CHAPTER ONE

GENERAL INTRODUCTION

Gastrointestinal parasites impose a substantial cost on the Australian sheep industry. The problem is by no means recent, as evident by a producer's submission to the Australian Pastoralist Review in 1892 which reads: *There are few or no subjects of more interest to the sheep owners of Australia than that of parasites of sheep* [and the investigation of their] *cause, proper treatment and, if possible, the prevention of worms in sheep* (quoted in Hall 1990).

In 1992, the Institute of Animal Production and Processing (IAPP) of the CSIRO developed a model to estimate the economic costs of major endemic sheep diseases in Australia (Collins 1992). Although the primary purpose of the model was to identify research and extension activities of CSIRO, it has been used to estimate the control costs and production losses associated with internal parasites.

A summary of the economic costs for worms in sheep in 1990-1991 is given in Table 1.1.

	DISEASE	CONTROL COST	PRODUCTION LOSS	TOTAL
Australia	Worms	92	245	337
	Flies	173	45	218
	Lice	120	194	314
	Footrot	63	18	81
NSW	Worms	37	85	122
	Flies	62	16	78
	Lice	43	99	142
	Footrot	43	11	54

Table 1.1 *Economic costs of some major endemic diseases in NSW and Australia for the year 1990-1991 ($m), from Collins (1992).*

McLeod, Collins, Barnes and Dobson (1992) used an economic model to estimate production losses from parasite burdens, using traditional and strategic control practices over a twenty year period, in the winter high rainfall area. Whilst this study is not directly transferable to the high summer rainfall area, the conclusions are valid for the area. These are that production gains from strategic control are mostly in weaner and lamb production. It was also concluded that strategic control could reduce the selection for resistance if correct management practices are adopted.

The introduction of Wormkill the strategic worm control program for the high summer rainfall area in 1980 (Dash, Hall and Love 1985) heralded a new era of internal parasite control on the New England Tablelands. This program was based on the epidemiological principals of Gordon (1948), but incorporated the use of Closantel, a long-acting anthelmintic specifically active against *Haemonchus contortus*. The feature

of the program was the reduction of anthelmintic treatments required to be given on an annual basis. Essentially, the high number of anthelmintic treatments used to control *H. contortus* prior to the introduction of Closantel had effectively controlled *Trichostrongylus spp.*, and with the reduction in these treatments *Trichostrongylus spp.* became an increasing problem (Holdsworth 1993).

Numerous field investigations by the author and other veterinary consultants working in the field with sheep flocks around this time indicated a major problem of illthrift in Merino weaners over their first winter period. The problem was in general due to nematode parasites associated with nutritional stress, or diseases considered to be secondary to these problems (Hall, Barger and O'Halloran 1990). This is reviewed in some detail in Chapter 2.

This research was initiated by a formal survey, discussed in Chapter 3, that identified nematode parasites and scouring (diarrhoea) as the major perceived problem, and that it was mostly a problem in young Merino sheep.

Sheep graziers, when discussing the results of this survey, indicated an enthusiasm for on-farm work to try to determine some of the factors underlying these problems, so that some practical solutions could be established. This led to the formation of trial flocks across the Tablelands in which weaner growth rates and parasite burdens were monitored, along with those epidemiological factors which were deemed relevant and practical to measure. On-farm trials are notorious for lack of scientific control and really only amount to on-farm observations, however, some trends emerged that encouraged further follow-up work. It also allowed some modifications to management programs by demonstrating the usefulness of providing adequate nutrition over the winter period for young sheep. These trials also demonstrated the advantages of providing 'clean' pastures (that is, pastures relatively free from parasite larvae) for young sheep in contrast to the traditional concept of using the same annual weaning paddocks, and the value of objectively monitoring as a gauge to performance and parasite control.

Some of these changes were incorporated as refinements to the "new" Wormkill program, an integrated version of the worm control program that was released in 1993 (Holdsworth 1993).

However, concern remained as to the ability of the program to control *Trichostrongylus spp.* in Merino weaners over their first winter, with clinical expression of diarrhoea, and poor growth leading to the development of a "tail" in the mob. This "tail" is a number of chronic illthrift animals of low body weight, poor condition, often with permanent diarrhoea that was not responsive to treatment. The new program, recognising this problem, actually introduced an extra anthelmintic treatment for weaners.

This problem was reported by Hall (1990), who speculated that this was due to the fact that Merino weaners failed to develop a satisfactory immunity to internal parasites during this period. It was suggested inadequate nutrition played an important role in compromising the development of early effective immunity to *Trichostrongylus spp.*

Work began in pen trials at this time to determine the effects of nutrition (van Houtert, Barger, Steel, Windon and Emery 1995b) in Merino weaners. Concurrently, the author began a number of field trials to determine what part nutritional supplements played in the development and control of parasites. Two field experiments, not reported here, were terminated. In one case, the drought was so severe that it was deemed pertinent to terminate the trial as it was effectively becoming an exercise in drought feeding. In a second one, sheep had to be relocated when dog attacks decimated sheep numbers, effectively terminating the trial.

Two field experiments are reported; the first (Chapter 5) was a field trial comparing short- and long-term nutritional affects, and the second (Chapter 6) compares nutritional treatments and genotypes in the short term on the development of parasite burdens in Merino weaners.

The implications of these results in terms of weaner management, parasite control strategies and the whole farm management practices are discussed in the General Conclusion (Chapter 7).

CHAPTER TWO

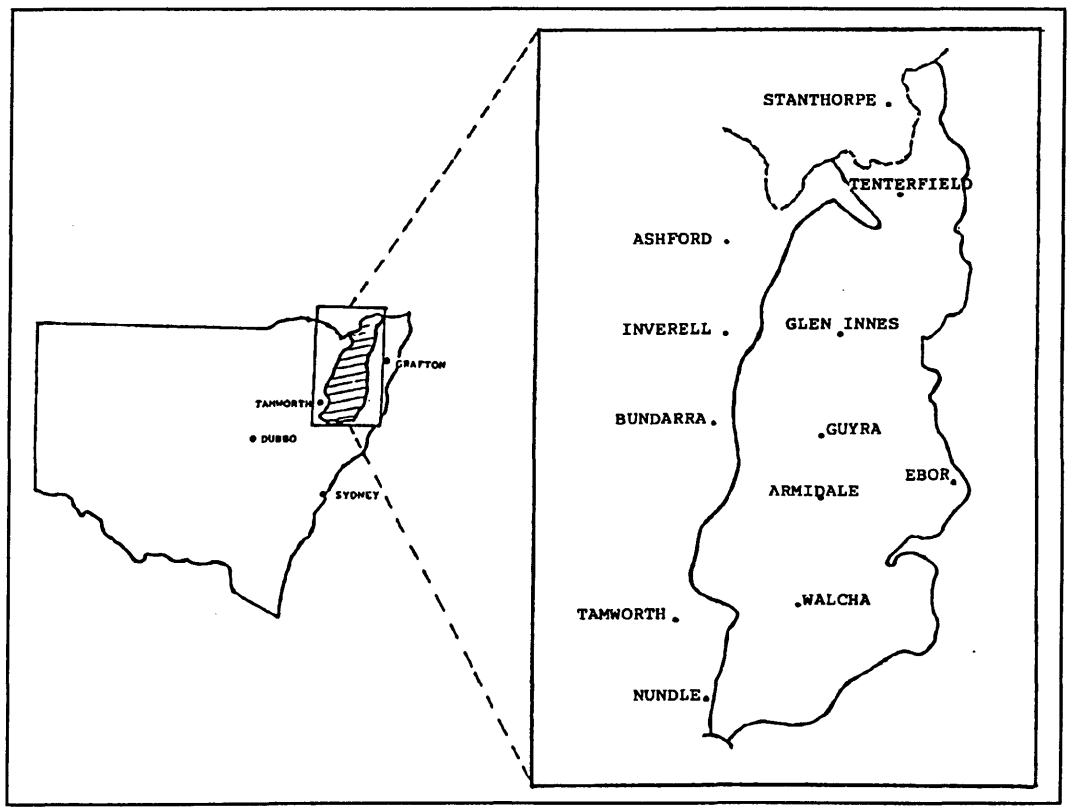
Literature review: nematode parasitism, diarrhoea and productivity in Merino weaners on the New England Tablelands.

2.1 INTRODUCTION

2.1.1 The New England Tablelands

The New England Tablelands (also known as the Northern Tablelands) is part of the NSW Tablelands complex, which ranges in altitude from 600 m to over 1500 m above sea level, extending along the Great Dividing Range.

The definition of New England Tablelands depends upon on the parameters used, but geographically, it is said to be that country in the area generally over 800 m above sea level. This extends from the Queensland Border in the north, southward along the Dividing Range until the high country ends in the Nundle region (McPhail 1977). This is illustrated in Map 2.1.



Map 2.1 *New England Tablelands.*

The climate is rather severe in winter, being cold and relatively dry, but persistent snow only occurs every 3 or 4 years. The rainfall is over 750 mm per annum (up to 1600 mm) and is a summer rainfall area. The majority of country is utilised for grazing and lends itself to pasture improvement.

A major portion of the country's super fine to fine best vegetable-free Merino wool (below 20 micron) is produced in the New England district centered around Walcha, Uralla, Armidale and Guyra. The quality of wool declines further north (towards Glen Innes) (D'Arcy 1972).

Annual Merino management programs on the New England Tablelands are planned to fit nutritional demands of livestock. Pasture production, being seasonal, is very defined (see Section 2.2), with the result that the annual breeding patterns are reasonably uniform, ie, ewes are joined in autumn, to lamb in spring.

Weaning has traditionally been carried out between 3 months to 6 months of age. This reflects the fact that excess feed is available over the summer and that weaning periods are not critical. In recent times, weaning at 12-14 weeks has been promoted (O'Halloran 1990). Merino "weaners" are defined as Merino lambs from weaning until two-tooth stage (approximately 12-14 months) when they become "hoggets".

2.1.2 Wormkill

The nematode parasite problems on the New England have traditionally been associated with *H. contortus* and control programs were based around controlling this parasite. This position was exacerbated in the 1950s with the introduction of extensive pasture improvement, which was made possible by the high usage of superphosphate fertilisers. This in turn lead to substantially higher stocking rates, changed management practices from running adult sheep only to complete Merino breeding enterprises, which had hither to been restricted by poor quality pasture production. Essentially, young sheep were sent "out west" to grow up (F L Clark, *pers comm.*)

The introduction in the 1960s of the modern broadspectrum anthelmintics, initially thiabendazole and subsequently levamisole (both of which at the time of introduction were very efficient against all major species of strongylids), lead to the effective chemical control of all nematodes including *H. contortus*.

These drenches were, however, used frequently (approximately 7 treatments for ewes and 8 for lambs per year) (Newman, Parton and Hardaker 1987) and resistance was widespread by 1984 (Love, Johns and Coverdale 1991). Concern for this developing resistance and the release of Closantel, a long-acting anthelmintic specifically active against *H. contortus*, allowed the formation of a strategic drench program based on epidemiological principles by Gordon (1948). Following field trials (Dash 1986), the WORMKILL drench program was launched in 1984 (Dash *et al.* 1985) (see Figure 2.1). One major feature of the program was the reduction in drenches given to sheep per year.

WORMKILL					Grazing Management	Added fluke control (all sheep)
Date	All sheep & Hoggets		Lambs & Weaners			
	Seponver	Broad-spectrum	Seponver	Broad-spectrum		
1st August	*	*				
1st November	*	*	*	*	Move ewes & lambs to low-worm pastures	
1st February	*		*	*	Move lambs to low-worm pastures	
1st April				*		*

Figure 2.1 The initial Wormkill (launched in 1984) - the strategic drench program for the New England Tablelands.

Despite the introduction of Ivermectin, a member of a third group of broadspectrum drenches (which was, and still is, highly effective), major problems developed with the program in the 1990s with a succession of wet mild autumn/winters. The most obvious was the field clinical signs of diarrhoea and illthrift that developed in Merino weaners due to trichostrongylosis.

The program was redefined as an integrated worm control program and incorporated the following changes (Holdsworth 1993):

(1) grazing management and monitoring (by faecal egg counts) were emphasised ahead of drenching

(2) lambs were to be weaned at 12-14 weeks and drenched then moved to "low-worm" pastures (whereas previously drenching was independent of weaning)

(3) the drench and move process was to be repeated 2 months later; this effectively allowed weaners to make good growth in worm-free conditions, while still relatively susceptible to trichostrongylosis

(4) regular monitoring of weaners, especially during the winter period, was further recommended.

WORMKILL Integrated Worm Control Program for Sheep on the New England Tablelands						
Date *	Monitoring - Grazing Management	Adult Sheep & Hoggets		Lambs/Weaners		Added Fluke Control (all sheep)
		Closantel	Effective Broadspectrum	Closantel	Effective Broadspectrum	
Late August	PRELAMBING WORMTEST (Ewes) + Prepare 1st LOW-WORM (weaner) pasture +		*			*
14th September	LAMBING					
October	MARKING	*		*		
November	Spell 1st LOW-WORM (weaner) pasture Prepare 2nd LOW-WORM (weaner pasture)					
22nd December	WEANING Drench and move weaners to 1st LOW-WORM pasture	*	*	*	*	
22nd February	Drench and move weaners to 2nd LOW-WORM pasture	*		*	*	
Late March/ April	WORMTEST to monitor program (all classes of sheep, eg, ewes, wethers, weaners)					
April/May	Drench and move weaners again if possible				*	*
June/July	WORMTEST (weaners)					

* - If these dates don't fit in with your management speak to your Veterinary Advisor
\+ - See over for pasture preparation hints
* - Most adult sheep (esp. wethers) will not require drenching at this time. WORMTEST results should be used to determine the necessity

Figure 2.2 *The current version of Wormkill - an integrated worm control program for the New England Tablelands.*

One major problem still confronting the grazier is the occasional failure of the program to adequately control *Trichostrongylus spp.* in Merino weaners over their first winter with the clinical expression of diarrhoea and anaemia exacerbated by inappetence, leading to chronic illthrift. This was recognised by Hall (1990), who speculated that this is due to Merino weaners failing to develop satisfactory immunity during this period, and that inadequate nutrition may play an important role in compromising the development of early effective immunity to *Trichostrongylus spp.*

2.2 MERINO WEANER GROWTH RATES

In contrast to the peri-natal and post-natal periods, very little field work has been carried out to determine growth rates (body weight for age) in Merino weaners in the New England area, although it is said to be poor in comparison with other areas (Fowler, May and Wilkins 1980).

A major factor limiting weaner growth is the lack of pasture availability in winter. This is demonstrated by the seasonal pasture growth curve in Figure 2.3 (Hilder 1956, Vickery 1972).

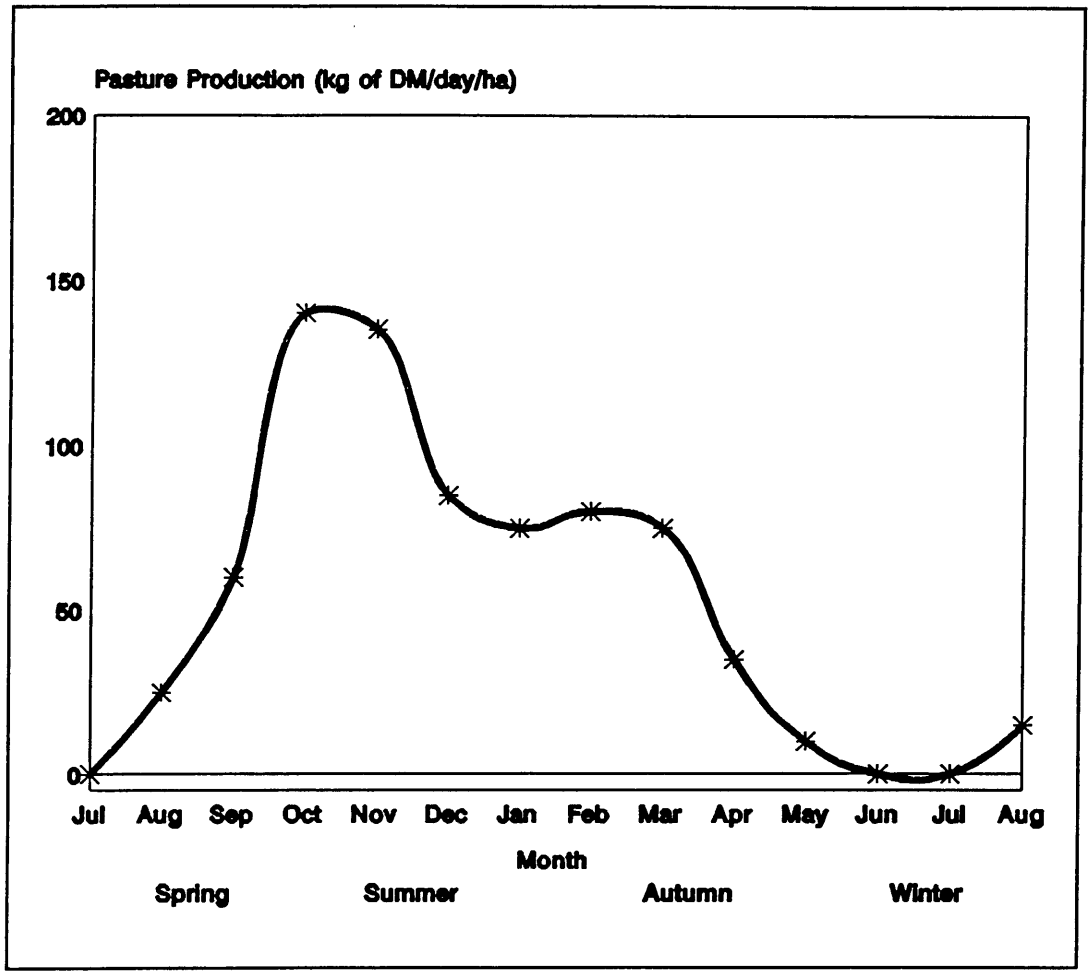


Figure 2.3 *An annual average pasture production curve for Armidale (adapted from Hilder 1956, Vickery 1972).*

An average of measured body weights and growth rates in Merino weaners summarised from Hamilton, Hutchinson and Swain (1970); Wilkins, Kilgour, Gleeson, Cox, Geedes and Simpson (1982) and Macfarlane (1993) is shown in Figure 2.4.

These figures demonstrate two problems: (1) Merino weaners only reach a weight sufficient to ensure survival over winter, and (2) weight loss (ie, negative daily growth rate) occurs in the period when pasture growth is negligible.

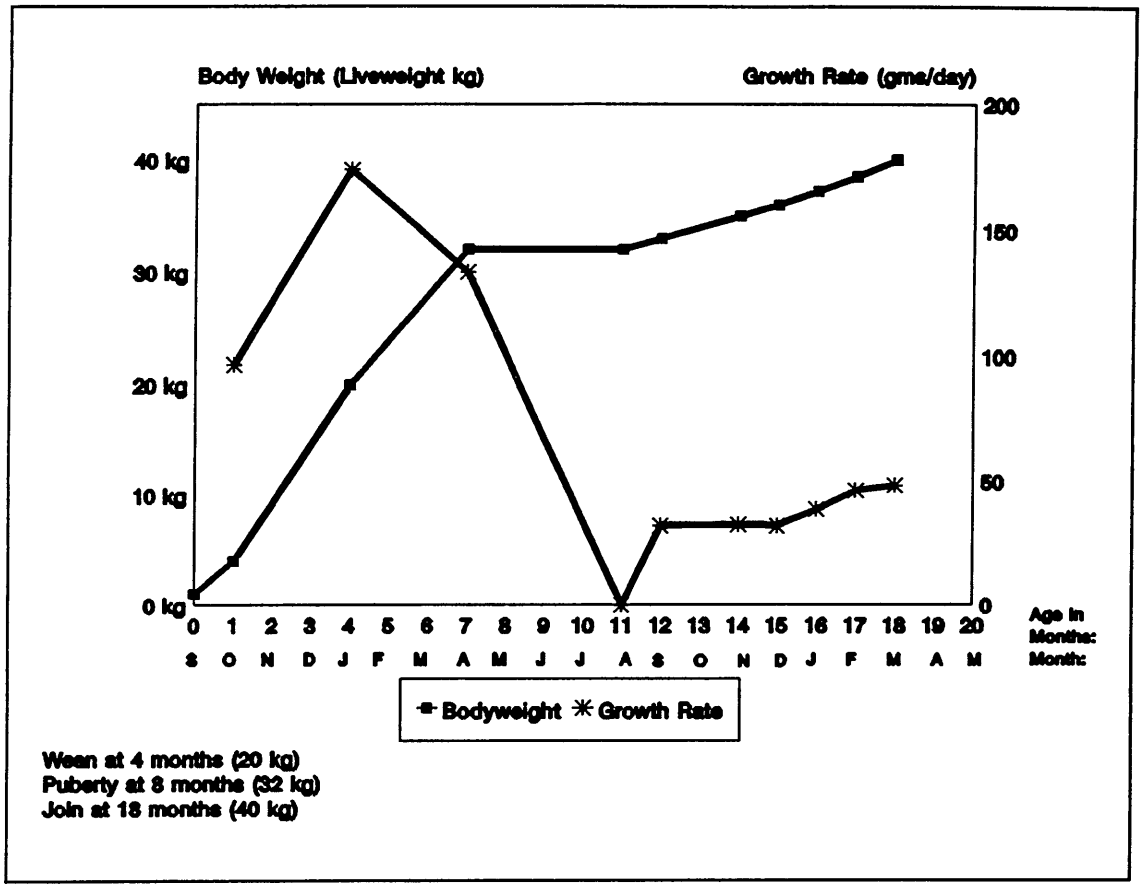


Figure 2.4 *Average body weights and daily growth rates of Merino weaners on the New England Tablelands (adapted from Hamilton et al. 1970; Wilkins et al. 1982 and Macfarlane 1993).*

In contrast, Figure 2.5 shows average target body weights for age considered as a minimum that will allow for maximum long-term production for Merino weaners (18 micron type) and required daily growth rates (adapted from Farquharson 1989, O'Halloran 1990 and Lollback 1992).

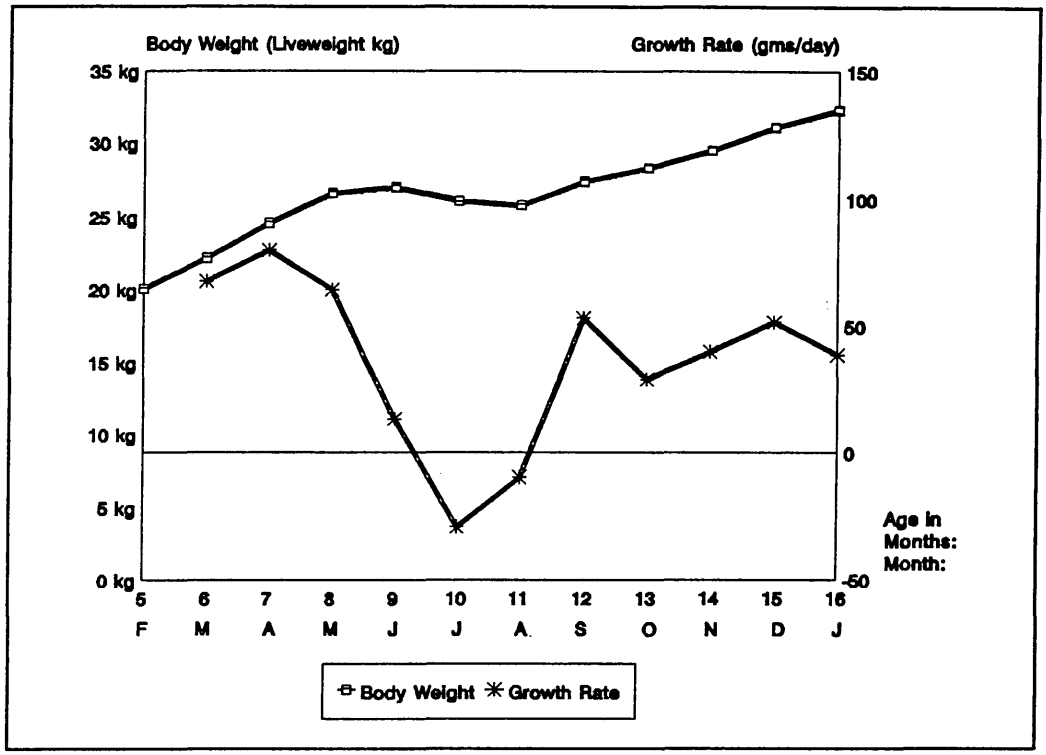


Figure 2.5 *Recommended average minimum body weights and daily growth rates for Merino weaners on the New England Tablelands (adapted from Farquharson 1989, O'Halloran 1990 and Lollback 1992).*

The difference between actual body weights and growth and recommended minimum standards is substantial over the winter period. The effect of this difference in terms of the effect and control of nematodes will be discussed later.

2.3 NEMATODE PARASITES

2.3.1 The Parasite Species

The major parasites causing reduction in growth and diarrhoea in Merino weaners on the New England Tablelands are the gastrointestinal nematodes. As outlined previously, the introduction of the Wormkill program gave good control of *H. contortus,* although in recent times, anthelmintic resistance is making this more difficult (Love, Lloyd and Davis 1998; Chick, Woodgate and Wooster 1998). Neither *H. contortus* or F. *hepatica* (liver fluke) will be considered further since both are relatively well-controlled by existing methods and do not belong to the diarrhoea-inducing nematodes, the main focus of this thesis.

Trichostrongylus spp. of the small intestine are the most important group whose harmful effects mainly manifest in weaners. The most common species is *T. colubriformis*, but *T. vitrinus* and *T. rugatus* also occur (Gordon 1948). Although *T. axei* of the abomasum may contribute to harmful effects in field infections, it has not been implicated as a principal species causing pathogenic infections (Seddon and Albiston 1967).

Ostertagia spp., mainly *O. circumcincta,* but including *O. trifurcata* and *O. davtiani,* are commonly found in field infections, but their pathogenic importance is difficult to quantify (Donald, Morley, Waller, Axelsen and Donnelly 1978). *Nematodirus spp.* (*N. spathiger* and *N. filicollis*) are often present in low levels in adults and are occasionally identified as pathogenic in Merino weaners (Seddon and Albiston 1967). Prior to the introduction of effective broadspectrum anthelmintics, *Oesophagostomum columbianum* (nodule worm) was reported to be a major problem, especially over the winter period when nutritional conditions were poor (Gordon 1942). Since that time, it's coincidence has fallen markedly (Anderson *et al.* 1978) and the incidence of *Oe. venlosum* has increased (Cole 1986).

2.3.2 Life Cycles

Life cycles of nematode parasites are discussed in detail by Soulsby (1982), with summaries in Donald *et al.* (1978) and Cole (1986). A diagram of the life cycle for nematodes is given in Figure 2.6.

Ostertagia and *Trichostrongylus spp.* have similar life cycles and will be discussed together. The life cycle is a simple direct life cycle, with reproduction confined to the adult parasitic stage. Each worm present in a sheep is separately acquired as an infective larva.

Sexually mature female nematodes produce large numbers of eggs which pass out in faeces of the host, but usually a few survive to infect the same or other sheep. Free-living development on pastures enables the parasite to infect new hosts and is the mechanism allowing dispersal and exchange of parasite populations between sheep. The speed and success of this development is determined by the pasture microclimate, principally temperature and the availability of moisture.

Providing environmental conditions are favourable, with respect to moisture and temperature, eggs develop rapidly to the unhatched larval stage. *Ostertagia* and *Trichostrongylosis spp.* can survive desiccation and moderately high temperatures as unhatched larvae. Under suitable conditions, eggs hatch to first stage larvae in 4-6 days. These feed on bacteria in the faeces, grow and moult to second stage larvae which similarly feed, grow and moult to form third stage or infective larvae. However, the second moult is incomplete and the infective larva remains enclosed in the old second stage sheath which prevents it from feeding. Thus, its survival, in the external environment, is dependent on the rate at which its stored nutrients are used. Infective larvae usually migrate up the pasture herbage through external water films, where they become available for ingestion by sheep.

When an infective larva is ingested, the second stage cuticle is shed. The stimulus to exsheath normally occurs in the organ immediately anterior to the site of infection, ie, abomasal parasites exsheath in the rumen and small intestinal parasites in the abomasum. Parasitic third stage larvae either enter, or become intimately associated with, the gut mucosa where they undergo the first parasitic moult to the fourth larval stage. In these species, especially *Ostertagia spp.*, larvae may sometimes be arrested in their development, commonly in the early fourth stage, and remain dormant within or on the surface of the mucosa for up to several months before some may resume

development. When development proceeds, sexual differentiation begins during the fourth stage and is completed after the final moult to adult male and female worms. These then mature, mate and the females begin egg production.

If development is not interrupted or delayed, the minimum period from ingestion of infective larvae to the production of eggs by females is about 3 weeks for these species.

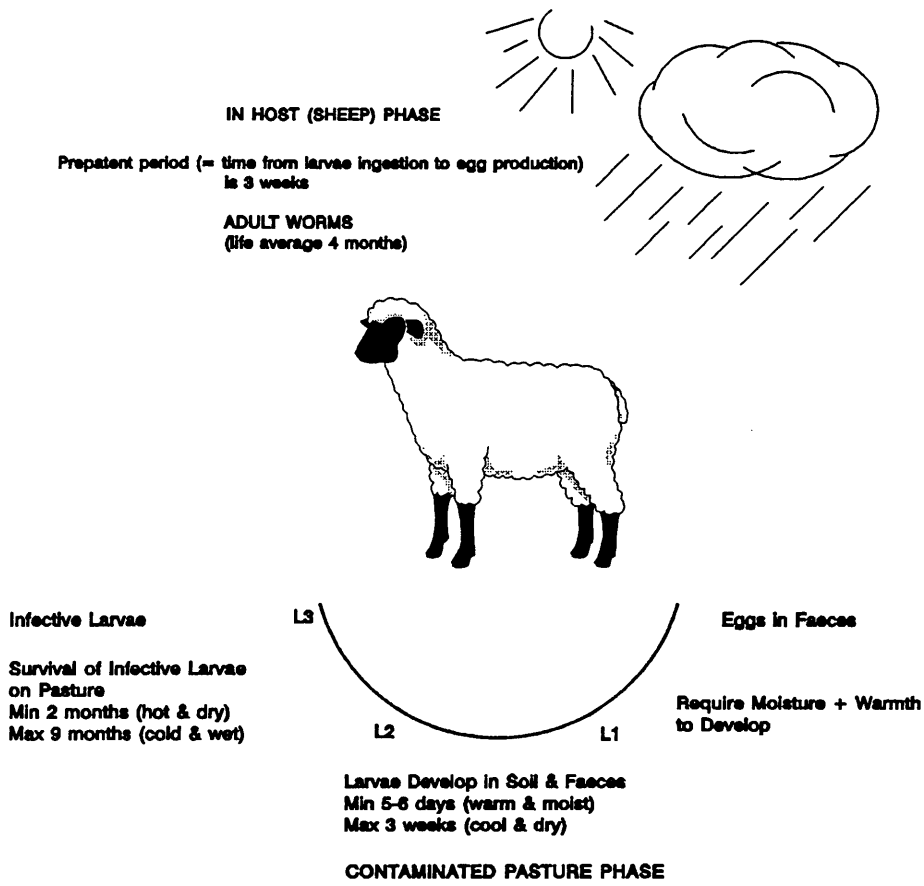


Figure 2.6 Life cycle diagram for nematodes (Donald et al. 1978).

2.3.3 Epidemiology

The epidemiology of parasite infections is determined in the first instance by the availability of free-living larvae to initiate infections in the grazing animal. The effect of the larvae on the host and parasite-host interactions are covered in the following sections.

The basic epidemiological factors were established by Gordon (1948) and quantified in later work (Gordon 1950). In this work, faecal egg counts were used to estimate the seasonal incidence of adult worm burdens and pasture contamination. This led to the development of the bioclimatograph to predict the variation in pasture larvae availability relative to specific seasonal weather patterns.

Subsequent studies on the development and survival of the free-living stages (reviewed by Anderson, Dash, Donald, Southcott and Waller 1978) has lead to a good understanding of the factors affecting the availability of infective larvae on pasture at any one time. Critical epidemiological factors include the level of pasture contamination with worm eggs, and the subsequent level of infective larval availability over given periods of time.

Temperature and available moisture are the important components of the micro-environment affecting larval numbers on pasture. Development and migration of larvae are more susceptible to variations in these factors compared to variations affecting survival of existing populations (Donald 1968). Thus, development and migration are likely to proceed at different rates compared to death rates and rates of removal by stock, so that numbers of available larvae may continue to increase for several weeks after sheep have been removed from pasture (Donald 1967).

On the New England Tablelands, autumn is commonly a favourable period for the survival and development of freshly deposited eggs as a result of moderate temperatures and evaporation rates. Although development and migration are slowed by falling temperatures, especially if winters are dry and frosty, infective larvae on herbage die only slowly and substantial numbers can persist until late spring (Donald 1968; Southcott, Major and Barger 1976). A relatively small proportion of the eggs deposited in winter survive to yield infective larvae and development is slow (Donald 1968). As a result, most larvae present on herbage in winter and spring are generally derived from autumn contamination (Southcott *et al.* 1976; Donald *et al.* 1978).

Spring is usually favourable for larval development and migration. With rising temperatures, larvae may reach the herbage simultaneously in mid to late spring from eggs deposited earlier in the season (Donald and Waller 1973).

The fate of summer contamination depends heavily on rainfall. When rainfall is high, maximum numbers of infective larvae are present on the herbage for one to two months after rain. Effectively, this means that from early summer (December) onwards, larval numbers rise during the rest of summer, then decline so that they are low by the end of autumn.

If summer rainfall is low, high temperatures and evaporation rates largely prevent development of eggs to infective larvae and very few larvae are available for ingestion during a dry summer (Anderson 1972, 1973). Some eggs of *Ostertagia* and *Trichostrongylus spp.* deposited in a dry summer are able to survive in faeces and may subsequently develop into infective larvae following rain in autumn (Anderson, Barger and Waller 1987).

Larval availability for a typical New England summer is demonstrated in Figure 2.7.

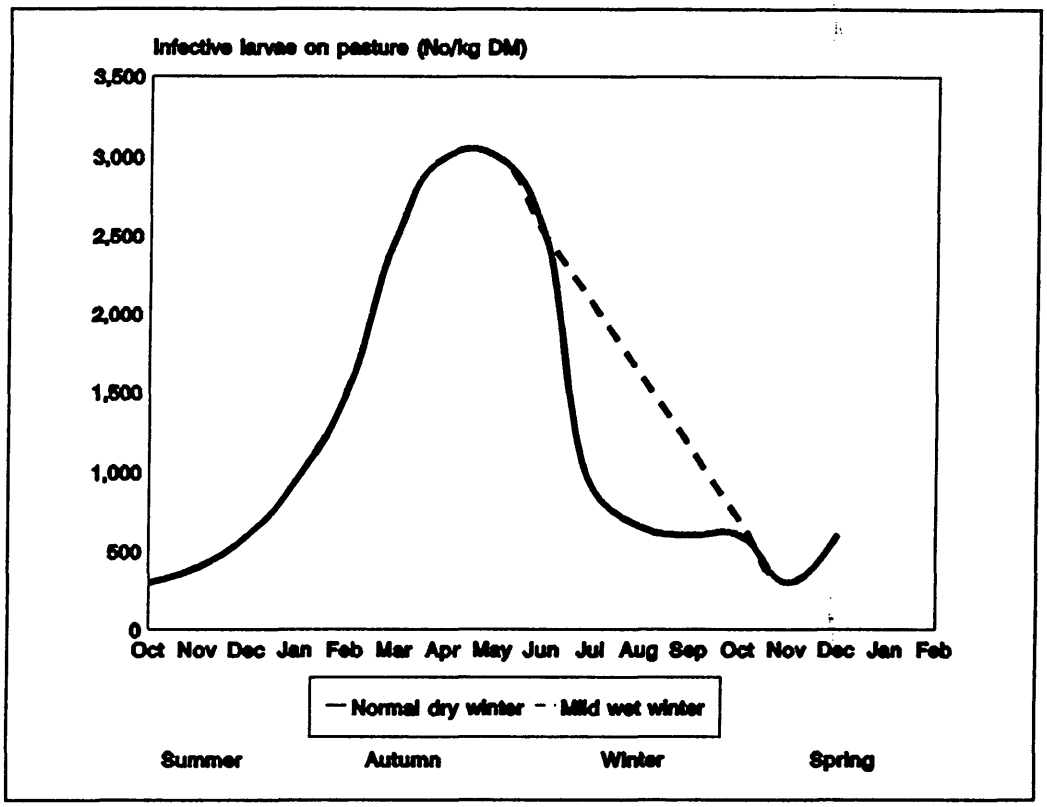


Figure 2.7 *Typical seasonal patterns of infective larval availability in New England Tablelands for Trichostrongylus spp. (from Waller, Anderson and Barger 1993).*

The worm burden that develops in Merino lambs from birth until 18 months is a result of this larval availability. Merino lambs have no infestation at birth, and nematodes are only very slowly accumulated while milk is the major source of lamb's food. Ingestion of infective larvae increases with increased pasture consumption, and is compounded with increasing larval availability.

The pattern of infection from birth to 18 months is shown in Figure 2.8.

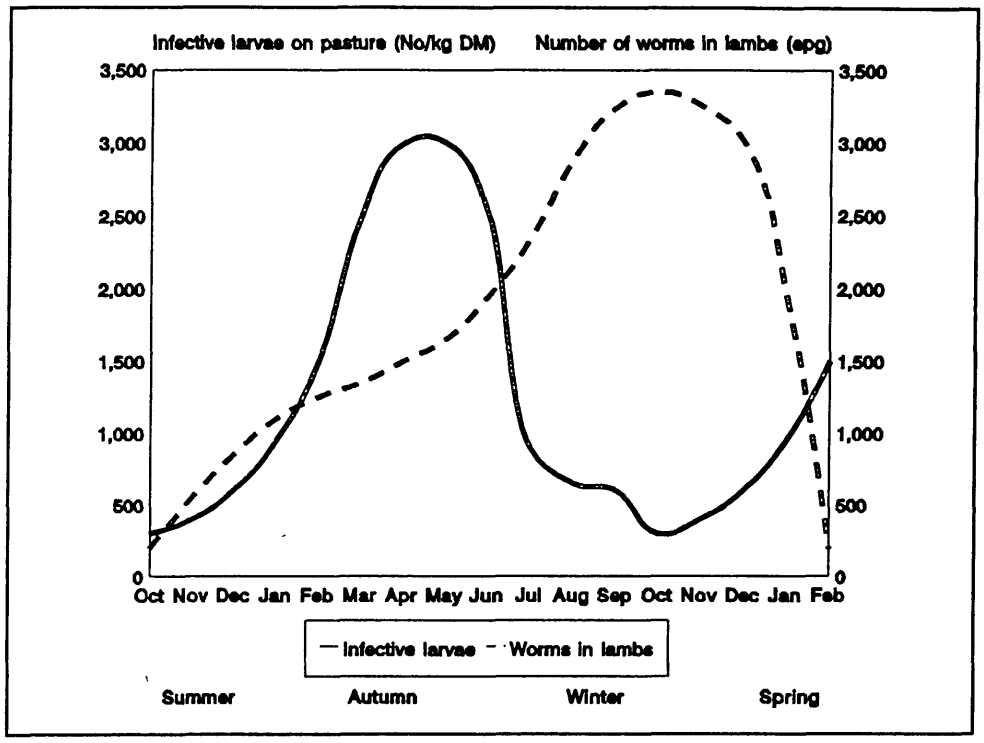


Figure 2.8 *Worm burden development in Merino lambs/weaners from birth to 18 months grazing contaminated pastures in Armidale (adapted from Southcott et al. 1976; Anderson et al. 1987 and Waller et al. 1993).*

2.4 THE SHEEP - NEMATODE PARASITE INTERACTION - IMMUNITY

2.4.1 The Mechanisms and Manifestations of Immunity

Adult sheep, unless stressed by pregnancy/lactation, poor nutrition or other factors develop an effective immunity to *Trichostrongylus spp.*

In a classical study on the manifestations of immunity to *N. spathiger* in young Merino sheep, Donald, Dineen, Turner and Wagland (1964) concluded that immunological responsiveness is expressed by:

(1) failure to establish at infective third larval stage
(2) arrested development at the fourth larval stage
(3) reduction in egg production
(4) expulsion of adult worms
(5) discrimination against the female in (2) and (4).

A similar immune response occurs with *Trichostrongylus spp.* infection. The characteristic phases of immunity against *T. colubriformis* are shown in Figure 2.9, summarised from Dobson, Waller and Donald (1990a) by Emery (1991).

Host immunity against *T. colubriformis* may develop as soon as 5 weeks after continuous infective larval intake. As a result, the proportion of ingested larvae which establish and develop in the host is depressed from around 80% to less than 5%, and some may be retarded in development. From 8 weeks onwards the egg output in faeces declines, and from 12 weeks onwards, established adult worms are rejected and expelled (Dobson *et al.* 1990a). However, this time frame will vary depending on factors discussed later.

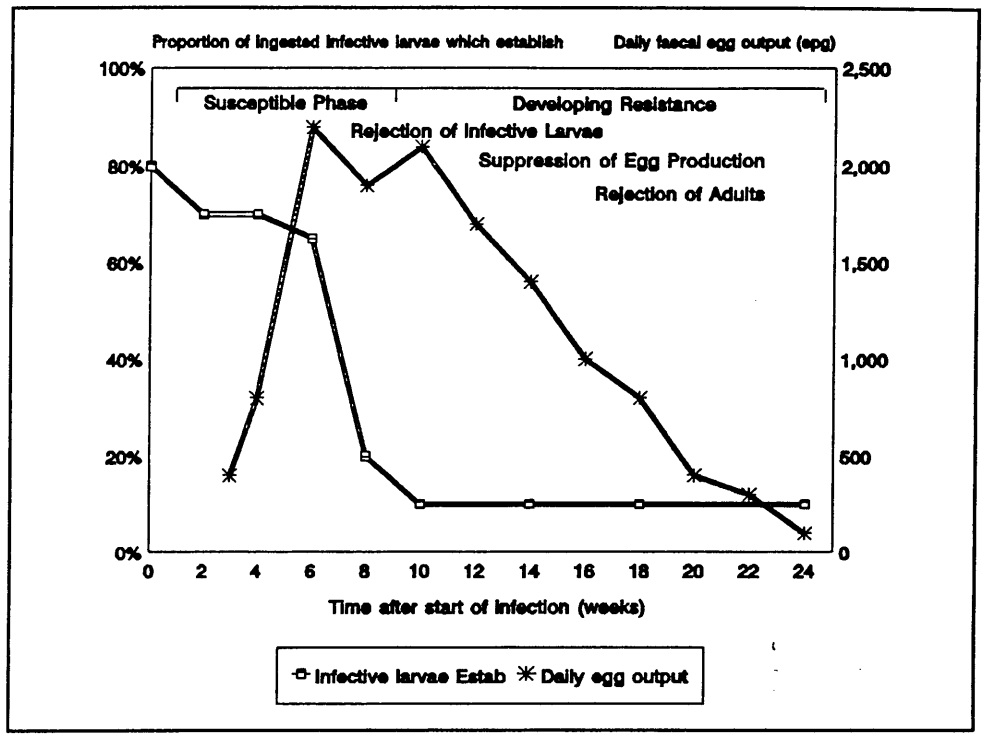


Figure 2.9 Development of resistance to T. colubriformis in lambs infected daily with 2000 larvae (Emery 1991).

Mechanisms of immunity to nematode parasites have been studied in depth. Nevertheless, many aspects are not fully understood. The basic mechanisms are relatively complex which is compounded by the complexity of the nematode parasites compared with more simple organisms (eg, bacteria). The immune system is primed by initial exposure to the ingested parasite larvae. Following this, the sheep are immunised against the parasite upon further ingestion of the parasite larvae. The development of immunity can be divided into two phases. The first phase (which is a specific phase) occurs when parasite components from the ingested parasite larvae are absorbed from the gut and reactivate the primed immune system. This is followed by the second (non-specific) phase in which the immune reaction attracts and activates non-lymphoid cells (eg, mast cells, basophils, eosinophils) in the gut lining and causes the release of chemical mediators into the gut lumen. These mediators paralyse or disorientate not only the parasite involved in initiating the immunity, but a range of nematode worms which may result in their expulsion. The cells involved in these steps have been deduced from extensive studies of the sequence of the process, including gut pathology, physiology and biochemistry (Emery 1991). Known aspects are discussed in reviews by Rothwell (1989) and Miller (1990).

The measurement of the degree of immunity is complex depending on which expression of immunity is used as a measure, as well as the type of challenge mounted. This makes comparisons of various studies difficult and results may appear to differ. Animals are usually either challenged with infective larvae by a large single dose or a smaller trickle dose over time. Subsequent measurements are made of either egg output or establishment rates at various times. In the latter case, developed and/or arrested larvae are measured by worm counts after slaughter of the host.

The development of immunity is dependent upon a number of factors. There must be an initial ingestion of larvae of sufficient numbers to induce the priming of the immune system. This must be followed by a dose of ingested larvae sufficient to induce a full immune response (Dineen, Gregg and Lascelles 1978).

In young sheep, resistance to larval establishment and rejection of adults developed with high level of larval intake, but not with low level of larval intake (Waller and Thomas 1981).

2.4.2 Effect of Age on Immunity

Full expression of immunity requires that the animal has developed a fully functional immune system. This development of immunity in sheep is affected by age (eg, Gibson and Parfitt 1972; Colditz, Watson, Gray and Eady 1996). It has been shown that Merino sheep under 5 months fail to amount an immune response and that sheep up to 15 months may respond poorly and/or slower than adult animals (Dobson *et al.* 1990a).

The reasons for this slow response remains unclear, but suggestions given by Dineen *et al.* (1978) remain unchallenged. These suggestions are that one or more of the following occur:

(1) induction of specific immunological tolerance in the immature animal
(2) a higher level of antigenic information is required to provoke a reaction in the immature animal
(3) inhibition of an active immune response by passively acquired maternal antibodies
(4) incapacity of the immature animal to generate certain immunologically non-specific mediators of inflammatory reactions which may be involved in the non-specific phase of parasite immunity.

On the last point, it is postulated that the non-specific phase of immunity is different in lambs than older sheep and that a component of this phase is deficient or reacts differently in lambs so that this phase is rendered ineffective (Emery 1991). Colditz *et al.* (1996) demonstrated that young sheep invariably mount smaller antibody responses than do mature animals. It is important to note that not only immune responses but also inflammatory responses are of a lower magnitude in immature animals (Watson and Gill 1991).

The age at which Merinos develop immunity to *T. colubriformis* infections has been examined by Dobson, Waller and Donald (1990b). Naive (ie, pen-fed) Merino lambs were subjected to trickle larvae infection from the ages of 12, 20, 28 and 36 weeks of age, and the development of immunity was measured as the number of larvae that developed to adults and the number that established but became arrested. Pasture grazed sheep were similarly tested using contaminated paddocks as the infection source.

In these trials, 12 week-old sheep took 9 weeks to develop immunity, whereas 36 week-old sheep took 5 weeks. The general conclusions were that:

(1) a threshold worm burden must be exceeded before any effective resistance develops
(2) once the threshold is exceeded, the rate of development of resistance is independent of infection rate but determined by host age
(3) arrested development is induced by the host immune response
(4) acquired immunity similar to that observed in pen experiments also develops naturally under field conditions
(5) as immunity develops, large within-group variability occurs.

Coop, Huntley and Smith (1995) suggested that this may be because there are competing demands for available nutrients in parasitised ruminants between growth, repair of gastrointestinal pathology and the immune response. They proposed that in the growing lamb, there may be a bias towards growth and repair at the expense of development of immunity, and were able to test this hypothesis by giving by-pass protein (sodium caseinate by abomasal catheter) to lambs infected with *O. circumcincta* and monitoring the subsequent development of immunity. Animals given the by-pass protein exhibited lower faecal egg counts and lower worm burdens after challenge, a smaller proportion of which had developed beyond the L4 stage. These animals also had higher concentrations of mucosal mast cells, as measured by local concentrations of their specific protease, suggesting that the above effects were due to an enhanced immune response because these cells are associated with expulsion of gastrointestinal nematodes (Miller 1990). The supplemented lambs were, however, slightly less efficient at using dietary protein for growth though possibly more efficient at directing the additional protein into the development of an effective immune response. Bown, Poppi and Sykes (1991) suggested that the phenomenon of age-dependent immunity to *O. circumcincta* is due to a relative protein deficiency in the young growing lamb, particularly in view of the results of a similar experiment with *T. colubriformis*, which demonstrated that the effect of casein supplementation was due to its protein content rather than calorific value.

Parkins and Holmes (1989) suggest that with the onset of puberty, body weight and condition may become more important than chronological age in affecting the development of the immune system. Merinos are capable of developing full immunity by the age of 9 months and it is suggested that from this age the nutritional status becomes the main factor affecting expression of immunity. This is probably because significant nutritional resources are required to mount the local and systemic immune responses necessary to ensure full expression of the immune response. The exact mechanisms by which lack of nutrition affects the development and expression of immunity are not yet understood.

2.4.3 Effect of Nutrition on Immunity

Nutritional status of sheep influences both the rate of development of host immunity to nematodes (Langland *et al.* 1984), as well as the subsequent maintenance of that immunity (Stewart and Gordon 1953). In general, the evidence demonstrates that malnutrition results in depressed general immunological competence and hence a reduced ability to combat infection (reviewed by Wakelin 1989). It has long been recognised that a high level of nutrition may reduce the number of parasites that become established as well as reduce the effects of those parasites which do become established (Gibson 1963). Steel (1978) found that in a single primary infection, the number of larvae that become established in a sheep on a high plane nutrition were reduced, more worms were eliminated, larval development was arrested to a greater degree, egg output occurred later, and fewer eggs were produced per female worm when compared to similar sheep on a low plane of nutrition.

The effect of nutrition on the maintenance of immunity to nematodes by sheep is still to be clarified. Clunies-Ross and Gordon (1933) found that sheep infected with *H. contortus* lost their immunity after a prolonged period on a low plane of nutrition. It has been shown that sheep maintained on a high plane of nutrition are more resistant to subsequent infection of nematodes (especially obvious with *Trichostrongylus spp.* (Brunsdon 1964). Recently, van Houtert *et al.* (1995c) have shown that Merino wethers maintained immunity to incoming larvae of *T. colubriformis* on a low plane of nutrition (sheep lost 15% body weight over 60 days). Clearly, much work is required to determine the extent and length of nutrition change that influences immunity (both to existing infections as well as incoming larvae) for the various species of nematodes.

2.4.4 Effect of Stress on Immunity

Adult sheep that have developed a fully functional immune system may have that immune system compromised by a number of stress factors. One natural "stress" factor is that of late pregnancy and subsequent lactation which exerts a profound effect on the immunity of the host to nematode parasites (Arundel and Ford 1969; O'Sullivan and Donald 1970; Southcott, George and Lewis 1972). This is not considered further here.

2.5 THE SHEEP - NEMATODE PARASITE INTERACTION - DISEASE AND PRODUCTION EFFECTS

2.5.1 Clinical and Pathological Effects

Trichostrongylus spp. cause clinical disease characterised by anaemia, inappetence, diarrhoea (black scour), illthrift and death. Subclinical disease affects productivity.

Both the pathology and pathophysiology have been examined to explain the pathogenesis of the disease processes. The pathology of diseases caused by nematode infections has been studied extensively and has been reviewed by Jubb, Kennedy and Palmer (1985). More recently studies have attempted to correlate pathological effects with physiological effects of infection.

The pathology of *Trichostrongylus spp.* is outlined as an example of the pathological effect of parasitic infection. Ingested third stage larvae exsheath in the acid abomasal environment and preferentially establish in the proximal 5 to 6 m of the small intestine in sheep. The larvae in the intestine enter the mucosa, mainly at the base of villi, and they persist throughout their life at least partially embedded in the epithelium. Usually infective larvae all develop into adult worms (over about 2 weeks), with a prepatent period of about 16 to 18 days.

Sheep that are clinically affected by trichostrongylosis are cachectic and dehydrated. Dark green, diarrhoea faeces will adhere to the skin or wool of the escutcheon or breech. There may be serous atrophy of internal fat depots, and marked atrophy of skeletal muscle. There may be oedema of the mesentery and some serous effusion into the body cavities, associated with hypoproteinemia, if dehydration is not severe. Mesenteric lymph nodes are enlarged and juicy. The intestines are flaccid, and the small bowel contains thin, fluid, green faeces, while duodenal contents may appear somewhat mucoid. The large intestine may contain similar fluid or pasty green faeces, which are usually foul-smelling.

The duodenal mucosa of terminally ill animals have patchy or diffuse areas of atrophy of villi, and fine white or translucent, threadlike worms, about 5-8 mm long, entwined on the mucosal surface. These are found in the proximal third of the small intestine (about 5-6 m) in severe clinical infections.

Experimental infections of intestinal *Trichostrongylus spp.* studied indicate that the pathology and pathogenesis of disease caused by them is similar (Coop, Angus and Sykes 1979). Villus atrophy occurs in areas of intestine populated heavily by the worms, and the severity of the lesion within individual animals is correlated with the local density of the worms. Lesions are characterised microscopically by villus atrophy, which may vary considerably in severity, in association with elongate, dilated, often straight crypts. In animals with severe atrophy, the surface epithelium is flattened between the openings of crypts, and erosions of the mucosa may be evident, from which inflammatory cells and tissue fluid effuse (Barker 1975a).

The lamnia propria in the affected area of intestine is populated by a moderately heavy mixed inflammatory cell population. Lymphocytes and plasma cells are prominent between crypts, with an admixture of eosinophils. In the superficial lamina propria, neutrophils often accumulate beneath the epithelium, and in areas of erosion or previous erosion, there may be a thin, traversely oriented layer of connective tissue. No specific attraction of inflammatory cells is evident to worms in tunnels in the surface epithelium. Abnormal permeability of the endothelium of capillaries of heavily infected mucosa has been demonstrated, and oedema of the lamina propria may be evident in these areas (Barker 1975b).

The mechanism by which villus atrophy occurs following infections with *T. colubriformis* has been studied in rabbits by Hoste and associates (Hoste 1989; Hoste, Kerboeuf and Parodi 1988; Hoste and Mariana 1989). In non-parasitised animals, the normal intestinal epithelium is maintained by a balance between the production of new cells in the glands of Lieberkühn and the loss of enterocytes occurring at the end of the migration of cells to the tips of the villi. The time necessary

for cells to reach the tip of the villi gives an estimate of cell life span, and any changes in cell transit time indicates a variation of cell loss rate. The striking reduction in cell transit time in *T. colubriformis* parasitised small intestine indicates enhanced cell loss on the villi.

Hoste and Mariana (1989) summarise that the presence of villus shortening, which are directly associated with increased crypt cell production rates (Hoste *et al.* 1988), is directly related to parasitic damage to villi, which in turn is directly related to the degree of infection; that is, the changes in cell turnover underlie the structural and enzymological features in *T. colubriformis* infected rabbits. This mechanism has yet to be confirmed as similar in sheep.

2.5.2 Anorexia

A most significant effect of parasitism on the host is depression in food intake. The degree of inappetence is dependent on the numbers of larvae ingested and the parasite species (Steel, Symons and Jones 1980). Level of infection affects anorexia. Steel *et al.* (1980) demonstrated that infection levels below 3000 infective *Trichostronglus spp.* larvae per week failed to reduce feed consumption when compared to uninfected controls, but high infection levels produced progressively greater reductions. Age and resistance status of the host sheep also influence the degree of anorexia with younger animals of low immune status showing greater reduction (Wagland, Steel, Windon and Dineen 1984; Kambara, McFarlane, Abell, McAnulty and Sykes 1993).

The effects of anorexia may be able to be overcome by increased availability of protein in the diet, demonstrated in pen experiments (van Houtert, Barger, Steel, Windon and Emery 1995b).

Kyriazakis, Oldham, Coop and Jackson (1994) demonstrated that when given a choice between a high protein or a low protein diet, sheep infected with *T. colubriformis* had a lowered voluntary food intake, but the proportion of high protein food increased, suggesting that sheep can modify their selection in order to moderate the effects of nematode infection.

There is a lack of data on the mechanism of this inappetence. This was highlighted in a review by Symons (1985). It appears to be dependent on the presence or action of the worm as intake increases when effective immunity develops (Steel, Jones and Symons 1982). Dynes, Ankersmit, Poppi, Barrell and Sykes (1990) established that the depression of food intake was due to signals of the central nervous system, mediated via the hypothalamus rather than peripheral circulating hormones. This was further confirmed in experiments by Dynes, Poppi, Barrell and Sykes (1991) but the exact mechanism still remains unresolved.

2.5.3 Protein Digestion and Absorption

The effect of parasitic infection on protein digestion and metabolism have been extensively studied and reviewed (Parkins and Holmes 1989; Poppi, Sykes and Dynes 1990). The primary effect of infection with *T. colubriformis* on sheep is not to reduce protein absorption *per se*, but to increase plasma protein leakage and especially

gastrointestinal mucus into the small intestine (Poppi, MacRae, Brewer and Coop 1986). Most of this increased endogenous protein production is reabsorbed from the gastrointestinal tract, but a portion flows onto the caecum.

In the caecum, the protein is subjected to the microbial degradation and may be converted into ammonia and absorbed, or pass out in faeces incorporated into microbes. The ammonia absorbed cannot be utilised and is detoxified by conversion to urea which is passed out in urine (Roseby 1977; Kimambo, MacRae, Walker, Watt and Coop 1988). Furthermore, the efficiency of utilisation of the absorbed protein is reduced through repositioning of protein from productive functions such as muscle, bone and wool towards repair and replacement of damage to intestinal tract, mucus production, plasma and immune responses (Steel *et al.* 1980; Bown, Poppi and Sykes 1986).

It has been shown protein supplementation may increase protein deposition and increase live weight gain in infected animals (Bown *et al.* 1986). However, it has not been possible to determine if this is due to simply supplying additional nutrients, or whether there is a secondary effect of reducing the pathological effects of parasites by enhancing the host immune response (Poppi *et al.* 1990).

2.5.4 Other Metabolic Effects

The effects infection with *T. colubriformis* on energy metabolism of the host have not been studied as extensively as the effect on protein metabolism. Sykes and Coop (1976) reported that the gross efficiency of utilisation of metabolisable energy (ME) in lambs infected with *T. colubriformis* was 45-50% lower than controls.

Subsequent studies in infected and pair fed control animals indicated a significant reduction in the digestibility of the ration and therefore in the metabolisable energy (ME) available to the infected animals. In contrast, there was no difference in efficiency of utilisation of absorbed energy between infected and pair fed controls (MacRae 1987).

Chronic subclinical infections of *T. colubriformis* impair skeletal growth in young sheep (Sykes, Coop and Angus 1975). Infected animals show reduced bone formation (ie, length and volume of bones are reduced), and osteoporosis (ie, reduced amount of bone matrix per unit of bone) (Poppi, MacRae, Brewer, Dewey and Walker 1985). The reasons for this remain undetermined, although it is possibly due to reduced protein absorption (Bown, McCall, Scott, Watson and Dow 1989) and the general effect on protein utilisation (Sykes 1982).

2.5.5 Productivity

The main effects of *T. colubriformis* infestation on animal production are to reduce liveweight gain, to reduce wool production and to cause death. With respect to the latter, mortality rates of 68% were recorded by Gordon (1964) in young sheep in New England Tablelands, and 50% by Barger and Southcott (1978). In field experiments in Australia mortality rates in untreated young sheep range from between 10 and 68% (summarised in Barger 1982). In terms of economic analysis of nematode infections in young sheep, mortality rates were found to be the major factor in altering financial

outcome of such infections (Johnstone, Darvill, Bowen, Butler, Smart and Pearson 1979).

The potential for reduced wool production has been demonstrated by Barger, Southcott and Williams (1973), who reported a 42% reduction in clean wool production with penned sheep receiving mild infections of *T. colubriformis*. However, this was over a short period and not likely to reflect the field situation over longer periods. Barger (1982) summarised 12 experiments in which reduced wool production attributable to mild helminth infections was measured in young grazing sheep. The average greasy fleece weight reduction was estimated at 19% with a range of 9% to 31%.

The effect of liveweight gain was also summarised by Barger (1982), and reductions in liveweight gains varied from 23% to 79%. As there are a large number of variables in these experiments, these results can only act as a guide to what might occur, and reflect variation expected in field infections. More controlled trials with penned sheep, in which larval dose rates were controlled have reported a 20% reduction in liveweight gains with a dose of 3000 *T. colubriformis* larvae per week. Steel *et al.* (1980) reported 39% reduction with a dose of 9500 *T. colubriformis* larvae per week.

2.6 DIARRHOEA

2.6.1 Introduction

Diarrhoea, usually known as scouring, is the presence of water in relative excess proportion to dry faecal weight. The pathogenesis has been reviewed (Ooms and Degryse 1986), as has the condition in sheep (Blackwell 1983).

Although there are many causes of diarrhoea in weaners, nematode parasites are certainly the most common cause, and are estimated to occur in at least 85% of submissions for sheep scours to the Regional Veterinary Laboratory, Armidale (O R Coverdale, *pers comm.)*. The predominant parasite species has been *Trichostrongylus spp.* (Holdsworth 1993).

There are many non-parasitic causes of diarrhoea in sheep, and although their overall incidence is low, on individual farms they can be responsible for severe losses. They may be secondary or follow parasitic infections and, in such cases, may substantially potentiate the pathological effects. The different causes of non-parasitic diarrhoea in weaner sheep and the epidemiology and pathogenesis associated with the various causes have been reviewed by Lenghaus (1987); Napthine (1988) and Glastonbury (1990).

It is beyond the scope of this review to cover those diseases, although a summary of those occurring on the New England Tablelands is given.

2.6.2 Nutritional Diarrhoea

Weaner sheep grazing lush green feed often develop diarrhoea. It occurs on pastures dominated by either grasses or clover during the early growing phase. The plant components are highly degradable at this stage and fibre component is very low (Walsh and Birrell 1987).

Two factors of the pasture causing diarrhoea associated with lush pastures are the high water component and, to a lesser extent, high levels of soluble sugars. The ruminal microbial composition is altered leading to the passage of a large volume of partly digested nutrients into the intestinal tract and a consequent increased flow rate. There is a shift in digestion from the rumen to the small intestine and caecum. The resulting elevated levels of soluble sugars in the caecum and colon depress reabsorption of water by their osmotic effect resulting in diarrhoea. However, the digestion of protein is favoured by these changes (unless severe) so that growth rates of animals are usually not affected (H Oddy, *pers comm.*).

The specific effect of various pasture species has generally not been studied, although certain plants are known to be associated with diarrhoea (eg, capeweed, *Arctotheca calendula*), probably due to high moisture content. The presence of high levels of endophyte (*Acremonium lolii*) in perennial ryegrass (*Lolium perenne*) has been demonstrated to cause a marked increase in diarrhoea in lambs (Fletcher and Sutherland 1993). This grass species is a major component (M Duncan, *pers comm.*) of improved pasture on the New England Tablelands.

Affected sheep remain bright and alert, but produce large volumes of green very fluid faeces. An appreciable volume of liquid ingesta in the rumen and fluid large intestinal contents are the only remarkable post mortem findings. A diagnosis of nutritional diarrhoea is indicated by the grazing history and the elimination of other causes of diarrhoea (Glastonbury 1990).

It must be noted that it is often not practical to determine if 'nutritional scour' is induced either totally or partially by parasites. Lush green pasture growth occurs in conditions which generally favour nematode parasite larvae development (Anderson *et al.* 1978), making distinction difficult. Secondly, sheep carrying relatively high burdens of *Trichostrongylus spp.* may not exhibit clinical signs of diarrhoea on dry pasture, but develop classic 'black scours' on lush green pasture.

2.6.3 Diarrhoea Due to Nematode Parasites

The classical syndrome, 'black scour', has been described for trichostrongylosis (see Section 2.5.1). More recently, Larsen, Anderson, Vizard, Anderson and Hoste (1994) reported on the presence of diarrhoea in Merinos. This study suggested the main cause of diarrhoea in sheep grazing early spring pastures was the ingestion of trichostrongylid larvae, and further this developed in sheep that had a well developed protective immune response to those parasites. Importantly, the severity of diarrhoea (as assessed by dag scores) was not related to worm burdens. Its repeatability and difference between bloodlines has been demonstrated (Larsen, Vizard, Webb-Ware and Anderson 1995b) confirming earlier New Zealand observations (Meyer, Harvey and Smeaton 1983).

2.6.4 Coccidiosis

Coccidiosis disease is caused by protozoa of the *Eimeria spp*. There are at least 11 species found in sheep and each species preferentially infects a particular section of the intestinal tract. The ability of the different individual species to cause disease varies,

but mixed infections are the normal occurrence in Australia (O'Callaghan, O'Donoghue and Moore 1987).

Young sheep acquire infection from their peers rather than from their dams or the environment. Considerable amplification occurs within the host with a modest infection of 1000 oocysts leading to the passing of over a billion oocysts 2 to 3 weeks later. A peak in the production of oocysts usually occurs between 20 and 90 days of age. The severity of disease is proportional to the infecting dose and the rate of uptake of oocysts. A large single dose is more likely to cause disease than smaller daily doses.

In acute infections, the characteristic clinical findings are anorexia, abdominal pain and diarrhoea or dysentery. Signs of more chronic disease include anorexia, poor growth, weakness, soft faeces which may be grey due to fibrinous exudation from diseased sections of the intestinal tract, recumbency and death.

Several factors affect the occurrence of coccidiosis. Firstly, maternal antibodies result in passive immunity to the disease in very young lambs that are resistant to the pathogenic effects of some coccidia while being able to respond to them immunologically. Secondly, synergism occurs when more than one species infect an animal concurrently and mixed infections cause more severe pathological effects. Finally, nutritional stress, inclement weather and overcrowding all predispose to infection, conditions which occur not uncommonly to Merino weaners.

The disease has been reported in Merino weaners in the New England under the conditions described (Clark and Campbell 1966), but it must be noted that the disease may be confused with, or occur concurrently with, trichostrongylosis (Walker 1958).

2.6.5 Yersiniosis

Yersiniosis is a bacterial disease that has been recognised in sheep for many years, but the enteric form has assumed increasing importance in New Zealand (McSporran, Hansen, Saunders and Damsteegt 1984) and more recently in Southern Australia (Slee and Button 1990a, 1990b). It is caused by *Yersinia pseudotuberculosis* and *Yersinia enterocolitica*. *Y. pseudotuberculosis*, the most common cause of the disease, is a normal inhabitant of the intestinal tract in a proportion of sheep. The disease may occur in sheep of any age, but is more common in weaners and hoggets.

Several epidemiological factors have been associated with yersiniosis in south eastern Australia. Infections with *Y. pseudotuberculosis* occur in a distinct seasonal pattern in late winter and early spring. These are periods of cold weather and generally higher rainfall. In many instances, the disease occurs concurrently with trichostrongylosis, and it is likely trichostrongylosis predisposes animals to the disease (Philbey, Glastonbury, Links and Matthews 1991).

Predisposing factors include high stocking rates on lush green pasture, a change of diet, onset of cold wet windy weather, weaning and shearing. Diarrhoea, dehydration, illthrift and mortality are the principal clinical findings. Diarrhoeic faeces vary from green and fluid to black and mucoid and cause faecal staining about the perineum.

The disease is less frequent in New England compared with southern New South Wales and Victoria. The suggested reasons for this are that weaners are older in winter (compared with autumn drop lambs in southern New South Wales), winters are usually drier, and nematode burdens are usually lower (Love 1989).

2.6.6 Weaner Bacterial Enteritis

This is a commonly used term where the specific cause remains unknown or where mixed infections occur under various circumstances. Besides *Yersiniosis spp.* already noted, *Campylobacter spp.* are commonly isolated (Stephens, Browning, Slee, Hayes and Tzipori 1984). The disease is associated with such factors as malnutrition and adverse climate conditions. Concurrent nematode infections are usually another complicating factor, and often the disease remains following elimination of parasites. It is not common on the New England Tablelands compared with southern New South Wales and Victoria for the same reasons Yersiniosis is uncommon (Lenghaus 1982). It is common in New Zealand under the name "Winter Scours" (Orr and Pearson 1988).

2.6.7 Selenium Deficiency

The New England Tablelands is a known selenium deficient area (Plant 1985) and the deficiency syndrome is associated with a variety of disease syndromes in sheep. While muscular dystrophy ("White Muscle Disease") is the classical and characteristic disease, there are a number of sub clinical syndromes loosely termed "selenium responsive" that occur in sheep, amongst these are weaner illthrift and "selenium responsive" diarrhoea (Hart 1985).

It is commonly speculated that the reason selenium deficiency causes diarrhoea is that it causes a depressed immunological response and hence sheep are more susceptible to pathogens. Although laboratory studies have shown that laboratory animals given selenium may develop higher antibody levels than deficient animals (reviewed by Petterson 1985), this effect has not been substantiated in the field (Suttle and Jones 1989). Ellis, Masters, Hustas, Sutherland and Evans (1990) found no difference in antibody response in Merino sheep with low selenium status.

Studies on the effect of selenium deficiency on cell mediated immunity suggest that the phagocytic activity of neutrophils is not impaired in selenium deficient animals, but their activity against some organisms is reduced (Arthur, Nicol, Boyne, Allen, Hayes and Beckett 1987).

An alternative suggestion from Langlands (*pers comm.*) is that the effect may be caused by a combination of low Vitamin E and selenium as the condition is reported commonly in seasons of lush green feed which is the period when selenium is lowest in pasture, and when the demand for Vitamin E is highest due to high levels of polyunsaturated fatty acids (Arthur 1992).

2.6.8 Productivity Effect of Diarrhoea

Diarrhoea in Merino weaners causes major management problems on the New England Tablelands. Affected sheep accumulate faeces around the breech (known as "dag"), which must be removed by crutching. This of itself is a direct cost and, in addition, potentially high-value wool is sold at a heavily discounted price. Larsen, Vizard, Anderson (1995a) qualified this in a Victorian study, but noted that any such calculations are sensitive to wool price, relative price premium for fine wool, treatment costs and production responses to treatments. Secondary costs included effects on growth of lambs.

Morley, Donald, Donnelly, Axelsen and Waller (1976) found a direct relationship between diarrhoea and breech strike by *Lucilia cuprina* in Merinos. Watts, Dash and Lisle (1978) found diarrhoea rendered mulesed Merino sheep more susceptible to breech strike on the New England Tablelands.

2.7 CONCLUSION

Modern anthelmintics when used in "Wormkill", the strategic drench program developed for the New England Tablelands, give adequate control of nematode parasites in sheep, but some "breakdowns" occur. One major problem is the failure of the program, despite the recent modifications, to give adequate control of *Trichostrongylus spp.* over the autumn/winter period in Merino weaners.

The reason for this may be that in Merino weaners poor nutrition reduces host resilience, ie, the ability of the host to handle parasitic infections. Nutritional supplementation assists the host by supplying the extra nutritional requirement imposed by trichostrongylosis infection, and hence alleviates the clinical expression and pathological affects of infection.

Secondly, poor nutrition may compromise the resistance to parasites which is developing in Merino weaners at this age, ie, prevent the development of an effective immunity to parasites which allows parasites to build up in the host, as well as reducing the host's ability to suppress egg production. This has the compounding effect of increasing larval contamination on pasture leading to parasite burdens that cause inappetence, illthrift associated with lower growth rates and production losses.

This suggests that further studies are required to quantify the effect of nutrition in weaners infected with parasites over this period and determine the specific effects of poor nutrition on the development of parasites.