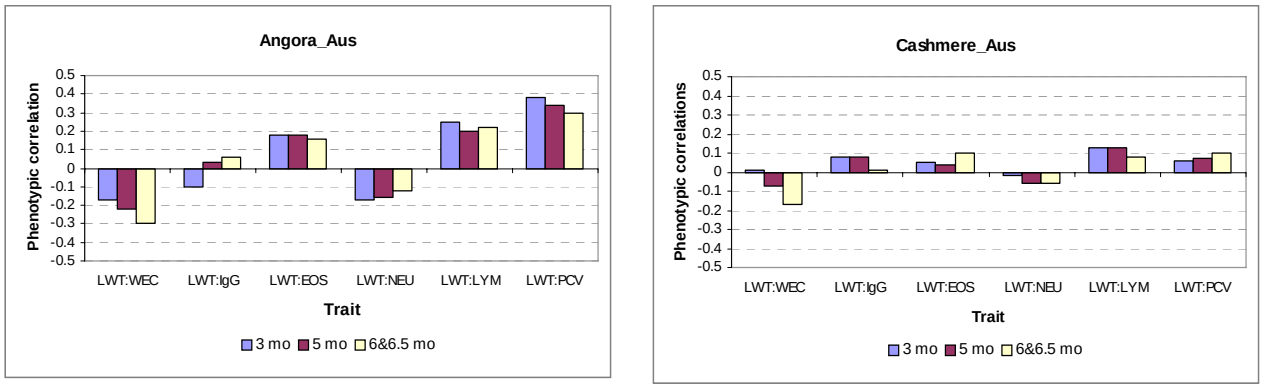

Figure 6.4 Phenotypic correlations between live weights at 6 months of age and parasite-associated traits at 3, 5 and 6 months of age in Angora and Cashmere goats

Phenotypic correlations between fleece traits and parasite-associated traits in Angora and Cashmere goats are reported in Table 6.13 and Table 6.14, respectively. In Angora goats, WEC had weak to moderate, but negative phenotypic correlations (between -0.11 and -0.29) with fleece traits from 6 to 18 months of age. PCV was positively correlated with fleece traits. LYM was positively associated with GFW and MFD at 6.5 months of age but not significantly. There were no significant relationships between fleece traits at the first two shearings and parasite-associated traits at 6.5 months of age in Cashmere goats.

Table 6.13 Phenotypic correlations between fleece traits at 6, 12 and 18 months of age and parasite-associated traits at 6.5 months of age in Angora goats

Trait	Phenotypic correlations (r_p)					
	$WEC^{0.5}$6.5	$IgG^{0.5}$6.5	$EOS^{0.33}$6.5	LYM6.5	NEU6.5	PCV6.5
GFW6	-0.11	0.01	-0.00	0.21	-0.07	0.04
MFD6	-0.16	-0.03	0.00	0.18	0.11	0.09
GFW12	-0.29	0.00	0.08	0.03	-0.01	0.10
MFD12	-0.25	0.02	0.07	0.04	0.08	0.13
GFW18	-0.17	0.02	0.09	0.06	-0.05	0.11
MFD18	-0.14	0.07	0.08	0.06	0.04	0.12

Table 6.14 Phenotypic correlations between fleece traits at 10 and 22 months of age and parasite-associated traits at 6.5 months of age in Cashmere goats.

Trait	Phenotypic correlations (r_p)					
	$WEC^{0.5}$6.5	$IgG^{0.5}$6.5	$EOS^{0.33}$6.5	LYM6.5	NEU6.5	PCV6.5
CSW10	-0.07	0.05	-0.02	0.03	0.02	-0.02
CSD10	0.04	-0.04	-0.07	0.05	-0.04	-0.02
CSW22	-0.04	-0.03	-0.07	0.05	-0.02	-0.07
CSD22	0.03	-0.01	-0.05	0.05	-0.01	0.03

6.3 Discussion

Fixed effects. Twin born kids were lighter than their single born counterparts at 6 months of age for both breeds, but the effect then waned being non-significant by 12 months of age in Angora goats. In cashmere goats a significant difference was still observed at 10 months of age but by 22 months the difference had ceased being statistically different. The effects of birth type on fibre variables were smaller. In Angoras twins had lighter fleeces (120g lighter) at their 1st, but not subsequent, shearings. This was reflected in a significantly lower MFD (0.93µm finer) at the 1st, but not subsequent, shearings. At the first shearing Angora twin born kids also had a lower SD of fibre diameter and a lower proportion of medullated fibres. Birth type had no significant effect on any fleece traits in Cashmere goats nor were there any non-significant trends.

Male Angora goats (wethers) were significantly heavier than female goats, had heavier greasy fleece weights and greater fibre curvature at all ages recorded. Wethers had a lower MFD and %MED at all shearings but the difference only achieved statistical significance at 12 months of age (0.38µm finer, 0.05% less medullation). Sex differences were not determined in Cashmere goats as only female kids were included in the study.

Year effects were highly significant for most variables in both Angora and Cashmere goats and complex to explain. Differences in climatic conditions can be partially explained by differences in climatic conditions during the project (see section 2.2.3) and the interaction of these effects with their effects on the level of gastrointestinal parasitism. However management practices, including supplementary feeding varied widely from year to year. The combination of these effects gave some very large year effects; for example those for 18 month weights in Angora goats which ranged from 20.4kg for 2001 born kids, to 33.6kg for kids born in 2002 (Figure 6.1).

For Cashmere goats, a management group paddock effect was observed on LWT at 3, 5 and 6 months of age, but not subsequently (Table 6.3). This reflects the fact that the kids spent the first 4-5 months of their life (prior to weaning) in the different management groups before being placed in a common environment. There were no significant overall effects of management group paddock on any fleece traits although significant year by paddock interactions were evident at both 10 month and 22 month shearings, particularly the latter. For the earlier shearing these may reflect carry-over effects from the pre-weaning period. For the 22 month shearing it would reflect the fact that the kids are now does and would be in new management groups themselves post-mating.

Heritability estimates

In Angora goats, heritability estimates for live weights were high, varying from 0.52 to 0.58. These estimates were higher than other estimates in the literature, which range between 0.13 and 0.50 (Table 1.3). However, after excluding the maternal permanent environment effect (m_{pe}), the estimates of direct heritability for live weight in Angora goats fell within the range of literature estimates. In the literature, heritability estimates for live weights in Cashmere goat breeds ranged between 0.10 and 0.35 (Table 1.4), which were in agreement with our findings (range 0.06-0.17). Angora goats also demonstrated higher heritability estimates for greasy fleece weight, mean fibre diameter and percentage of medullation at 12 and 18 months of ages than those reported for other Angora goat populations (Table 1.3). The heritability estimates for percentage of kemp was similar to that in New Zealand Angora goats studied by (Nicoll *et al.*, 1989). For Cashmere goats, the heritability estimates for all fleece characteristics were in accordance with other estimates reported in literature (Table 1.4). There are no reports on estimates of genetic parameters for mean fibre curvature in Angora and Cashmere goats. Our estimates of heritabilities for mean fibre curvature showed that the fibre curvature is a moderately heritable trait. The estimates of heritability for the majority of fleece traits and live weights were moderate to high, indicating that rapid genetic progress is possible through objective measurement and selection for these traits.

Maternal effects

We did not have enough repeated records of dams in the Angora goat herd to fit maternal effect as a genetic effect in our model. In order to estimate any possible effect from the dam we fitted only the maternal permanent environment (m_{pe}) effect (model 3). Hence, we are not sure whether these maternal effects are genetic or environmental. Our results indicated that the permanent environment effect of the dam should be taken into account in estimating the heritability of live weight at early ages (3, 5, and 6 months). In both Angora and Cashmere goats, the m_{pe} effect was non-significant for any studied fleece characteristics except GFW at 6 months of age. A similar significant maternal effect for live weight but not for fleece characteristics was also obtained in Scottish crossbred cashmere-producing goats (Bishop and Russel, 1996; Vagenas *et al.*, 2002).

Correlations between production traits

Strong positive genetic correlations were obtained between GFW and CSW or CSY, suggesting that GFW could be used as indirect measure of CSW and CSY in the absence of direct measurement of the latter traits. However, as reported in previous studies, positive (unfavourable) genetic relationships between mean fibre diameter (MFD) and fleece weights were observed in both Angoras and Cashmeres. Therefore, both CSW and CSD should both be included in selection indices for selection for heavy down weight while either holding or slightly reducing down diameter (CSD). With the advent of OFDA testing of cashmere yield and diameter, the costs of objective measurement of cashmere traits have declined and testing can be more widely implemented. Estimates of genetic and phenotypic correlations in this study fall within the range of estimates reported in literature (Tables 1.4 and 1.5). In both populations, fibre curvature was negatively (genetically and phenotypically) correlated with almost all other fleece traits as well as with live weight. This means that selection on heavier live weight, higher greasy fleece weight and cashmere weight, and higher cashmere yield lead to reduced fibre curvature and broader fibres. Because of small size of data sets, standard errors of estimated genetic parameters were comparatively high in both populations.

Phenotypic correlations between production and parasite-associated traits

Weak to moderate associations between live weights and parasite-associated traits including WEC, IgG, EOS, NEU, LYM and PCV were observed in the two studied populations. Phenotypic associations were stronger in Angora goats than in Cashmeres although generally in the same direction. Negative correlations between live weights and WEC or NEU suggest that heavier kids shed fewer worm eggs and have lower circulating concentrations of neutrophils compared with lighter kids. On the other hand, positive correlations between LWT and EOS, LYM and PCV indicate that kids with high eosinophil and lymphocyte counts and having high packed cell volumes, have heavier body weights (Figure 6.4). A negative phenotypic relationship (-0.10) between WEC and live weight was also observed in goats under African conditions (Baker *et al.*, 2001). The relationships between body weight and haematological variables are consistent with the effects of stress on these variables. Elevated stress or immunosuppressive hormones such as cortisol induce elevated neutrophil counts and depressed lymphocyte and eosinophil counts (Jain, 1986) as well reducing growth rate. It is possible that phenotypic differences in nutritional, disease and environmental stress status are in part mediated by the hypothalamo-pituitary-adrenal stress axis. In Angora goats significant negative correlations were observed between parasite-associated traits and GFW and MFD at 6, 12 and 18 months of age. Thus Angora kids that had high WEC tended to have lighter fleece weight and finer fibre. There were no other important associations between parasite-associated traits and fibre traits in Angora goats, and none at all in Cashmere goats.

In the absence of estimable genetic correlations between production and parasite-associated traits in this study, the reader is referred to section 1.5 of the report where the literature on the topic is reviewed.

6.3.2 Summary

- Cashmere kids were heavier than Angora kids at all ages although caution must be exercised in interpreting this due to the different environments in which the different goat breeds ran. In both populations, year of birth had a great impact on production traits at all ages. Sex and age in days significantly affected live weights and fleece weights. Effect of birth type was important for live weights at early ages.
- A simple animal model without maternal effects gave the best fit for fleece traits and some live weights. Maternal (permanent environment) effects were important for live weights at 3, 5 and 6 months of age in Angora goats and for live weight at 3 months of age in Cashmere goats.
- The heritability estimates of production traits in Angora goats were higher than those for Cashmere goats and other published estimates. The majority of fleece traits were highly heritable in both studied populations as reported in the literature. The heritability estimates for mean fibre curvature are novel for goats and were moderate. Estimates of heritability of percentage of kemp in Angora fleece were low.
- Strong genetic and phenotypic correlations existed between greasy fleece weight, cashmere weight and cashmere yield in Cashmere goats. Mean fibre diameter was positively correlated with fleece weight and cashmere yield, an unfavourable direction for fibre producers who want to increase fibre production and reduce diameter simultaneously. Mean fibre diameter was negatively correlated with mean fibre curvature indicating that finer fibres have a higher crimp count.
- In the absence of laboratory-based objective measurement the best estimates of fibre production and diameter in the current study were fleece weight for the former and curvature (crimp) for the latter. The strong association between curvature and fibre diameter in both goat breeds suggests that selection for increased crimp frequency would lead to correlated reductions in fibre diameter. Cashmere down length was not recorded in the current project, but it is another well-established alternative to measurement of cashmere down production (Pattie and Restall, 1991).
- Live weights were weakly but negatively correlated with faecal egg counts and circulating neutrophil counts. Negative phenotypic relationships between WEC and GFW or MFD were observed in Angora goats only. There were no significant correlations between fleece traits at 10 and 22 months of age and parasite-associated traits at 6.5 months of age in Cashmere goats. Due to the size and structure of the data set, genetic correlations between parasite-associated traits and production traits could not be estimated. However based on the phenotypic relationships, there is little evidence that selection for parasite resistance would result in unfavourable effects on production traits.

References

Al-Atiyat R (2004) Molecular population genetics of Merino sheep. PhD thesis, University of New England, NSW, Australia.

Albers GAA, Gray GD, Burgess SE, Adams DB, Baker JSF, Le Jambre LF, Piper LR (1984) Breeding *Haemonchus contortus* resistant sheep - Problems and prospects. In 'Immunogenetic approaches to the control of endoparasites with reference to parasites in sheep'. (Eds JK Dineen, PM Outteridge) pp. 41-52. (CSIRO and Australian Wool Corporation: Melbourne).

Albers GAA, Gray GD, Piper LR, Barker JSF, Le Jambre LF, Barger IA (1987) The genetics of resistance and resilience to *Haemonchus contortus* infections in young Merino sheep. *Int. J. Parasitol.* **17**, 1355-1363.

Allain D, Roguet JM (2003) Genetic and nongenetic factors influencing mohair production traits within the national seletion scheme of Angora goats in France. *Livestock Production Science* **82**, 129-137.

Allain D, Roguet JM (2006) Genetic and non-genetic variability of OFDA-medullated fibre contents and other fleece traits in the French Angora goats. *Small Ruminant Research* **65**, 217-222.

Aumont G, Mandonnet N, Bouix J, Vu Tien Khang J, Gruner L, Menedez Buxadera A, Varo H, Arquet R (1998) Genetic resistance to gastro-intestinal parasites in Creole goats bred under a humid tropical environment. In '6th World congress on genetics applied to livestock production'. Armidale, NSW, Australia pp. 265-268.

Bain (1999) Irradiated larvae for helminth control in livestock. *International Journal for Parasitology* **29**, 185-191.

Baker L, Rege JEO, Tembly S, Mukasa-Mugerwa E, Anindo D, Mwamachi DM, Thorpe W, Lahlou-Kassi A (1998) Genetic resistance to gastrointestinal nematodes parasites in some indigenous breeds of sheep and goats in East Africa. In '6th World congress on genetics applied to livestock production'. Armidale, NSW, Australia pp. 269-272.

Baker RL, Audho JO, Aduda EO, Thorpe W (2001) Genetic resistance to gastro-intestinal nematode parasites in Galla and Small East African goats in the sub-humid tropics. *Animal Science* **73**, 61-70.

Baker RL, Mwamachi DM, Audho JO, Aduda EO, Thorpe W (1998) Resistance of Galla and Small East African goats in the sub-humid tropics to gastrointestinal nematode infections and the peri-parturient rise in faecal egg counts. *Vet Parasitol* **79**, 53-64.

Baker RL, Mwamachi DM, Audho JO, Aduda EO, Thorpe W (1999) Genetic resistence to gastro-intestinal nematode parasites in Red Massai, Dorper and MassaixDorper ewes in the sub-humid tropics. *Animal Science* **69**, 335-344.

Baker RL, Nagda S, Rodriguez-Zas SL, Southey BR, Audho JO, Aduda EO, Thorpe W (2003) Resistence and resilience to gastro-intestinal nematode parasites and relationships with productivity of Red Maasai, Dorper and Red Maasai x Dorper crossbred lambs in the sub-humid tropics *Animal Science* **76**, 119-136.

Baker RL, Rege JEO, *et al.* (1994) Genetic resistance to diseases and other stresses in improvement of ruminant livestock in the tropics. In '5th World Congress of Genetics Applied to Livestock Production' pp. 405-412.

Baker RL, Watson TG, Bisset SA, Vlassoff A, Douch PGC (1991) Breeding sheep in New Zealand for resistance to internal parasitess: Research results and commercial application. In 'Breeding for Disease Resistance in Sheep'. (Eds GD Gray, RR Woolaston) pp. 19-32. (Wool Research and Development Coorporation: Melbourne).

Balic A, Bowles VM, Meeusen EN (2000) The immunobiology of gastrointestinal nematode infections in ruminants. *Advances in Parasitology* **45**, 181-241.

Barger I (1994) Rotational grazing for control of gastrointestinal nematodes of goats in a wet tropical environment. *Veterinary Parasitology* **53**, 109-116.

Barger IA, Southcott WH (1978) Parasitism and production in weaner sheep grazing alternately with cattle. *Australian Journal of Experimental Agriculture and Animal Husbandry* **18**, 340-346.

Barnes EH, Dobson RJ (1993) Persistence of acquired immunity to *Trichostrongylus colubriformis* in sheep after termination of infection. *International Journal for Parasitology* **23**, 1019-1026.

Barnett NL, Purvis IW, van Hest B, Franklin IR (1999) The accuracy of current dam pedigree recording strategies employed by stud Merino breeders. *Proceedings of the Association for the Advancement of Animal Breeding and Genetics* **13**, 373-376.

Benitez-Usher C, Armour J, Duncan JL, Urquhart GM, Gettinby G (1977) A study of some factors influencing the immunization of sheep against Haemonchus contortus using attenuated larvae. *Veterinary Parasitology* **3**, 327-342.

Besier RB, Love SCJ (2003) Anthelmintic resistance in sheep nematodes in Australia: the need for new approaches. *Australian Journal of Experimental Agriculture* **13**, 1383-1391.

Bigham ML, Morris CA, Southey BR, Baker RL (1993) Heritabilities and genetic correlations for live weight and fibre traits in New Zealand Cashmere goats. *Livestock Production Science* **33**, 91-104.

Bishop SC, Russel AJF (1996) The inheritance of fibre traits in crossbred population of cashmere goats. *Animal Science* **63**, 429-436.

Bishop SC, Stear MJ (1999) Genetic and epidemiological relationships between productivity and disease resistance: gastro-intestinal parasite infection in growing lambs. *Animal Science* **69**, 515-524.

Bisset SA, Morris CA, Squire DR, Hickey SM (1996) Genetics of resilience to nematode parasites in young Romney sheep: Use of weight gain under challenge to assess individual anthelmintic treatment requirements. *New Zealand Journal ofAgricultural Research* **39**, 313-323.

Bisset SA, Morris CA, Squire DR, Hickey SM, Wheeler M (1994) Genetics of resilience to nematode parasites in Romney sheep. *New Zealand Journal of Agricultural Research* **37**, 521-534.

Bisset SA, Vlassoff A, Morris CA, Southey BR, Baker RL, Parker AGH (1992) Heritability and genetic correlations among faecal egg counts and productivity traits in Romney sheep. *New Zealand Journal of Agricultural Research* **35**, 51-58.

Blattman AN, Hulme DJ, Kinghorn BP, Woolaston RR, Gray GD, Beh KJ (1993) A search for associations between major histocompatibility complex restriction fragment length polymorphism bands and resistance to Haemonchus contortus infection in sheep. *Anim Genet* **24**, 277-282.

Brohede J, Ellegren H (1999) Microsatellite evolution: polarity of substitutions with repeats and neatrality of flanking sequences. *Proceedings of the Royal Society of London* **266**, 825-833.

Buddle BM, Jowett G, Green RS, Dough PGC, Risdon PL (1992) Association of blood eosinophilia with the expression of resistence in Romney lambs to nematodes. *International Journal for Parasitology* **22**, 955-960.

Colvin AF, Walkden-Brown SW, Knox MR, Scott JM (2007) Intensive rotational grazing assists control of gastrointestinal nematodiasis of sheep in a cool temperate environment with summer-dominant rainfall. *Veterinary Parasitology*, (submitted).

Connan RM (1967) Observations on the post-parturient rise in the faecal nematode egg count of ewes. *The Veterinary Record* **80**, 401-404.

Coop RL, Holmes PH (1996) Nutrition and parasite interaction. *Int. J. Parasitol.* **26**, 951-962.

Costa CAF, Viera LS, Berne MEA, Silva MUD, Guidoni AL, Figueiredo EAP (2000) Variability of resistance in goats infected with *Haemonchus contortus* in Brazil. *Veterinary Parasitology* **88**, 153-158.

Cummins LJ, Thosmpson RL, Yong WK, Riffin GG, Goddard ME, Callinan APL, Saunders MJ (1991) Genetics of Ostertagia selection lines. In 'Breeding for Disease Resistance In Sheep'. (Eds GD Gray, RR Woolaston) pp. 11-18. (Wool Research and Development Corporation: Melbourne).

Dakin EE, Avise JC (2004) Microsatellite null alleles in parentage analysis. *Journal of Heridity* **93**, 504-509.

Davies G, Stear MJ, Bishop SC (2005) Genetic relationships between indicator traits and nematode parasite infection levels in 6-month-old lambs. *Animal Science* **80**, 143-150.

Dawkins HJ, Windon RG, Eagleson GK (1989) Eosinophil responses in sheep selected for high and low responsiveness to *Trichostrongylus colubriformis*. *International Journal for Parasitology* **19**, 199-205.

Donald AD, Southcott WH, Dineen JK (Eds) (1978) 'The epidemiology and control of gastrointestinal parasites of sheep in Australia.' (CSIRO: Melbourne).

Douch PGC, Green RS, Morris CA, Bisset SA, Vlassof A, Baker RL, Watson TG, Hurford AP, Wheeler M (1995) Genetic and phenotypic relationships among anti-*Trichostrongylus colubriformis* antibody level, faecal egg count and body weight traits in grazing Romney sheep. *Livestock Production Science* **41**, 12-132.

Douch PGC, Green RS, Morris CA, Hickey SM (1995) Genetic factors affecting antibody response to four species of nematode parasites in Romney ewe lambs. *International Journal for Parasitology* **25**, 823-828.

Eady SJ, Woolaston RR, Lewer RP, Raadsma HW, Swan AA, Ponzoni RW (1998) Resistance to nematode parasites in Merino sheep: correlation with production traits. *Australian Journal of Agricultural Research* **49**, 1201-1211.

Eady SJ, Woolaston RR, Mortimer SI (1994) Internal parasite resistence of Merino flocks selected for production. In *5th World Congress of Genetics Applied to Livestock Production*, pp. 289-292

Emery DL, McClure SJ, Davey RJ, Bendixsen T (1999) Induction of protective immunity to *Trichostrongylus colubriformis* in neonatal Merino lambs. *International Journal for Parasitology* **29**, 1037-1046.

Falconer DS, Mackay TFC (1989) Introduction to quantitative genetics. 3rd edition, Longman Group Ltd, London.

Garrick DJ, Blair, H. T. and Bisset, S. A. (1993) . (1993) Selection of Romney sheep grazing nematode infested pasture. In 'Australian Association of Animal Breeding and Generics' pp. 448-451.

Gifford DR, Ponzoni RW, Ellis NJS, Levinge FCR, Milne ML (1989) Phenotypic and genetic parameters for production characteristics of Cashmere goats in South Australia. *Proceedings of the 3rd International Conference, Adelaide, Australia*, .

Gifford DR, Ponzoni RW, Lampe RJ, Burr J (1991) Phenotypic and genetic parameters of fleece traits and live weight in South Australian Angora goats. *Small Ruminant Research* **4**, 293-302.

Gill HS (1991) Genetic control of acquired resistance to haemonchosis in Merino lambs. *Parasite*

immunology **13**, 617-628.
Gilmour AR, Gogel BJ, Gullis BR, Welham SJ, Thompson R (2002) ASReml user guide release 1.0. VSN International Ltd. Hemel Hempstead, U.K.
Gray GD (1995) Genetic variation in resistance to parasites. In 'Breeding for resistance to infectious diseases in small ruminants'. (Eds GD Gray, RR Woolaston, BT Eaton) pp. 43-51. (ACIAR: Canberra).
Gray GD (1997) The use of genetically resistant sheep to control nematode parasitism. *Veterinary Parasitology* **72**, 345-366.
Greeff JC, Karlsson LJE (1998) The genetic relationship between faecal consistency, faecal worm egg counts and wool traits in Merino. In '6th World Congress on Genetics Applied to Livestock Production'. Armidale pp. 63-66. (World Congress on Genetics Applied to Livestock Production).
Gruner L, Lantier F (1995) Breeding for resistance to infectious diseases of small ruminants in Europe. In the breeding for resistance to infectious diseases in small ruminants (eds.Gray, G.D., Woolaston, R.R., and Eaton, B.T.). ACIAR, Canberra, Australia.
Heyen DW, Beever JE, Da Y, Evert RE, Green C, Bates SRE, Ziegle JS, Lewin HA (1997) Exculsion probabilities of 22 bovine microsatellite markers in flourescent multiplexes for semiautomated parentage testing. *Animal Genetics* **28**, 21-27.
Hohenhaus MA, Outteridge PM (1995) The immunogenetics of resistance to Trichostrongylus colubriformis and Haemonchus contortus parasites in sheep. *Br Vet J* **151**, 119-140.
Hoste H, Chartier C, Le Frileus Y (2002) Control of gastrointestinal parasitism with nematodes in dairy goats by treating the host category at risk. *Veterinary Research* **33**, 531-545.
Hoste H, Torres-Acosta JF, Paolini V, Aguilar-Caballero A, Etter E, Lefrileux Y, Chartier C, Broqua C (2005) Interactions between nutrition and gastrointestinal infections with parasitic nematodes in goats. *Small Ruminant Research* **60**, 141-151.
Howse SW, Blair HT, Garrick DJ, Pomroy.W.E. (1992) A comparison of internal parasitism in fleece weight selected and control Romney sheep. *Proceedings of the New Zealand Society of Animal Production* **52**, 57-60.
Ibarguchi G, Gissing GJ, Gaston AJ, Boag PT, Friesen VL (2004) Male-biased mutation rates and the oversetimation of extrapair paternity: problem, solution, and illustration using thick-billed murres (*Uria lomvia*, Alcidae). *Journal of Heredity* **95**, 209-216.
Isberg SR, Chen Y, Barker SG, Moran C (2004) Analysis of microsatellites and parentage testing in saltwater crocodiles *Journal of Heridity* **95**, 445-449.
Jackson F (2000) Options for the sustainable control of gastrointestinal nematode infections in goat production systems in Europe. *Proceedings 7th International Conference on Goats, France*, pp. 789-792.
Jain NC (1986), 'Schalm's Veterinary Haematology.' (Lea and Febiger: Philadelphia).
Jamieson A, Taylor SCS (1997) Comparisons of three probability formulae for parentage exclusion. *Animal Genetics* **28**, 397-400.
Jarret WFH, McIntyre WIM, Jennings FW, Mulligan W (1957) The natural history of parasitic bronchitis with notes on prophylaxis and treatment. *Vetrinary Record* **69**, 1329-1340.
Javanovic M, Sokolic A, Morsesijan M, Cuperlovic K (1965) Immunisation of sheep with irradiated larvae of *Dictyocaulus filaria*. *British Veterinary Journal* **121**, 119-113.
Kahn LP, Walkden-Brown SW, Scrivener CJ, Crampton A (2006) A structured, evidence-based approach as an aid when deciding the need for anthelmintic treatment of sheep. In 'Proceedings of the Australian Sheep Veterinarians Annual Conference'. Wagga. (Ed. C Trengrove) pp. 48-52. (Australian Sheep Veterinarians).
Karlsson LJE, Greeff JC, Harris JF (1995) Genetic Trends In A Selection Line For Low Faecal Worm Egg Count. In 'Australian Association of Animal Breeding Genetics'. Adelaide, South Australia pp. 122 - 125. (Australian Association of Animal Breeding Genetics).
Kaufmann J (1996) Parasitic infections of domestic animals: A diagnostic manual. Switzerland.
Khusro M, Van der Werf JHJ, Brown DJ, Ball A (2004) Across flock (co)variance components for faecal worm egg count, live weight, and fleece traits for Australian merinos. *Livestock Production Science* **91**, 35-43.
Knox DP, Smith WD (2001) Vaccination against gastrointestinal nematode parasities of ruminants using gut-expressed antigens. *Veterinary Parasitology* **100**, 21-32.
Le Jambre L (1984) Stocking rate effects on the worm burdens of Angora goats and Merino sheep. *Aust. Vet. J.* **61**, 280-282.
Le Jambre LF (1993) Molecular variation in trichostrongylid nematodes from sheep and cattle. *Acta Tropica* **53**, 331-343.
Le Jambre LF, Royal WM (1976) A comparison of worm burdens in grazing merino sheep and angora goats. *Aust. Vet. J.* **52**, 181-183.
Luikart G, Biju-Duval M-P, Ertugrul O, Zagdsuren Y, Maudet C, Taberlet P (1999) Power of 22 microsatellite markers in flourescent multiplexes for parentage testing in goats (*Capra hircus*). *Animal Genetics* **30**, 431-438.
MacRae JC (1993) Metabolic consequences of intestinal parasitism. *Proc. Nutr. Soc.* **52**, 121-130.
MAFF (1986) 'Manual of veterinary parasitological laboratory techniques.' (Her Majesty's Stationary Office: London).

Mandonnet N, Aumont G, Fleury J, Arquet R, Varo H, Gruner L, Bouix J, Vu Tien Khang J (2001) Assessment of genetic variability of resistance to gastrointestinal nematode parasites in Greole goats in the humid tropics. *Journal of Animal Science* **79**, 1706-1712.

Marshall TC, Slate J, Kruuk LEB, Pemberton JM (1998) Statistical confidence for likelihood-based paternity inference in natural populations. *Molecular Ecology* **7**, 639-655.

MathSoft (1999) 'S-PLUS 2000 User's Guide.' (Seatle, Washington, USA).

McClure SJ, Emery DL, Bendixsen T, Davey RJ (1998) Attempts to generate immunity against *Trichostrogylus colubriformis* and *Haemonchus contortus* in young lambs by vaccination with viable parasites. *International Journal for Parasitology* **28**, 739-746.

McDonald BJ (1988) Estimation of cashmere production from cashmere fibre length in goats. *Australian Journal of Experimental Agriculture* **28**, 37-39.

McEwan JC, Dodds KG, Greer GJ, Bain WE, Duncan SJ (1995) Genetic estimates for parasite resistance traits in sheep and their correlations with production traits. *New Zealand Journal of Zoology* **22**, 177.

McEwan JC, Dodds KG, Watson TG, Gree GJ, Hosking BC, Douch PGC (1995) Selection for host resistance to round worms by the Newzealand sheep breeding industry: The WormFEC service. In ' Proceedings of Australian Association of Animal Breeding and Genetics' pp. 70-73.

McEwan JC, Mason P, Baker RL, Clarke JN, Hickey SM, Turner K (1992) Effect of selection for productive traits on internal parasite resistance in sheep. In 'Proceedings of the New Zealand Society of Animal Production' pp. 53-57.

McMillan WH, Hall DRH, Watson TG (1992) Reproduction in flocks selected for and against lamb faecal egg count. In 'Proceedings of the New Zealand Society of Animal Production' pp. 65-68.

Meeusen ENT (1999) Immunology of helminth infections, with special reference to immunopathology. *Vet. Parasitol.* **84**, 259-273.

Merchant M, Riach DJ (2003) Changes in skin and fleece characteristics of Scotish cashmere goats following selection for increased annual production and decreased fibre diameter. *Animal Science* **76**, 517-512.

Miller JE, Bishop SC, Cockett NE, McGraw RA (2006) Segregation of natural and experimental gastrointestinal nematode infection in F2 progeny of susceptible Suffolk and resistant Gulf Coast Native sheep and its usefulness in assessment of genetic variation. *Veterinary Parasitology* **10**, 1016-1022.

Miller TA (1978) Industrial development and field use of canine hookworm vaccine. *Advances in Parasitology* **16**, 33-36.

Morris CA, Bisset SA, Vlassoff A, Baker RL, Watson TG, Wheeler M (1997) Yearling and ewe fleece weights in Romney and Perendale flocks selected for Divergence in Faecal nematode egg count. In 'Association for the Advancement of Animal Breeding and Genetics'. Dubbo, NSW, Australia pp. 50-52. (Association for the Advancement of Animal Breeding and Genetics).

Morris CA, Wheeler M, Hosking BC, Watson TG, Hurford AP, Foote BJ, Foote JF (1997) Genetic parameters for milk yield and faecal nematode egg count in Saanen does. *New Zealand Journal of Agricultural Research* **50**, 523-528.

Mulligan W, Gordon H, Stewart DF, Wagland BM (1961) The use of irradiated larvae as immunizing agents in *Haemonchus contortus* and *Trichostrongylus colubriformis* infections of sheep. *Australian Journal of Agricultural Research* **12**, 1175-1187.

Nicoll GB, Bigham ML, Alderton MJ (1989) Estimates of environmental effects and genetic parameters for live weights and fleece traits of Angora goats. *Proceedings of the New Zealand Society of Animal Production* **49**, 183-190.

Niven P, Anderson N, Vizard AL (2002) The integration of grazing management and summer treatments for the control of trichostrongylid infections in Merino weaners. *Australian Veterinary Journal* **80**, 559-566.

O'Connor LJ, Walkden-Brown SW, Kahn LP (2006) Ecology of the free-living stages of major trichostrongylid parasites of sheep. *Veterinary Parasitology* **142**, 1-15.

O'Meara TJ, Raadsma HW (1995) Phenotypic and genetic indicators of resistance to ectopathogens. In 'Breeding for resistance to infectious diseases in small ruminants'. (Eds DG Gray, RR Woolaston, BT Eaton) pp. 187-218. (ACIAR: Canberra).

Olayemi ME (2004) Genetic resistance to gastrointestinal nematodes in fibre-producing goats and sheep. PhD, University of New England.

Pattie WA, Restall BJ (1989) The inheritance of Cashmere in Australian Goats. 2. Genetic Parameters and breeding Values. *Livestock Production Science* **21**, 251-261.

Peterson AD, Gherardi SG (1996) Measurement of cashmere yield and mean fibre diameter using the Optical Fibre Diameter Analyser. *Australian Journal of Experimental Agriculture* **36**, 429-435.

Piper LR (1987) Genetic variation in resistance to internal parasites. In 'Merino Improvment Program In Australia'. (Ed. BJ McGuirk) pp. 351-363. (Australian Wool Cooperation: Melbourne).

Piper LR, Le Jambre LF, Southcott WH, Chang TS (1978) Natural worm burdens in Dorset Horn, Merino and Corriedale weaners and their crosses. *Proceedings of Australian Association of Animal production*, pp. 276.

Piper LRaB, 1. A. (1988) Proceedings Of , (1988) Resistance to gastrointestinal strongyles: Feasibility of breeding programme. In 'The 3rd World Congress On Sheep And Beef Cattle Breeding'. Paris, pp. 291-294.

Pomroy W, Charleston W (1989) Development of resistance to Trichostrongylus colubriformis in goats. *Vet. Parasitol.* **33**, 283-288.

Pomroy WE (1996) Anthelmintic resistance in goats. In 'VI International Conference on Goats'. Beijing, May 5-11, 1996. (Ed. PJ Host) pp. 717-726. (International Academic Publishers, Beijing.).

Pritchard RK (1990) Anthelmintic resistance in nematodes: extent, recent understanding and future directions for control and research. *International Journal for Parasitology* **20**, 515-523.

Raadsma HW, Gray GD, Woolaston RR (1997) Genetics of disease resistance and vaccine response. In 'The Genetics of Sheep'. (Eds L Piper, A Ruvinsky) pp. 199-224. (CAB International: Wallingford Oxon UK).

Rainbird MA, Macmillan D, Meeusen ENT (1998) Eosinophil-mediated killing of Haemonchus contortus larvae: effect of eosinophil activation and role of antibody, compliment and IL-5. *Parasite immunology* **20**, 93-103.

Rickard JM (1990) Current research on anthelmintics in goats. In 'Goat health and production. Proceedings 134 of the post-graduate committee in Veterinary Science, University of Sydney.' (Ed. KG Thompson) pp. 287-299. (Post-graduate committee in Veterinary Science, University of Sydney, Sydney.).

Rothwell J, Sangster N (1997) Haemonchus contortus: the uptake and metabolism of closantel. *Int J Parasitol* **27**, 313-319.

Ruiz A, Molina JM, Gonzalez JF, Conde MM, Martin S, Hernandez YI (2004) Immunoprotection in goats *Haemonchus contortus* after immunization with cysteine protease enriched protein fractions. *Veterinary Research* **34**, 565-572.

Sambrook J, Fritsch E, Maniatis T (1989) Molecular cloning: a laboratory manual, Book 2. Cold spring harbor laboratory press, New York, USA.

Scarfe AD (2003) Approaches to managing gastrointestinal nematodes. Meat goat production Handbook, retrieved from http://www.clemson.edu/agronomy/goats/handbook/toc.html, REDC, Raleigh, North Carolina.

Schibler L, Vaiman D, Oustry A, Giraud-Delville C, Chibiu EP (1998) Comparative gene mapping: a fine-scale survey of chromosome rearrangements between ruminants and humans. *Genome Research* **8**, 901-915.

Schnabel RD, Ward TJ, Derr JN (2000) Validation of 15 microsatellites for parentage testing in North American bison, *Bison bison* and domestic cattle. *Animal Genetics* **31**, 360-366.

Scrivener CJ, Kahn LP, Walkden-Brown SW (2006) No loss of sheep production with IPM-sheep control programs. In 'Proceedings of the Australian Sheep Veterinarians Annual Conference'. Wagga. (Ed. C Trengrove) pp. 27-37. (Australian Sheep Veterinarians).

Shelton M, Bassett JW (1970) Estimation of certain genetic parameters relating to Angora goats. Texas Agricultural Experiment Station. Research report (PR-2750): 2738-2741.

Smith WD, Angus KW (1980) Haemonchus contortus: attempts to immunise lambs with irradiated larvae. *Res Vet Sci* **29**, 45-50.

Snyman MA, Olivier JJ (1996) Genetic parameters for body weight, fleece weight and fibre diameter in south African Angora goats. *Livestock Production Science* **47**, 1-6.

Southcott WH, Barger IA (1975) Control of nematode parasites by grazing management –II. Decontamination of sheep and cattle pastures by varying periods of grazing with alternate host. *International Journal for Parasitology* **5**, 45-49.

Stear MJ, Bardain K, *et al.* (1997) How hosts control worms. *Nature* **389**, 27.

Stear MJ, Strain S, Bishop SC (1999) Mechanisms underlying resistance to nematode infection. *International Journal for Parasitology* **29**, 51-56.

Strain SAJ, Stear MJ (2001) The influence of protein supplementation on the immune response to *Haemonchus contortus*. *Parasite immunology* **23**, 527-531.

Sunduimijid B (2007) Comparative genetic studies of Angora and Cashmere goats. PhD, University of New England.

Taddeo HR, Allain D, J M, Rochambeau H, Manfredi E (1998) Genetic parameter estimates of production traits of Angora goats in Argentina. *Small Ruminant Research* **28**, 217-223.

Urquhart GM, Jarret WFH, Jennings F.W, MacIntyre W.I.M, Mulligan W (1966) Relationship between age and succesful vaccination with irradiated larvae. *American Journal of Veterinary Research* **27**, 1645-1648.

Vagenas D, Jackson F, Russel AJF, Merchant M, Wright IA, Bishop SC (2002) Genetic control of resistance to gastro-intestinal parasites in crossbred cashmere-producing goats: responses to selection, genetic parameters and relationships with production traits. *Animal Science* **74**, 199-208.

Van Treuren R (1998) Estimating null allele frequencies at a microsatellite locus in the oystercatcher (*Haematopus ostralegus*). *Molecular Ecology* **7**, 1413-1417.

van Wyk J (2001) Refugia--overlooked as perhaps the most potent factor concerning the development of anthelmintic resistance. *Onderstepoort Journal of Veterinary Research* **68**, 55-67.

Van-Houtert MF, Sykes AR (1996) Implications of nutrition for the ability of ruminants to withstand gastro-intestinal nematode iinfections. *Int. J. Parasitol.* **26**, 1151-1167.

Vanimisetti HB, Andrew SL, Zajac AM, Notter DR (2004) Inheritance of faecal egg count and packed cell volume and their relationship with production traits in sheep infected with *Haemonchus contortus*. *Journal of Animal Science* **82**, 1602-1611.

Vervelde L, Bakker N, Kooyman NJ, Cornelissen WCA, Bank MC, Nyame AK, Cummings RD, van Die I (2003) Vaccination-induced protection of lambs against the parasitic nematode *Haemonchus contortus* correlates with high IgG antibody responses to the LDNF glycan antigen. *Glycobiology* **13**, 795-804.

Visscher PM, Woolliams JA, Smith D, Williams JL (2002) Estimation of pedigree errors in the UK dairy population using microsatellite markers and the impact on selection. *Journal of Diary Science* **85**, 2368-2375.

Vlassoff A, Bisset SA, McMurtry LW (1999) Faecal egg counts in Angora goats following natural or experimental challenge with nematode parasites: within-flock variability and repeatabilities. *Veterinary Parasitology* **84**, 113-123.

Vlassoff A, McKenna PB (1994) Nematode parasites of economic importance in sheep in New Zealand. *New Zealand Journal of Zoology* **21**, 1-8.

Walkden-Brown SW, Kahn LP (2002) Nutritional modulation of resistance and resilience to gastrointestinal nematode infection. *Asian-Australasian Journal of Animal Science* **15**, 912-924.

Walkden-Brown SW, Maslen AJ (2002) Specificity of antibody responses of goats to infections with three species of gastrointestinal nematode. *Proc. Aust. Soc. Anim. Prod.* **24**, 367.

Waruiru R, Ngotho J, Mutune M (2004) Effect of urea-molasses block supplementation on grazing weaner goats naturally infected with gastrointestinal nematodes. *Onderstepoort J Vet Res.* **71**, 285-289.

Watson TG, Baker RL, Harvey TG (1986) Genetic variation in resistance or tolerance to internal nematode parasites in strains of sheep at Rotomahana. In 'Proceedings of the New Zealand Society ofAnimal Production' pp. 23-26.

Watson TG, Hosking BC, Hurford AP (1992) Breed variation in expression of faecai nematode egg count. In 'Proceedings of the New Zealand Society of Animal Production' pp. 69-71.

Watson TG, Hosking BC, Hurford AP, Mather BC (1992) Developments in breeding Perendale sheep for resistance or susceptibility to internal nematode parasites. In 'Proceedings of the New Zealand Society of Animal Production' pp. 61-64.

Windon RG (1996) Genetic control of resistance to helminths in sheep. *Veterinary Immunology and Immunopathology* **54**, 245-254.

Windon RG, Dineen JK (1981) The effect of selection of both sire and dam on the response of F1 generation lambs to vaccination with irradiated *Trichostrongylus colubriformis* larvae. *International Journal for Parasitology* **11**, 11-18.

Windon RG, Dineen JK, Gregg P, Griffiths DA, Donald AD (1984) The role of thresholds in the response of lambs to vaccination with irradiated larvae *Trichostrongylus colubriformis* larvae. *International Journal for Parasitology* **14**, 423-428.

Woolaston RR, Barger IA, Piper LR (1990) Response to helminth infection of sheep selected for resistance to Haemonchus contortus. *Int J Parasitol* **20**, 1015-1018.

Woolaston RR, Eady SJ (1995) Australian research on genetic resistance on nematode parasites. In the breeding for reistance to infectious diseases in small ruminants (eds. Gray, G.D., Woolaston, R.R., and Eaton, B.T.). ACIAR, Canberra, Australia.

Woolaston RR, Manueli P, Eady SJ, Barger IA, Le Jambre LF, Banks DJD, Windon RG (1996) The value of circulating eosinophil count as a selection criterion for resistance of sheep to trichostrongyle parasites. *International Journal for Parasitology* **26** 123-126.

Woolaston RR, Piper LR (1996) Selection of Merino sheep for resistence to *Haemonchus contortus*: Genetic variation. *Animal Science* **62**, 451-460.

Woolaston RR, Singh R, Tabunakawai N, Le Jambre LF, Banks DJD, Barger IA (1992) Genetic and environmental influences on worm egg counts of goats in the humid tropics. *Proceedings of the Australian Association of Animal Breeding and Genetics*, pp. 147.

Woolaston RR, Windon RG (2001) Selection of sheep for response to *Trichostrongylus colubriformis* larvae: genetic parameters. *Animal Science* **73**, 41-48.

Woolaston RR, Windon RG, Gray GD (1991) Genetic variation in resistance to internal parasites in armidale experimental flocks. In 'Breeding for Disease Resistance in Sheep'. Melbourne. (Eds GD Gray, RR Woolaston) pp. 1-9. (Australian wool corporation).

Yalcin B (1982) Angora goat breeding. *Proceedings of the 3rd International Conference on Goat Production and Disease*, Tuczon, Arizona. pp. 269-278.

Zhou HM, Allain D, Li JQ, Zhang WG, Yu XC (2002) Genetic parameters of production traits of Inner Mongolia cashmere goats in China. *Journal of Animal Breeding and Genetics* **119**, 385-390.