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Parasites and their impact

K. Wilson *University of Stirling*
B. T. Grenfell *University of Cambridge*
J. G. Pilkington *University of Edinburgh*
H. E. G. Boyd *University of Cambridge*
and
F. M. D. Gulland *Marine Mammal Center, Sansalito, USA*

5.1 Introduction

Highly pathogenic *epidemic* disease agents, like the rinderpest and myxomatosis viruses, have obvious and often dramatic consequences for the dynamics and evolution of their host populations (Osterhaus and Vedder 1988; Roelke-Parker *et al*. 1996; Vogel and Heyne 1996; Hudson 1997; Hochachka and Dhondt 2000). In contrast, the effects of *endemic* diseases are often more subtle and, until recently, had largely been overlooked (Grenfell and Dobson 1995; Hudson 1997; Hudson *et al*. 2002). The mathematical models of Anderson and May in the late 1970s (Anderson and May 1978, 1979, 1982; May and Anderson 1978, 1979) were the first to highlight the potential of endemic parasites and pathogens to regulate host populations, and more recent theoretical studies have also implicated parasites as potentially important driving forces in the evolution of their hosts. Their impact is believed to extend to the evolution of secondary sexual characters (Hamilton and Zuk 1982; Read 1987, 1988; McLennan and Brooks 1991; Hamilton and Poulin 1997) and optimal life-history strategies (Michalakis and Hochberg 1994; Sheldon and Verhulst 1996; Richner 1998); the manipulation of host behaviour (Moore 1984; Poulin 1994; Moore and Gotelli 1996); the maintenance of genetic diversity and even to the evolution of sex (Van Valen 1973; Hamilton 1980; Hamilton *et al.* 1990; Moritz *et al*. 1991; Howard and Lively 1994).

Empirical support for these models comes largely from laboratory studies (Grenfell and Dobson 1995; Clayton and Moore 1997; Hudson

Soay Sheep: Dynamics and Selection in an Island Population, ed. T. Clutton-Brock and J. Pemberton.
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et al. 2002). Data from natural systems are much thinner on the ground, particularly for long-lived vertebrates, due mainly to the logistical difficulties of collecting long-term demographic data. These problems have largely been overcome on St Kilda, where demographic data have been collected continuously since 1985. Other problems associated with most other natural host–parasite systems include the complication of competing herbivores and predators (e.g. Hudson *et al.* 1992a; Stenseth 1995) and the possibility of migration into and out of the study population. These problems are much reduced on St Kilda, where the system can be characterized as a plant–herbivore–parasite interaction with zero dispersal and restricted sheep movement about the island (Grenfell *et al*. 1992, 1998; Coulson *et al*. 2001; Chapter 3).

In this chapter, we assess the importance of parasites for the evolution and population dynamics of their hosts, focussing on the Soay sheep of St Kilda. Specifically, we address the following questions: What effect does island life have on the parasite community? Which parasites are likely to cause most damage to the health of their hosts and how can we measure their abundance? What factors are likely to generate variation in parasite loads? What are the costs of parasitism for the host, in terms of growth, survival and reproduction? And, do parasites cause changes in the population density of their hosts or merely reflect them?

One of the advantages of studying the parasitology and epidemiology of Soay sheep is that there is a large complementary literature for their domestic relatives. Clearly, there will be similarities and differences between the domestic and wild situation, and throughout this chapter emphasis is placed on making these comparisons, in an attempt to understand both systems better. We also attempt to use the Soay sheep system to draw some insights into the impact of parasites on other vertebrate host populations in the wild.

5.2 The parasite community

THE PARASITES AND THEIR PATHOLOGICAL CONSEQUENCES

Island life potentially has a number of important implications for parasites. First, parasite communities on islands are likely to be

influenced by 'founder effects' (e.g. Grant 1998). These will determine not only which parasite species are carried to the island, but also the genetic make-up of the founding host population, which may affect their relative susceptibility to particular parasite species. A second consequence of island life is that the host population is typically more isolated from neighbouring populations. We can therefore expect some parasite species to be absent from islands merely as a consequence of the fact that they lack the capacity to reach, and subsequently infect, their hosts. Third, islands tend to harbour fewer species in general, and this will affect the transmission potential of those parasite species that rely on one or more intermediate host species in order to complete their life cycles. Thus, we can expect hosts living on islands typically to be infected with relatively fewer indirectly transmitted parasite species (i.e. those species that require intermediate hosts or vectors). A final consequence of island life is that host populations are generally smaller than those on the mainland. As a consequence, the number or density of hosts may be below the 'critical threshold level' for the persistence of some parasite and pathogen species (e.g. measles is endemic only in human populations of greater than 5 million people; Anderson 1993). This is because the parasite or pathogen runs out of susceptible hosts to infect (i.e. uninfected immigrants or newborns). This effect is likely to be particularly important for highly virulent viral or bacterial pathogens that either kill their hosts or induce long-lasting effective immunity (see Swinton *et al.* (2002) for a full discussion of the persistence of microparasite infections of wildlife). Conversely, immunity against protozoans and macroparasites (i.e. nematodes, tapeworms, flukes, etc.) tends to be short-lived and infections can persist in individual hosts for long periods of time. As a consequence, most protozoan and macroparasite populations can survive endemically in small host populations with low inputs of susceptibles (Anderson 1993). Thus, we expect rather few macroparasite and protozoan species to be absent from the parasite fauna on islands. So, what do we observe on St Kilda?

The parasites of the Soay sheep on St Kilda were first investigated in the early 1960s (Cheyne *et al.* 1974). Although these studies were rather limited in duration, most of the parasites now known from

St Kilda were first identified during this period (Table 5.1), and the conclusions drawn from these initial investigations remain largely true today (Cheyne *et al.* 1974). The parasite community is fairly typical of domestic hill sheep in Scotland (Soulsby 1982) with one or two interesting differences. For example, the sheep tick, *Ixodes ricinus*, and larval stages of the blow fly *Lucilia sericata*, are notable absentees from the ectoparasite faunae (Boyd *et al*. 1964; Cheyne *et al.* 1974), and the highly pathogenic and widespread nematode *Haemonchus contortus* is also conspicuous by its absence (Soulsby 1982). As expected, many common bacterial and viral infections of sheep also appear to be absent, presumably because the host population size is too small to sustain them (Grenfell and Dobson 1995; Hudson *et al*. 2002). These include Johne's disease (*Mycobacterium paratuberculosis*), enzootic abortion of ewe (*Chlamydia psittaci*), *Mycoplasma ovipneumoniae*, Border disease virus and Maedi–Visna virus (St Kilda Soay Sheep Project Annual Report 2001, unpublished). Conversely, there is a rich community of protozoans present on the island (Table 5.1).

The tapeworm *Taenia hydatigena* is a surprise inclusion to the parasite fauna. This parasite was first recorded on St Kilda in the 1960s, when 49% of adult sheep were infected (Cheyne *et al.* 1974). The prevalence of infection has remained high ever since, with 31% of adults harbouring this parasite in 1989 (Gulland 1992) and 30–53% in 1992 (Torgerson *et al.* 1992, 1995). The presence of *T. hydatigena* on St Kilda is noteworthy because its cystercercal stage, which encysts in the linings of the sheep's rumen and abomasum, must be ingested by a carnivore intermediate host before its life cycle can be completed. The absence of carnivores on St Kilda since the removal of dogs in the 1930 human evacuation (Chapter 2) makes the presence of this parasite on the island paradoxical (see above). It seems most likely that eggs of this parasite are being brought over to the islands repeatedly by birds from the mainland (Torgerson *et al*. 1995). Despite its relatively high prevalence, individuals rarely harbour more than two cystercerci (Torgerson *et al.* 1992) and so it is unlikely that this parasite is an important source of morbidity or mortality on St Kilda. The same can be said of the only other tapeworm on the island, *Moniezia expansa*. Adults of this species live in the small intestine, and

Table 5.1. *List of parasite species associated with Soay sheep on St Kilda*

Taxon	Specific name	Location
Protozoa	*Cryptosporidium parvum*	Small intestine
	Giardia duodenalis	Small intestine
	Eimeria ahsata	Small intestine
	Eimeria bakuensis	Small intestine
	Eimeria crandallis	Small intestine/large intestine
	Eimeria faurei	Small intestine/large intestine
	Eimeria granulosa	Unknown
	Eimeria intricata	Small intestine/large intestine
	Eimeria marsica	Unknown
	Eimeria ovinoidalis	Small intestine/large intestine
	Eimeria pallida	Unknown
	Eimeria parva	Small intestine/large intestine
	Eimeria weybridgensis	Small intestine
Bacteria	*Dermatophilus congolensis*	Skin
Flies	*Melophagus ovinus*	Wool
Lice	*Damalinia ovis*[a]	Wool
Tapeworms	*Moniezia expansa*	Small intestine
	Taenia hydatigena	Abdominal cavity
Nematodes	*Dictyocaulus filaria*	Lungs
	Muellerius capillaris	Lungs
	Teladorsagia circumcincta[b,c]	Abomasum
	Teladorsagia trifurcata[b,c]	Abomasum
	Teladorsagia davtiani	Abomasum
	Trichostrongylus axei	Abomasum/small intestine
	Trichostrongylus vitrinus	Abomasum/small intestine
	Capillaria longipes	Small intestine
	Strongyloides papillosus	Small intestine
	Nematodirus battus	Small intestine
	Nematodirus filicollis	Small intestine
	Nematodirus helvetianus	Small intestine
	Bunostomum trigonocephalum	Small intestine
	Trichuris ovis	Large intestine
	Chabertia ovina	Large intestine

[a]Pseudonym *Bovicola* or *Trichodectes*.

[b]Pseudonym *Ostertagia*.

[c]Now believed to be a single species (see text).

Sources: Cheyne *et al.* (1974), Gulland (1992), J. G. Pilkington unpublished data, B. H. Craig, unpublished data.

infected individuals usually harbour just a single worm (K. Wilson, unpublished data).

Both lice and keds (wingless flies) are common on St Kilda. In 1964, the prevalence of these two ectoparasites was greater than 90% (Cheyne *et al.* 1974). Although absolute counts of keds and lice have not been made since, relative measures of ked abundance in August have been determined since 1988 (a count is made of the total number of keds observed during a 1-minute search of the wool on the sheep's belly). These indicate that keds are numerous only on young animals, particularly ram lambs. Sheep keds live in the wool of their hosts, but feed on blood. Heavy infestations can severely reduce host condition and cause anaemia; however their main damage results from the irritation they cause. Sheep lice also feed mainly on fragments of wool and other epidermal products, but occasionally feed on blood from open wounds. As with keds, the chief effects of lice on their hosts are due to the irritation they cause: infected sheep may become restless and reduce the amount of time they devote to sleeping and feeding (Soulsby 1982).

Two species of lungworm are found in sheep on St Kilda. *Dictyocaulus filaria* is a widespread, directly-transmitted, parasite. The adult worms live in the tracheae and bronchi and cause a catarrhal parasitic bronchitis, which may develop into pneumonia (Soulsby 1982). Infected lambs may be observed coughing up parasite eggs, which are then swallowed and hatch as they pass through the alimentary tract, before being voided on to the pasture as larvae. This parasite predominantly affects lambs, whilst older animals are more commonly infected with *Muellerius capillaris*. This is probably the commonest lungworm of sheep in Europe and requires molluscs as intermediate hosts (Soulsby 1988). Adults of *M. capillaris* live in the alveoli and pulmonary parenchyma. Infected animals generally show no clinical signs, but heavy infections weaken the lungs and may lead to a reduction in the general fitness of the host (Soulsby 1982). Little is known about its prevalence or impact on St Kilda.

Soay sheep harbour a range of gastrointestinal nematodes, the most numerous of which belong to the genus *Teladorsagia*. During the

population crash of 1989, these 'brown stomach-worms' comprised approximately 75% of the total number of worms living in the gut and, based on morphology, *T. circumcincta* (formerly referred to as *Ostertagia circumcincta*) represented about 85% of these, with *T. trifurcata* and *T. davtiani* making up the remainder (Gulland 1992). However, recent genetic studies on the worms from St Kilda could find no evidence to separate these three putative species based on nuclear or mitochondrial DNA sequences (Braisher 1999). It seems likely, therefore, that there is just a single *Teladorsagia* species on St Kilda and we will refer to this as *T. circumcincta*.

On St Kilda, individuals have been known to harbour more than 20 000 of these small (7–12 mm) parasites in their abomasum (Gulland 1992). On the mainland, *T. circumcincta* is extremely common, and pathogenic: symptoms of infection include poor weight gain or weight loss, loss of appetite and diarrhoea (reviewed by Holmes 1985; Symons 1985). Because the pathological consequences of parasitism by these trichostrongylids are dose-dependent (Downey *et al.* 1972; Coop *et al.* 1982), a range of control measures are commonly employed by farmers on the mainland to reduce levels of infection in domestic sheep, including treatment with anthelminthic drugs and transfer to clean pastures.

Post-mortems performed during the population crash of 1989 (Gulland 1992) found that the sheep were emaciated and had no fat reserves. The abomasal walls were reddened and lesions were present. These observations, and biochemical evidence (Gulland 1992), are consistent with the animals having died from protein-energy malnutrition, accentuated by damage caused by trichostrongylids (Cheyne *et al.* 1974; Gulland 1992). Thus, as well as being the numerically dominant parasite on St Kilda, for most of the sheep *T. circumcincta* also appears to be commonly the most pathogenic. For these reasons, the majority of the parasitological and epidemiological studies on St Kilda have centred on this parasite and *T. circumcincta* is the focus of the remainder of this chapter (for a discussion of some of the other parasites on St Kilda see Cheyne *et al.* 1974; Gulland 1992; Gulland and Fox 1992).

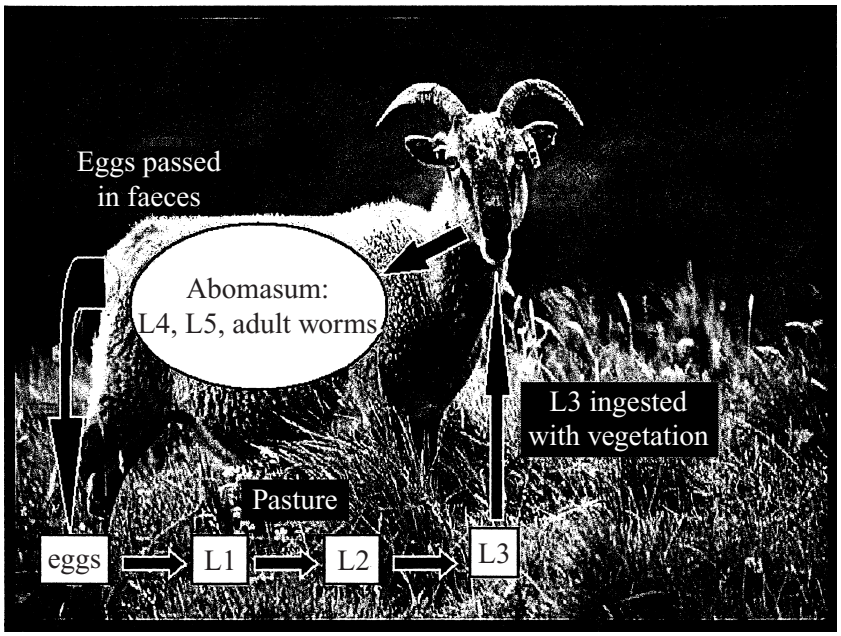


FIG. 5.1. Life cycle of *Teladorsagia circumcincta*. L1–L5 refer to first to fifth instar larvae. See text for further details.

LIFE-CYCLE OF *TELADORSAGIA CIRCUMCINCTA*

Teladorsagia circumcincta is a directly-transmitted, gastrointestinal trichostrongylid and its life cycle (Fig. 5.1) is typical of this family as a whole (for reviews see Dunn 1978; Soulsby 1982; Gulland 1991). Adult females produce eggs, which develop to the morula stage before being voided in the faeces. Hatching may take place in as little as 24 hours, but eggs can survive on the pasture for several months prior to hatching, depending on environmental conditions. The emerging first-stage larvae (L1) moult in to the second stage (L2), which undergo a second moult to become the infective third-stage larvae (L3). The cuticle of the L2 is retained as a loose sheath around the L3 and provides it with some protection against adverse climatic conditions.

Following ingestion by the host, the L3 larvae exsheathe in the rumen and pass to the abomasum, where they enter the gastric glands on the second or third day. In the glands, the larvae undergo a third

moult, to become early fourth-stage larvae (EL4) followed by a fourth moult to the fifth stage, or immature adult. By day 12, most of the larvae have reached the mature-adult stage and, by day 16, they begin to emerge from the gastric glands as adult worms and attach themselves to the walls of the abomasum. These worms become sexually mature, copulate and the females lay eggs, which first appear in the faeces 17 or 18 days post-infection. The average lifespan of an adult worm is probably around 50 days, but mortality of adult worms may occur from 16 days post-infection. Although development within the host normally takes about three weeks, larvae may become arrested at the EL4 stage. This process, referred to as *hypobiosis*, occurs within the mucosa and may last for up to three months (Armour *et al.* 1969). The mechanisms determining whether, and for how long, a larva undergoes arrested development are not yet fully understood, and from an epidemiological point of view this is currently the largest gap in our knowledge (see below). However, genetic, climatic, density-dependent and immunological effects have all been implicated (Michel 1974; Gibbs 1986). In mainland Scottish sheep infected with *T. circumcincta*, the probability of an EL4 arresting development increases from low levels in late summer (August–September) to nearly 100% in late winter (December–January). De-arrestment occurs in early spring (April–May), coinciding with lambing (Reid and Armour 1972).

MEASURES OF PARASITISM

Since the sheep on St Kilda are protected, it is not possible to assess their worm burdens directly by sacrifice and post-mortem examination, and indirect measures have to be used, such as faecal egg counts (FEC). This involves collecting faecal samples from free-ranging, ear-tagged sheep and determining the density of parasite eggs per gram (wet weight) of faeces using a modification of the McMaster technique (Ministry of Agriculture Fisheries and Food 1971). Eggs are then classified in to several taxa: *Moniezia expansa*, *Capillaria longipes*, *Trichuris ovis*, *Nematodirus* spp. and strongyles (comprising *Teladorsagia* spp., *Trichostrongylus* spp., *Chabertia ovina, Bunostomum trigonocephalum,*

Strongyloides papillosis). In the remainder of this chapter, the term faecal egg count will be used to refer exclusively to counts of strongyle eggs. Counts of *Dictyocaulus filaria* larvae are made using similar methods (see Gulland and Fox 1992), but these will not be discussed in detail here. In addition to measuring parasitism rates, the densities of infective strongyle larvae on the pasture per kilogram of grass (wet weight) are also measured, using standard techniques (Gulland and Fox 1992).

Parasitism rates are frequently described in terms of *prevalence* (proportion of animals infected) and *intensity* (mean parasite burden per animal). On St Kilda, the prevalence of strongyle infection is high and shows relatively little systematic variation (Gulland and Fox 1992). Therefore, in this chapter we focus mainly on variation in parasite intensity, as measured by strongyle faecal egg counts (see Wilson *et al*. (2002) for a discussion of the biases associated with indirect measures of parasitism). A key assumption of these analyses is that FEC and strongyle worm burdens are positively correlated. However, because FEC is a function of both worm burden and worm fecundity, this assumption will not be true if there is strong non-linear density-dependence in worm fecundity (Cabaret *et al*. 1998). Fortunately, several lines of evidence suggest that FEC and worm burden are strongly correlated in Soay sheep. First, Gulland (1992) found that there was no density-dependence in the fecundity of female worms in sheep dying during the 1989 crash. Second, in Soay sheep dying naturally on St Kilda during the population crash of 1992, there was a strong positive linear relationship between FEC (measured several days prior to death) and the absolute number of *Teladorsagia* worms harboured in the abomasum (measured within 24 hours following death) (closed squares in Fig. 5.2) (Wilson 1994; Grenfell *et al*. 1995). Third, a similar relationship was observed in a feral population of Soay sheep culled on the island of Lundy in 1995 (open squares in Fig. 5.2) (Boyd 1999). Most importantly, despite spanning worm burdens that differed by nearly 100-fold, there was no significant difference between the two populations in the slopes or intercepts of the linear regressions describing the relationship between log-FEC and log-worm burden (Fig. 5.2). Thus, faecal egg count appears to be a useful measure of parasitism in this population.

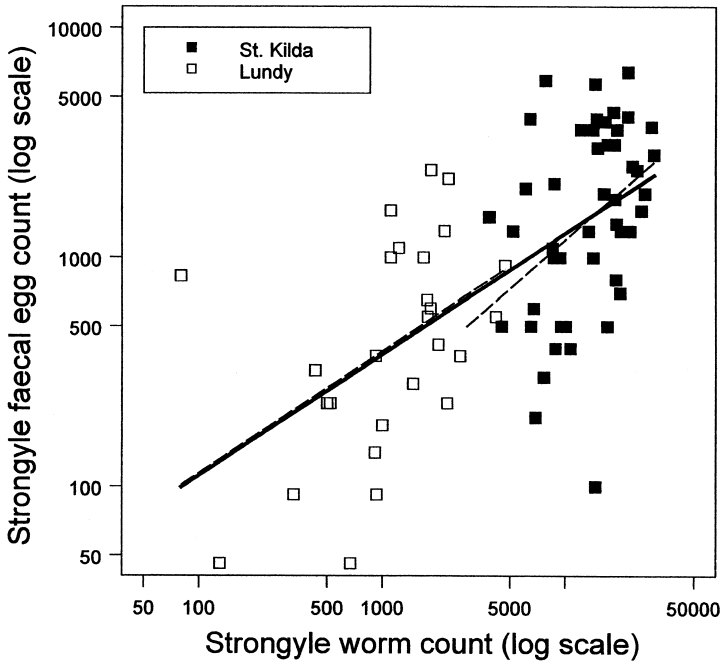


FIG. 5.2. Relationship between faecal egg count and strongyle worm burden. Filled symbols are for Soay sheep dying naturally on St Kilda (K. Wilson, unpublished data) and open symbols are for Soay sheep culled on Lundy (Braisher 1999; Boyd 1999). Strongyle faecal egg counts are number of strongyle eggs per gram of faeces. For the Lundy sheep, faecal egg counts were taken immediately prior to culling, whereas those for the St Kilda sheep were recorded approximately two weeks prior to death (K. Wilson, unpublished data). Worm counts are numbers of *Teladorsagia* adults in the abomasum. Lines represent least-squares fits of linear models; dashed lines are for the two separate data sets and the solid line is for the combined data set. The regressions for both St Kilda ($r^2 = 0.147$, $F_{1,46} = 7.899$, $p = 0.007$) and Lundy ($r^2 = 0.216$, $F_{1,25} = 6.91$, $p = 0.015$) were statistically significant. Neither the slopes ($t = 0.531$, $df = 73$, $p > 0.5$) nor intercepts ($t = 0.608$, $df = 73$, $p > 0.5$) differed significantly between studies. Overall regression: $\log_{10}$(faecal egg count) = 0.992 + 0.528*$\log_{10}$(worm count), $r^2 = 0.392$, $F_{1,73} = 47.05$, $p < 0.0001$.

5.3 Variation in parasitism rates

QUANTIFYING VARIATION

Parasite distributions are usually highly aggregated, with some hosts having lots of parasites and most having just a few (Fig. 5.3) (Wilson

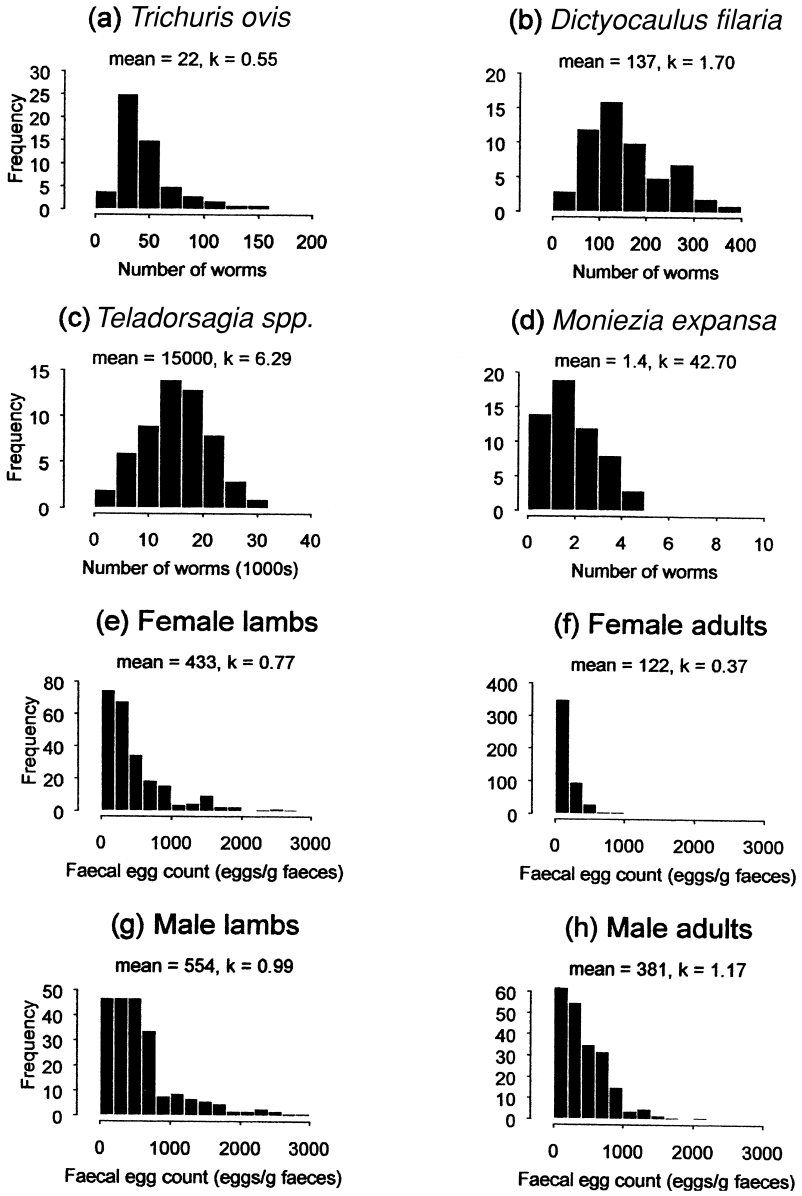


FIG. 5.3. Frequency distributions for worm burdens and faecal egg counts. (a)–(d): Data are post-mortem worm burdens for four taxa of parasites found in Soay juveniles that died on St Kilda during the winter of 1991–2: *Trichuris ovis* from the large intestine, *Dictyocaulus filaria* from the lungs, *Teladorsagia* spp. from the abomasum and *Monieza expansa* from the small intestine. (e)–(h): Data are faecal egg counts for four sex–age classes collected during the period 1988–93. For each distribution, its mean and exponent k (of the negative binomial) are shown. See Wilson *et al.* (1996) for details.

et al. 2002). Patterns like these are often best described by the negative binomial distribution (Fisher 1941; Pennycuick 1971), which is defined by its mean and an exponent, k, which determines the degree of skew in the data. When k is small, the distribution is highly aggregated with a long tail (e.g. Fig. 5.3a); whereas when k is large (>20), the distribution is often bell-shaped and approximates the Poisson distribution (e.g. Fig. 5.3d) – at this point, the parasites are randomly distributed across hosts.

The exponent k is a useful parameter to quantify: statisticians need to measure k because this determines the degree of skew in the parasite distribution that must be controlled for when performing statistical analyses (Wilson *et al.* 1996; Wilson and Grenfell 1997); while, for parasitologists, k is important because it identifies heterogeneities in parasitism and can lend some insights into the relative importance of different epidemiological processes, such as parasite-induced mortality and acquired immunity (Anderson and Gordon 1982; Pacala and Dobson 1988; Grenfell *et al*. 1995; Wilson *et al*. 2002). The shape of the parasite distribution is also important for evolutionary biologists and population dynamicists (Anderson and May 1978; May and Anderson 1978; Poulin and Vickery 1993): if parasites affect the health and survival only of those individuals with high parasite burdens (i.e. those in the tail of the parasite distribution), then the proportion of highly parasitised hosts will be determined not only by the *mean* of the parasite distribution, but also by its *shape*. Thus, if the parasites are randomly distributed across hosts (and follow the Poisson distribution), then a relatively larger proportion of individuals will be in the susceptible tail of the distribution, relative to when this distribution is highly skewed. In the former case, therefore, parasites are likely to be relatively more important as both a selection pressure (Poulin and Vickery 1993) and a regulatory influence (Anderson and May 1978; May and Anderson 1978). Knowing the shape of the parasite distribution is therefore important from both a statistical point of view and in terms of understanding the parasite–host interaction. However, only rarely have changes in the shape of the parasite distribution been tracked through time or across cohorts for a naturally infected host population (Grenfell *et al*. 1995).

In Soay sheep, the adult parasite distributions show variable degrees of aggregation (Gulland 1992; Wilson *et al.* 1996): *Trichuris ovis* is highly aggregated ($k = 0.55$), whereas *Dictyocaulis filaria* ($k = 1.70$) and *Teladorsagia* spp. ($k = 6.29$) are less so, and the tapeworm *Moniezia expansa* exhibits little aggregation ($k = 42.70$) and conforms to the Poisson distribution (Fig. 5.3a–d). In both Soay sheep (Gulland 1992) and domestic sheep (Hong *et al.* 1987; Grenfell *et al*. 1995), adult parasites tend to become increasingly aggregated with host age. These trends are probably due to the effects of genetic and developmental heterogeneities on host immunity (Grenfell *et al*. 1995).

Faecal eggs counts also conform to the negative binomial distribution (Fig. 5.3e–f) and k values on St Kilda generally range between about 0 and 2.5 (Grenfell *et al*. 1995). These values are much smaller than equivalent values for adult *Teladorsagia* worms (cf. Figs. 5.3c and e), but this partly reflects the much lower means for FEC than worm counts. The distribution of FEC tends to become increasingly aggregated with age in females (cf. Figs. 5.3e and f), but not in males (cf. Figs. 5.3g and h), indicating that different density-dependent processes are acting on the two sexes (see below). It is interesting that in Soay sheep and domestic sheep, parasite distributions tend to become increasingly aggregated as their means decline (i.e. the k of the distribution is positively correlated with its mean); Grenfell *et al*. (1995) discuss possible interpretations of this trend.

Because parasite distributions are aggregated, average parasite loads are often expressed in terms of their geometric means (back-transformed mean of the logged data) or are displayed on log-transformed axes. Such conventions are generally followed here.

TEMPORAL AND SPATIAL VARIATION IN EXPOSURE TO PARASITES

The density of infective (L3) larvae on the pasture shows two seasonal peaks (solid line in Fig. 5.4a). One peak in L3 counts occurs in the late spring (May–June), mainly due to the development of eggs deposited on the pasture by immunocompromised peri-parturient ewes (see below). A second peak in L3 density occurs in midsummer (around August), due to the development of eggs shed onto the pasture in the previous months predominantly by immunologically naive lambs

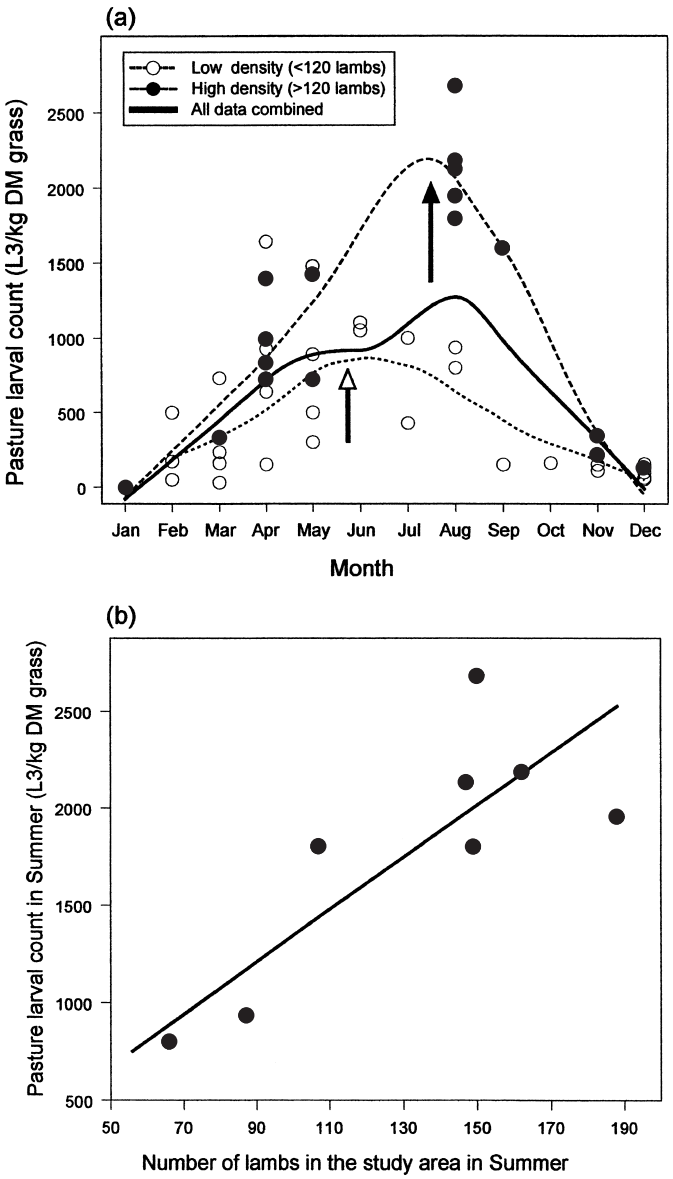


FIG. 5.4. Relationship between number of lambs using the study area and number of infective L3 larvae on the pasture (a) within-years and (b) across-years. In (a), data cover the period 1987–97 and are split into high lambing years (>120 lambs) and low lambing years (<120 lambs); the peak in L3 density is later in high density years than in low density years (lines are Loess fits and the peak densities are indicated by the arrows). In (b), data are for August in the years 1988–97 inclusive (excluding 1994 and 1995, for which there are no data). The linear regression equation is: Density of L3 on pasture in summer = 163.4 + 12.3 × number of lambs in the study area in summer, $r^2 = 0.64$, $F_{1,6} = 10.61$, one-tailed $p = 0.009$.

Table 5.2. *Pearson's correlations between the density of infective L3 larvae on the pasture and the density of sheep*

Population estimate[a]	Spring L3 count		Summer L3 count		Autumn L3 count	
	r	$p^b(n = 9)$	r^c	$p^{b,c}(n = 8)$	r	$p^b(n = 9)$
Lambs	0.391	0.150	**0.800**	**0.009**	0.571	0.054
Ewes	0.039	0.461	0.475	0.117	0.249	0.259
Rams	0.162	0.338	0.256	0.271	0.017	0.483
Total	0.214	0.290	**0.689**	**0.029**	0.439	0.119

[a]Estimated population size in same year as L3 count was made, except for spring correlations, which used the population estimate in previous year. For spring (April–May) and autumn (October–December), L3 counts cover the period 1989–97 inclusive. For summer (August), counts are for 1988–97 inclusive (excluding 1994 and 1995 for which there are no data). Lambs are all animals less than twelve months old; ewes are all females more than twelve months old; rams are all males more than twelve months old; total = lambs + ewes + rams.

[b]One-tailed p values, based on the prediction that L3 counts would be positively correlated with sheep density.

[c]Numbers in bold are significant at the 5% level.

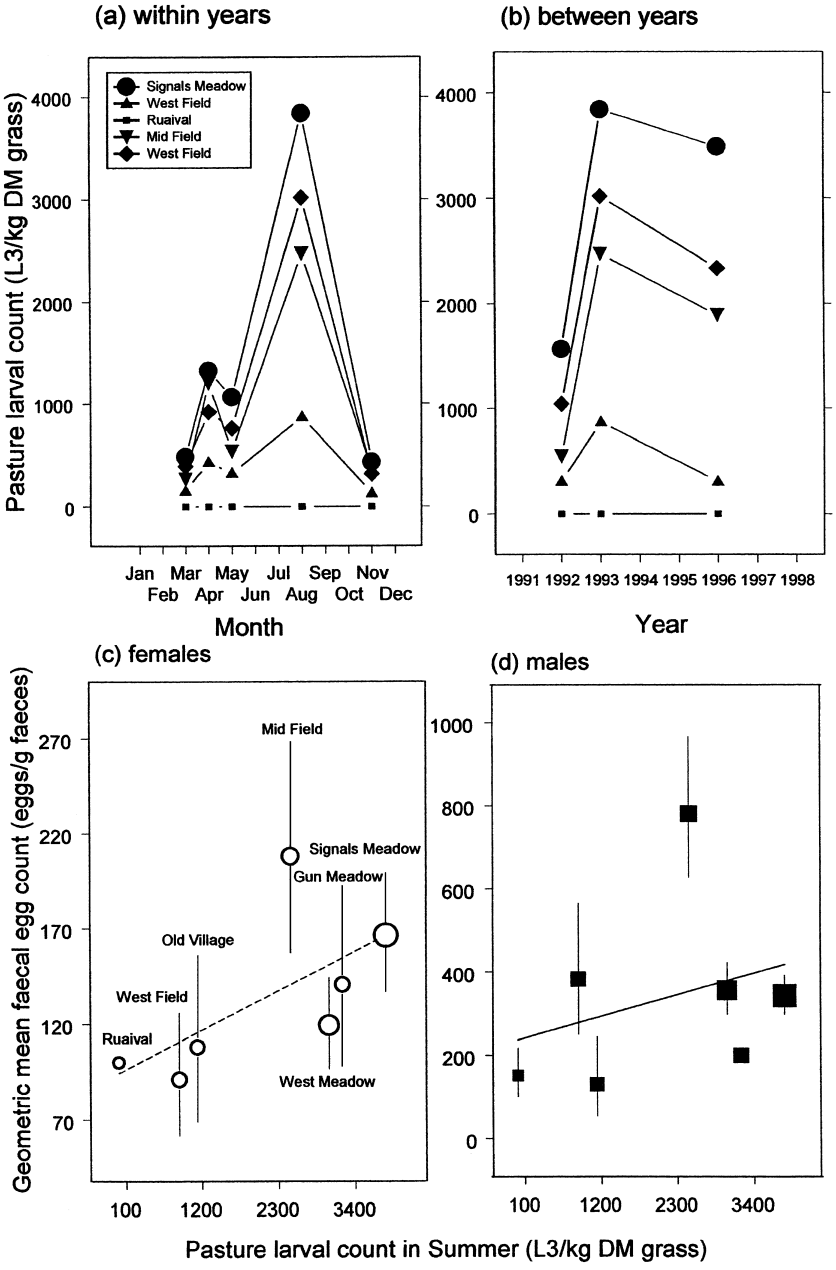
(pasture counts decline in late summer partly due to the immunity acquired by these lambs). A closer look at Fig. 5.4a reveals that the magnitude of this second peak is dependent on the number of lambs in the population: in years when there is a small crop of lambs, there is generally a single L3 peak in late spring, whereas when the density of lambs is high, the second L3 peak in midsummer is revealed (cf. dotted lines in Fig. 5.4a).

Across years, the density of L3 in Village Bay in August increases in a linear fashion with the current size of the lamb population, but not with the density of adult males or females (Table 5.2; see also Fig. 5.4b). In both spring and autumn, the density of infective larvae on the pasture is again most strongly correlated with the number of lambs feeding on the pasture, but neither of these correlations is statistically significant (Table 5.2). Thus, although 'stocking density' influences the number of infective larvae on the

pasture at some times of year (e.g. summer), as found for domestic sheep (Cameron and Gibbs 1966; Downey and Conway 1968; Thamsborg *et al*. 1996), other influences such as climate (temperature and rainfall) appear to predominate for much of the year (see also Ollerenshaw and Smith 1969; Paton *et al.* 1984; Besier and Dunsmore 1993).

The sheep on St Kilda segregate into hefts or social groups, which differ in their frequency of specific genotypes and in their survival and reproductive rates (see Chapters 2 and 4). Parasites could be partly responsible for generating these heterogeneities and so here we address spatial variation in the parasite distribution, both within and outside the host. There is consistent spatial variation in the density of infective larvae on the pasture both within (Fig. 5.5a) and between (Fig. 5.5b) years. Within years, the repeatability, r ($\pm$ approximate standard error; see Lessells and Boag 1986) is 0.30 ± 0.15 ($p = 0.018$) and across years, it is 0.74 ± 0.15 ($p = 0.007$). So, within years approximately 30% of the variation in the density of infective larvae on the pasture is due to variation between areas, whereas across years the explained variation increases to more than 70%. This spatial variation is partly maintained by variation in the density of sheep in the different areas (as indicated by the sizes of the different symbols in Fig. 5.5), but it is also influenced by variation in local topography and microclimate (e.g. some areas are particularly prone to flooding following heavy rainfall) (Suryahadiselim and Gruner 1985; Gulland and Fox 1992).

Since the density of infective larvae varies between areas, sheep feeding in these different areas face different parasitological threats, and we would expect this to be reflected in their faecal egg counts. Current analyses suggest that although there is consistent spatial variation in FEC for both males and females, this is only partially explained by variation in the local density of infective larvae (Fig. 5.5c and d). Other factors likely to influence spatial variation in parasitism rates include variation in the quality of the forage available in the different areas, stocking densities, and spatial differences in the 'quality' of the sheep themselves (i.e. genotype, body condition, previous parasitological history, etc).


(a) within years
(b) between years
Signals Meadow
West Field
Ruaival
Mid Field
West Field
Pasture larval count (L3/kg DM grass)
0
1000
2000
3000
4000
Jan Mar May Jul Sep Nov
Feb Apr Jun Aug Oct Dec
Month
1991 1992 1993 1994 1995 1996 1997 1998
Year
(c) females
(d) males
Geometric mean faecal egg count (eggs/g faeces)
70
120
170
220
270
Mid Field
Signals Meadow
Gun Meadow
Old Village
West Field
Ruaival
West Meadow
0
200
400
600
800
1000
100 1200 2300 3400
Pasture larval count in Summer (L3/kg DM grass)

EARLY DEVELOPMENT OF PARASITISM

A recurrent theme throughout the remainder of this chapter is the striking difference between the two sexes, both in their resistance to parasite accumulation and in the impact of parasites on their survival and reproduction. As this next section shows, these sex differences become apparent early on in life.

The eggs of strongyle parasites first appear in the faeces of lambs when they are about 45 days old (Gulland and Fox 1992; Boyd 1999) (Fig. 5.6). Since the *prepatent period* (i.e. the time from infection until parasite eggs first appear in the faeces) for *T. circumcincta* is around of 17 days, this means that infective larvae are first acquired when the lambs are less than one month old. Parasite loads (and FEC) gradually increase over the next few months, plateauing in midsummer (August–September). In most years, FEC then declines through the autumn and early winter, but in high-density years (like the one illustrated in Fig. 5.6) FEC may remain high thoughout this period (see also Gulland and Fox 1992). In lambs of both sexes, FEC rises again towards the end of winter when forage is in short supply, and again this trend is particularly marked in high-density years (see below). A sex difference in FEC first appears when animals are just ten weeks

FIG. 5.5. Spatial variation in parasitism. Spatial variation in the number of infective L3 larvae on the pasture is shown (a) within years and (b) between years; spatial variation in faecal egg counts is shown for (c) females and (d) males. In (a) and (b), the lines join L3 counts for one of five different areas within Village Bay. In (a), all of the data are for 1993; in (b), the data are for August L3 counts in 1992, 1993 and 1996 (in all other years, L3 counts were not separated into different areas). In (c) and (d), the symbols and bars are the geometric mean $\pm$ standard error faecal egg counts for animals occupying one of seven different areas within Village Bay in Summer 1993 (the five areas illustrated in (a) and (b), plus two others). Note that the *y*-axis scales differ in (c) and (d). In all four figures, symbol size reflects average sheep density in the different areas. These data show that there is consistent spatial variation in the density of L3 on the pasture both within and between years and although there is spatial variation in faecal egg counts, this is only partly explained by the density of infective L3 larvae on the pasture (neither regression line is statistically significant; $p > 0.1$).

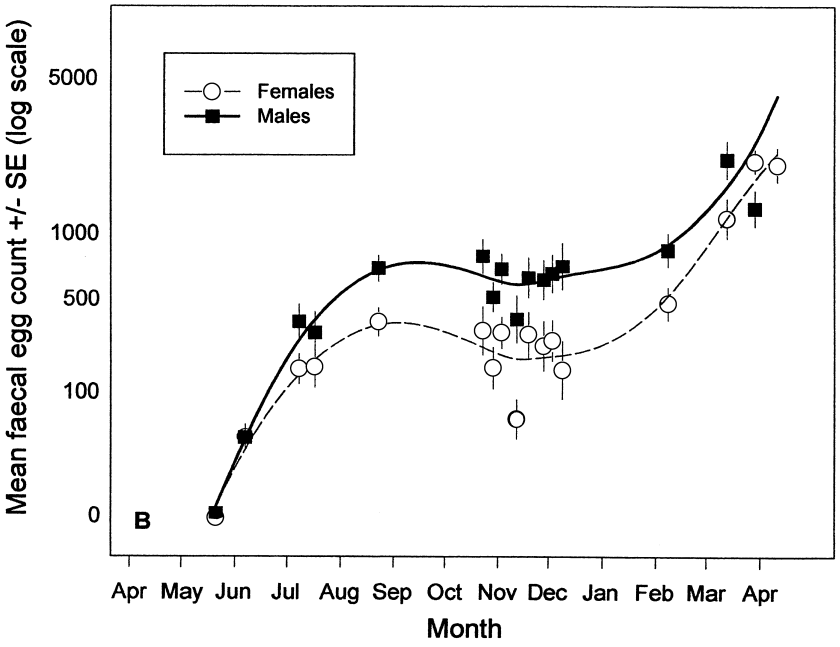


FIG. 5.6. Early development of parasitism in Soay sheep on St Kilda. The data are serial faecal egg counts collected from a single cohort of animals born in April 1994 (Boyd 1999). Females are the open symbols and males the closed symbols. Fitted lines are smoothing splines. B = mean birth date.

old (Fig. 5.6); at this time, the average FEC for ram lambs is more than 60% higher than that for ewe lambs (geometric means $\pm$ approx. standard errors: 487 ± 89 (males) versus 293 ± 51 (females); $t = 2.03$, df $=$ 81, $p = 0.046$). By August this difference has increased (828 ± 106 versus 486 ± 72), and by the beginning of October, male FEC is more than double that of females (929 ± 152 versus 443 ± 114; $t = 2.44$, df $=$ 39, $p = 0.019$).

The higher FEC of ram lambs could occur because parasites have a higher rate of establishment in males than females, or because established worms survive longer, or because parasites grow larger and hence females are more fecund. Our data do not allow us to distinguish between these possibilities, but experimental infections on the mainland suggest that a similar sex difference observed in domestic sheep is primarily due to a sex difference in the establishment rates

of worms (Dobson 1964; Knight *et al.* 1972), especially for sheep on low-protein diets (Bawden 1969). The effects of host sex on parasite survival and fecundity are less clear (Dobson 1964; Frayha *et al.* 1971; Molan and James 1984).

The importance of sex differences in parasite establishment rates is indicated by the results of an experiment we conducted on St Kilda in August 1995 (K. Wilson *et al*. unpublished data). In this experiment, one group of lambs was given a short-acting anthelminthic 'drench' to remove its parasites, whereas a control group was given a placebo. The parasitism rates of the two groups of lambs were then monitored during the following autumn to determine how quickly the lambs re-acquired parasites. We found that by the first week of October, when faecal egg counts were first taken, the FEC of ram lambs in the treated group were not significantly different from those of animals in the control group (drenched: 293 $\pm$ 88, control: 536 $\pm$ 120; $t = 1.631$, df $= 26$, $p = 0.115$) and prevalences were approximately the same (Fig. 5.7). In contrast, the FEC of treated ewe lambs were substantially lower in early October than those of control animals (drenched: 60 $\pm$ 29, control: 392 $\pm$ 90; $t = 4.393$, df $= 30$, $p < 0.001$), and were not comparable until early December (week 9). A closer look at these data indicated that although the FEC of *infected* females were similar in both the drenched and control animals throughout the period October–December (data not shown), the prevalences were markedly different until December (weeks 9 and 10) (Fig. 5.7). This observation strongly suggests that it is the rate of establishment of new infections that differs most between the two sexes at this age, rather than the survival rates or fecundities of worms in existing infections. This result is consistent with those from a study conducted by Gulland (1991) using five-month-old captive Soay and black-face sheep which showed that, for a given larval intake rate, the percentage of larvae establishing was significantly higher for males than females ($F_{1,19}$, $= 5.40$, $p < 0.05$). This result was independent of breed, though Soays had proportionately lower establishment rates than blackface sheep ($F_{1,19}$, $= 6.17$, $p < 0.05$), suggesting that they may be genetically more resistant to *T. circumcincta* than black-face sheep.

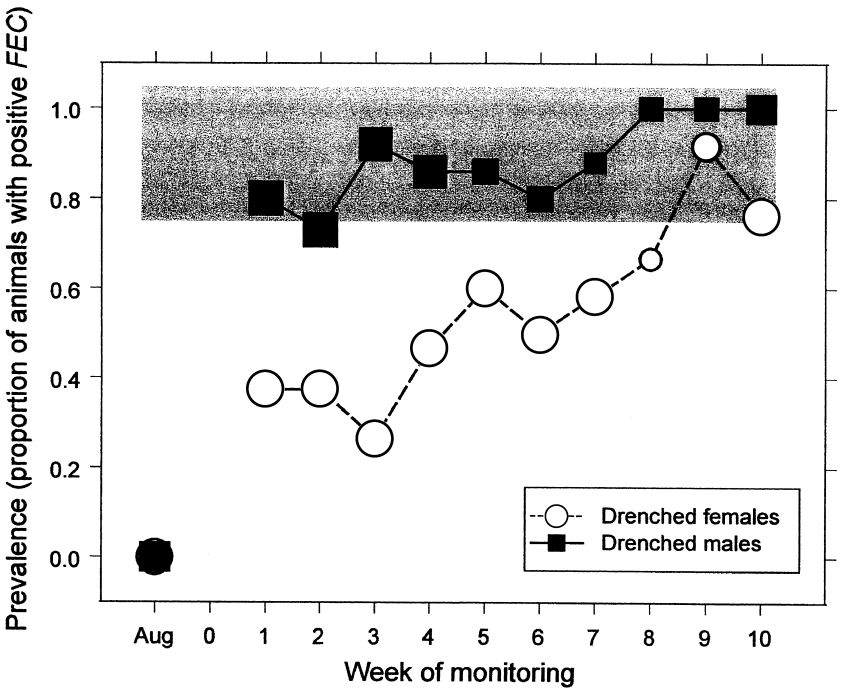


FIG. 5.7. Effect of sex on the rate of parasite establishment following drenching. Lambs were given an anthelminthic drench in August when they were approximately five months old and the prevalence of infection in both sexes was 100%. Drenching removed all parasites and faecal egg counts of all treated lambs were zero immediately following treatment. By week 1 of monitoring (first week of October), more than 80% of ram lambs had positive faecal egg counts, compared with just 40% of ewe lambs; by week 10 (second week of December), both sexes had prevalences of 80–100%. During the sampling period (October–December), the average prevalence of infection in control animals was 80–100% (indicated by the shading in the figure). Symbol size is positively correlated with sample size.

SEASONAL PATTERNS OF PARASITISM

Seasonal variation in FEC is determined by temporal trends in both the number of infective larvae available for ingestion on the pasture (as discussed above) and the immunological status of the sheep (determined by the sheep's age, nutritional plane and levels of immunologically depressive hormones). Both of these factors (as well as attributes of the parasites themselves) interact to determine temporal changes in parasite arrestment, development, mortality and

fecundity. Whilst we know little at present about how the immunological status of Soays varies through the year, we can measure seasonal variation in FEC and compare this with the variation observed in domestic sheep, where the immunology is better understood.

We begin by comparing the seasonal patterns observed for ewes on St Kilda with those seen in female Scottish hill sheep on the mainland (Morgan *et al.* 1950, 1951) (we restrict the comparison to females, because there is little comparable data available for males on the mainland). As Fig. 5.8 illustrates, the temporal trends in the two populations are broadly similar (Morgan *et al.* 1951; Gulland and

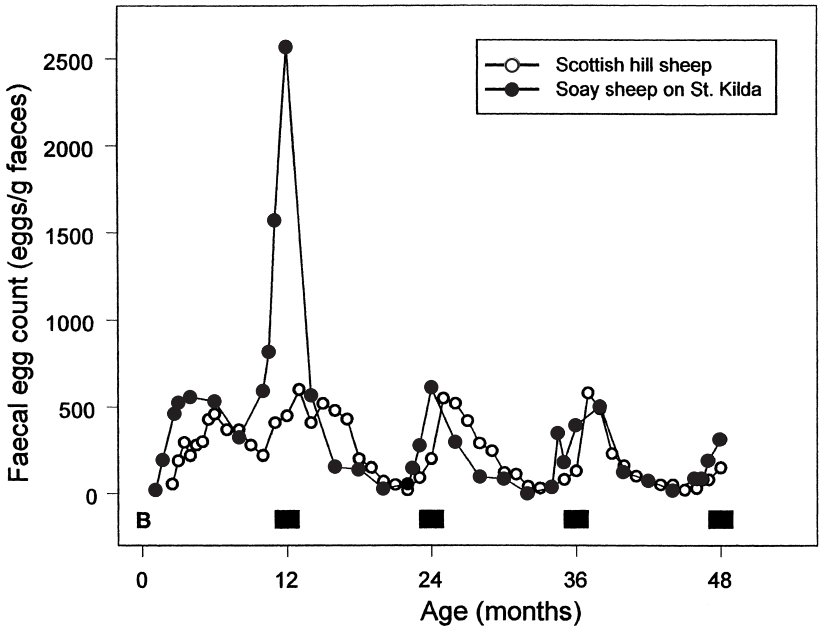
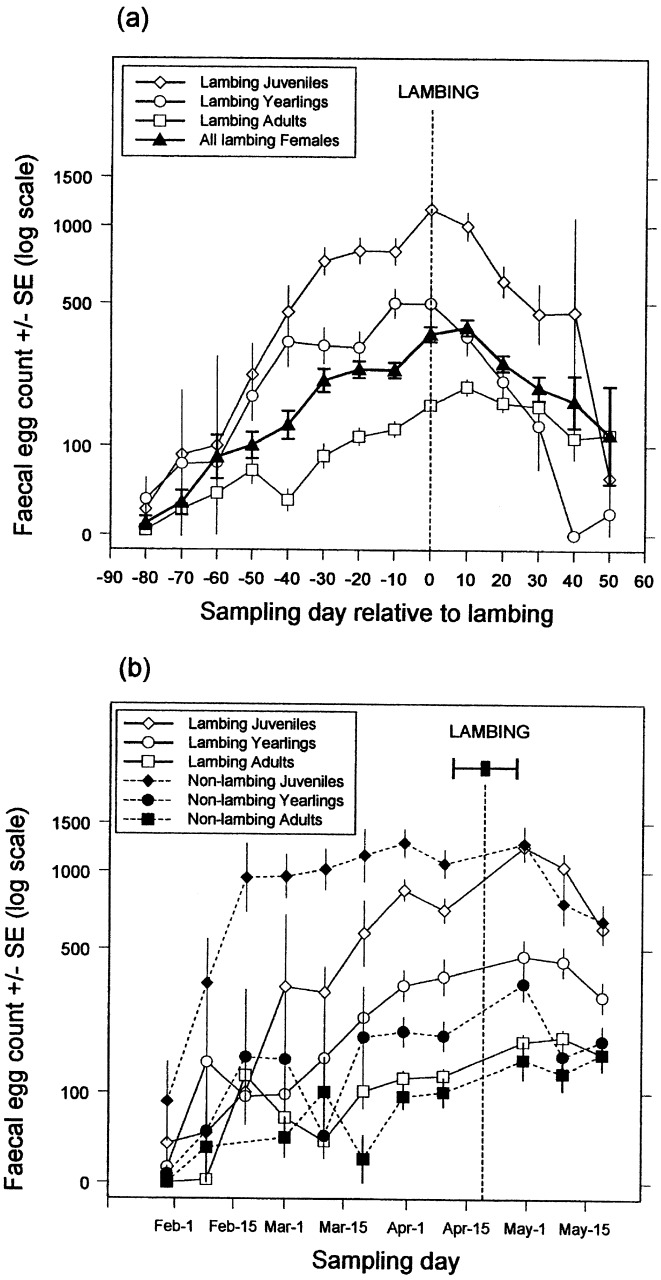


FIG. 5.8. Temporal variation in faecal egg counts in Soay sheep on St Kilda (closed symbols) and hill sheep on the mainland Scotland (open symbols). The data are serial egg counts collected from animals over a single year and joined together to indicate how the pattern changes over the first four years of life (beginning at time B). The Soay data are for animals sampled between August 1993 and April 1994 (except the first four points which are from ewe lambs May–July 1995). The hill sheep data are from Morgan *et al*. (1950). For both populations, lambing occurs in April (indicated by the solid boxes). Soay ewes often lamb for the first time when they are twelve months old, whereas Scottish hill sheep do not lamb until they are twenty-four months old. Note the arithmetic scale.

Fox 1992). In both populations, lambs first acquire strongyle parasites within one or two months of birth and parasite loads continue to increase during the first five to six months of life. On St Kilda, faecal egg counts first peak in August, when the sheep are about five months old, and they generally remain high until late autumn or early winter (Gulland and Fox 1992; Boyd 1999) (see also Fig. 5.6). On the mainland, a similar pattern is observed but the peak in FEC is delayed by one to two months. In both populations, from the second calendar year onwards, temporal variation in ewe parasitism is dominated by a characteristic increase in FEC during April–June (Fig. 5.8). Because this spring rise in FEC usually coincides with lambing, it is often referred to as the *peri-parturient rise* (or PPR).

On St Kilda, the peak of the PPR occurs within about ten days of parturition (Fig. 5.9a), whereas in domestic sheep it generally peaks around two to four weeks later (e.g. Crofton 1954; Brunsdon 1970) (cf. the open and closed symbols in Fig. 5.8). The difference in the timings of these two peaks may be associated with the absence of *Haemonchus contortus*, which is prevalent in most mainland sheep populations (Procter and Gibbs 1968; Blitz and Gibbs 1972). The importance of pregnancy and/or lactation in generating the PPR is illustrated by the relative magnitudes of the PPRs of Scottish hill sheep and St Kilda Soays in their first spring: at this time, most Soay ewes are lambing

FIG. 5.9. The peri-parturient rise in faecal egg counts in Soay sheep on St Kilda. Data are mean ± standard error faecal egg counts for ten-day periods (a) in relation to the day that each female lambed (day 0) and (b) in relation to calendar date (note log scale). Average lambing day ± SD was 20 April ± 8 days (range 31 March – 18 May), and did not vary between the age classes ($F = 0.573$, df = 2,404, $p = 0.449$). In both figures, data are for the springs of 1989–95. In (a) data are only for females that gave birth to singletons and in (b) a comparison is made of the temporal patterns observed in females that lambed that year (solid symbols) and those that did not (open symbols). Note in (a) that for females in the two youngest age classes (one and two year olds) the peak FEC is during the week of lambing; whereas for older sheep the PPR peaks during the two to three weeks following lambing. Note in (b) that for yearlings, females that fail to lamb have faecal egg counts that tend to be higher and peak sooner than females that do lamb successfully.


(a)
Lambing Juveniles
Lambing Yearlings
Lambing Adults
All lambing Females
LAMBING
Faecal egg count +/- SE (log scale)
1500
1000
500
100
0
-90 -80 -70 -60 -50 -40 -30 -20 -10 0 10 20 30 40 50 60
Sampling day relative to lambing
(b)
Lambing Juveniles
Lambing Yearlings
Lambing Adults
Non-lambing Juveniles
Non-lambing Yearlings
Non-lambing Adults
LAMBING
Faecal egg count +/- SE (log scale)
1500
1000
500
100
0
Feb-1 Feb-15 Mar-1 Mar-15 Apr-1 Apr-15 May-1 May-15
Sampling day

for the first time (Clutton-Brock *et al*. 1992) and this is associated with a very high PPR, whereas mainland hill sheep do not lamb until the following year (Morgan *et al.* 1950; Paver 1955) and their first PPR is considerably lower, though still evident (Fig. 5.8). Thus, variation in the fecundity schedules of sheep on St Kilda and the mainland is reflected in their seasonal patterns of parasitism.

Despite being of considerable applied interest, the proximate causes of the PPR remain unclear, even in domestic sheep (Parnell *et al.* 1954; Field *et al.* 1960; Brunsdon 1964). Early studies identified an association between the PPR and lactation and this was later confirmed experimentally: when lambs were removed from their mothers at a very young age, these non-suckling females failed to exhibit the dramatic rise in FEC following lambing that was observed in lactating females that retained their lambs (Connan 1968; Brunsdon and Vlassoff 1971). No such experiment has yet been conducted on St Kilda, but it is interesting to observe that yearling females that fail to lamb, or lose their lamb at a young age, tend to have *higher* FEC than those which raise a lamb successfully (Fig. 5.9b). This is probably because young females that are in poor condition have both high parasite loads and low conception and weaning rates, rather than because there is no parasitological cost of bearing and suckling a lamb (see section 5.4 below). This observation emphasises the advantages of experimental studies for examining these issues.

The PPR occurs at the end of winter and beginning of spring, when fresh vegetation is only just becoming available to the sheep (Procter and Gibbs 1968; Brunsdon 1970). On St Kilda, this period coincides with the time of lowest body condition and peak over-winter mortality for the sheep (Gulland 1992; Clutton-Brock *et al*. 1997a). Thus, it seems likely that at least part of the rise in FEC is due to the stresses associated with food shortage. A number of studies of strongyle infections of domestic sheep suggest that the establishment and pathogenicity of parasites is greater in malnourished hosts (e.g. Taylor 1934; Brunsdon 1962; Gordon 1964), although the relationship between nutrition and susceptibility to infection may not be a simple one (Abbott *et al.* 1985; Abbott and Holmes 1990). The importance of malnutrition, and other sex-independent mechanisms, in generating the PPR is highlighted

by a comparison of the seasonal patterns in FEC during 1989 and 1990 (Gulland and Fox 1992). In spring 1989, which followed a high-density winter when nearly 60% of the sheep died due to malnutrition (Gulland 1992), both males and females exhibited a marked rise in FEC. Conversely, in spring 1990, when the winter population density was much lower, the spring rise in males was absent. Clearly, the male rise in 1989 was not due to any factors associated with lambing and must have been linked to the high population density and food shortage of that year (Gulland and Fox 1992).

In males, there is little seasonal variation in FEC except during late winter and early spring in high-density years, when FEC increases dramatically in response to food shortages (Gulland and Fox 1992).

PATTERNS IN PARASITISM ASSOCIATED WITH HOST DEMOGRAPHY

In this section, we examine variation in FEC (and prevalence) in relation to host age and population density, and how these patterns differ between the two sexes. We restrict our discussion to variation in summer FEC for which we have comparable data for both males and females.

In females, there is a striking decline in August FEC with age (Fig. 5.10a), and statistical models distinguish between four distinct age classes with successively lower faecal egg counts: lambs (four months old), yearlings (sixteen months), two-year olds (twenty-eight months) and older animals (over forty months). When the female population is characterised in the same way as in Coulson *et al*. (2001), the statistical model explains slightly less variation in FEC, but there is a similar monotonic decline with age (lambs > yearlings > prime-age females > seniles); there is no evidence for an increase in FEC in senile females that could contribute to the higher mortality of this age class. Age-related declines in the levels of parasitism are often interpreted as evidence for *acquired immunity*, i.e. resistance due to the development of protective immunity in response to the accumulation of exposure to parasite antigens. However, interpretation of such trends must be guarded because there are several alternative explanations for them (Anderson 1993; Wilson *et al*. 2002). For example, a decline in FEC with age might simply be due to the fact that as

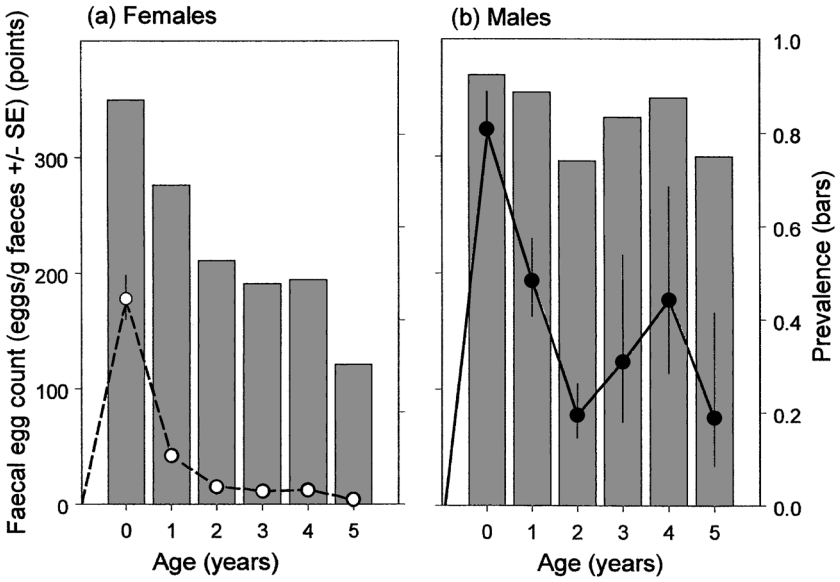


FIG. 5.10. Sex differences in faecal egg counts in Soay sheep on St Kilda. The symbols joined by lines are age-specific geometric mean ± SE summer faecal egg counts for (a) females and (b) males. The bars show the mean prevalence (proportion infected). Female faecal egg counts decline during each year of life for at least the first four years; male egg counts decline over the first year or two, but not subsequently. Females have lower prevalences than males, except when they are lambs.

animals age they get bigger, produce more faeces and their parasite eggs become 'diluted'. Alternatively, it might be that younger individuals are exposed to higher levels of infective larvae because of where, or how, they forage. A third possibility is that animals with high FEC are more likely to die from their parasites and so generate a decline in parasitism with age as the heavily infected individuals are selected out of the population. Finally, younger animals may have higher innate susceptibilities to infection regardless of their previous exposure. We have reviewed these competing hypotheses and conclude that although innate responses are likely to be important for Soay sheep during their first year of life, the subsequent decline in female FEC with age is primarily due to acquired immunity. Experimental infections with domestic sheep further support this assertion: protective

immunity begins to develop when lambs are three to six months old (depending on genetic background) (Windon *et al.* 1980; Stear *et al.* 1996) and, subsequently, immunity increases with the animal's experience of infection.

In males, prevalence remains high throughout life, and any decline in August FEC with age generally extends only to the yearling stage (Fig. 5.10b); the faecal egg counts of animals aged one year and older are statistically indistinguishable from each other (unpublished analysis). The difference in FEC between males in the two youngest age classes is probably due to the fact that FEC is first measured when the animals are just four months old, before any protective immunity has developed; the data suggest that acquired immunity does not increase much beyond the yearling stage. The pattern for male Soays is similar to that observed in lungworm-infected bighorn ewes in North America, where lambs and adults differ in their faecal larval counts but age has no effect on the faecal larval counts of adult females (Festa-Bianchet 1991b).

Our analyses show that there is considerable year-to-year variation in parasitism rates in both sexes, much of which can be explained by adult population density (Fig. 5.11). This contrasts with the situation observed in bighorn sheep, where faecal larval counts of lungworm showed little variation from one year to the next, over an eight-year period (Festa-Bianchet 1991b). For female Soays, the density of infective larvae on the pasture explains the remainder of the variation in August FEC, whereas for males L3 density appears to have little impact of August faecal egg count. Thus, in females especially, year-to-year differences in parasitism can be attributed to the combined effects of sheep and infective larval density. This suggests that FEC is a function of the population's age-structure: the number of lambs in the population sets the upper limit to the density of L3 on the pasture, and the number of adult sheep determines food availability and immunocompetence (because adults consume most of the vegetation; see Chapter 4).

Despite the considerable variation in faecal egg counts between years, individual animals show consistent relative levels of parasitism. Across years, female faecal egg counts are highly repeatable: overall,

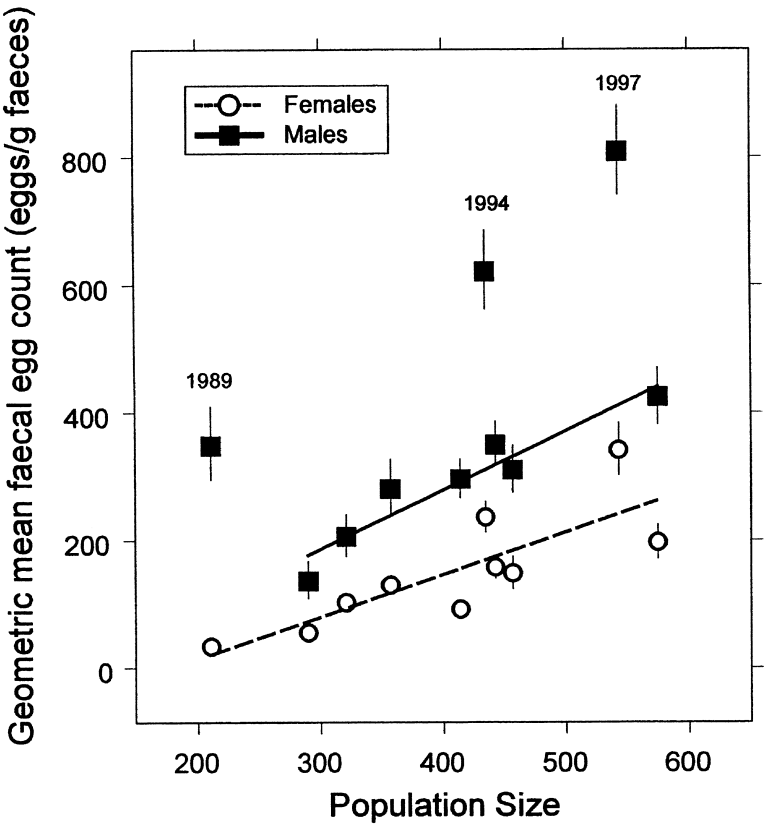


FIG. 5.11. The relationship between population density and faecal egg count in Soay sheep on St Kilda. The symbols and bars are mean ± SE faecal egg counts for all females (open circles) and males (solid squares). Overall, there is a significant positive relationship between population density and faecal egg count for females ($y = -126.87 + 0.6953x$, $r^2 = 0.631$, $F_{1,8} = 13.72$, $p = 0.0060$), but not for males ($y = -133.66 + 1.3093x$, $r^2 = 0.369$, $F_{1,8} = 4.67$, $p = 0.062$), probably due to unusually high egg counts for males in 1989, 1994 and 1997. The regression for females is shown by the dashed line; the solid line is the regression for males, excluding 1989, 1994 and 1997.

the mean repeatability (± SE) is $r = 0.45 \pm 0.04$ ($p < 0.001$) and, after accounting for the decline in FEC with age and year-to-year variation in average FEC, the mean repeatability increases to $r = 0.58 \pm 0.03$ ($p < 0.001$). Thus, across years, approximately 45% of the variation in female FEC is due to differences between individual ewes, and once

age and sampling year effects have been accounted for, the amount of variation explained increases to 58%. This compares with a value of $r = 0.25$ ($n = 238$ female–years, $p < 0.001$) for the repeatability of faecal lungworm counts from bighorn ewes (Festa-Bianchet 1991b). The repeatability of FEC for male Soays is much lower overall ($r = 0.14 \pm 0.09$; $p = 0.074$) but, after accounting for the decline in FEC with age and year-to-year variation in average FEC, the mean repeatability increases significantly ($r = 0.42 \pm 0.07$; $p < 0.001$). These results are important because the repeatability of a trait sets an upper limit to its heritability (Falconer and Mackay 1996).

GENETIC VARIATION AND THE HERITABILITY OF PARASITISM

Heterogeneities in parasitism rates are important because they may influence the stability of the host–parasite interaction; as a rule, the more extensive the heterogeneities, the more stable the interaction (Anderson and May 1978, 1979; May and Anderson 1978, 1979). Genetic heterogeneities are likely to be particularly important in this respect for two reasons: first, because they may evolve through time and, second, because (via antagonistic pleiotropy) they may be associated with variation in other important traits, such as growth rates (May and Anderson 1983; Read *et al*. 1995). Here, we examine the evidence for heritable variation in parasitism on St Kilda and associated genetic correlations with other important traits. Chapter 8 discusses the evidence for variation in parasite loads associated with specific genetic markers, such as particular alleles of adenosine deaminase and variation within the Major Histocompatibility Complex.

A number of studies of domestic sheep have estimated the heritability of FEC (Table 5.3), and published estimates range between 0.13 ± 0.07 (McEwen *et al*. 1992) and 0.53 ± 0.15 (Baker *et al*. 1991). Recently, the heritability of FEC in Soay sheep on St Kilda was estimated using pedigree information and multiple trait, restricted-estimate maximum-likelihood models implemented by the program VCE (Groeneveld and Kovac 1990; Groeneveld 1995). After removing sources of fixed effect variation (due to age class, sampling year, cohort, twin status and date), heritability estimates ranged between $h^2 = 0.11 \pm 0.02$, for male FEC in the summer, and $h^2 = 0.14 \pm 0.01$,

Table 5.3. *Heritability of nematode parasitism in (a) domestic sheep breeds*

Sheep breed	Infection type	Worm species	$h^2 \pm$ SE	Reference
Romney	Natural	Mixed	0.13 ± 0.07	McEwan *et al.* (1992)
Romney	Natural	*Haemonchus contortus*	0.21 ± 0.05	Bisset *et al.* (1992)
Merino	Experimental	*H. contortus*	0.23 ± 0.03	Woolaston and Piper (1996)
Romney	Natural	*Nematodirus* spp.	0.25 ± 0.09	McEwan *et al.* (1992)
Romney	Experimental	Mixed	0.27 ± 0.07	Bisset *et al.* (1994)
Red Maasai	Natural	Mixed	0.33 ± 0.10	Baker *et al.* (1994)
Scottish Black-face	Experimental	*Teladorsagia circumcincta*	0.33 ± 0.15	Bishop *et al.* (1996)
Merino	Experimental	*H. contortus*	0.34 ± 0.10	Albers *et al.* (1987)
Romney	Natural	Mixed	0.34 ± 0.19	Watson *et al.* (1986)
Romney	Natural	Mixed	0.39 ± 0.13	Morris *et al.* (1993)
Merino	Experimental	*T. colubriformis*	0.41 ± 0.04	Woolaston *et al.* (1991)
Merino	Experimental	Mixed	0.42 ± 0.14	Cummins *et al.* (1991)
Merino	Experimental	*H. contortus*	0.49 ± 0.17	Sreter *et al.* (1994)
Romney	Natural	Mixed	0.53 ± 0.15	Baker *et al.* (1991)

Source: For details, see Smith (1996).

Table 5.4. *Heritability of nematode parasitism[a] in Soay sheep on St Kilda*

Model	n^b	Mean $h^2 \pm$ SE	p
Males (summer)	687/493	0.11 ± 0.02	<0.001
Males (autumn)	836/306	0.13 ± 0.03	<0.001
Females (summer)	1250/576	0.13 ± 0.01	<0.001
Females (spring)	2294/348	0.14 ± 0.01	<0.001

[a]Nematode parasitism was measured as $\log_e$ faecal egg count.
[b]Number of observations/individuals.

for female FEC in the spring (Table 5.4) (Coltman *et al*. 2001a) . Thus, although all heritability estimates for parasite resistance were significantly different from zero ($p < 0.001$), they were towards the lower end of the distribution of previously published estimates (Table 5.3), and approximately 50% lower than previous estimates from the same population (mean $h^2 = 0.26$), which used parent–offspring regression and sibling analyses (Smith *et al.* 1999).

There are several reasons why the heritability estimates generated by Coltman *et al*. (2001a) might differ from those generated in other studies of sheep. First, the method of analysis used by Coltman *et al*. (2001a) was far more robust than those employed in most other studies, in that it makes better use of the pedigree data, uses larger sample sizes, and generates much smaller standard errors. Second, many of the previously published heritability estimates are likely to be inflated by maternal effects, which were significant on St Kilda and were estimated separately by Coltman *et al*. (2001a). Finally, a direct comparison between the heritability estimates from St Kilda and elsewhere may be inappropriate because the sheep used in the mainland studies were generally experiencing regular drug treatment regimes whereas, on St Kilda, anthelminthics are rarely used and so sheep are usually exposed to continual mixed infections.

Theoretical considerations would lead us to predict that traits closely related to fitness (such as parasite resistance) should be subject to strong selection and that this should deplete levels of additive genetic variation (Gustafsson 1986). In line with this expectation, the

heritability of parasite resistance in Soay sheep was significantly lower than most morphometric traits (e.g. in females, the heritabilities of body weight and hindleg length were $h^2 = 0.28 \pm 0.02$ and $h^2 = 0.35 \pm 0.21$, respectively, whereas the heritability of summer FEC was $h^2 = 0.13 \pm 0.01$). However, contrary to expectation, it does not appear that this was a consequence of the depletion of additive genetic variance due to selection, because parasite resistance traits had considerable additive genetic variance when measured by the coefficient of additive genetic variance. Instead the low heritability of parasite resistance was a consequence of high residual variance (Coltman *et al*. 2001a).

So, high levels of additive genetic variance for parasite resistance are maintained in the Soay sheep population, despite strong selection (see below). This leads to the obvious question of what is the mechanism maintaining this variation? One possibility is antagonistic pleiotropy, whereby negative genetic correlations across traits, such as parasite resistance and growth rate, result in a genetic trade-off between the two traits. However, there is no evidence, at present, for a genetic trade-off maintaining additive genetic variation in parasite resistance. Instead, Coltman *et al*. (2001a) found that there were *positive* genetic correlations among six of the eight pairwise comparisons of morphometric traits and parasite resistance traits (Table 5.5), indicating that selection on morphometric traits indirectly reinforces selection in favour of parasite resistance. This indicates that growth and parasite resistance are not traded off, but rather that genetically resistant individuals experience better growth. Other potential explanations for the maintenance of additive genetic variation in parasite resistance in this population are discussed by Coltman *et al*. (2001a) and in Chapter 8.

5.4 Costs of parasitism

Many studies have demonstrated a significant negative impact of parasites on host survival, reproduction and growth (see reviews by Grenfell and Gulland 1995; Gulland 1995). In Soay sheep, there is a consistent negative correlation between over-winter survival and faecal egg count (Illius *et al*. 1995; Coltman *et al.* 1999b; Milner *et al.* 1999b) and a number of experimental studies have shown that when parasites

Table 5.5. *Genetic correlations between nematode parasitism and morphometric traits*

Traits[a]	Hindleg length	Weight	FEC (summer)	FEC (spring/autumn)[b]
Hindleg length	–	$+0.78 \pm 0.05$ $p < 0.001$	-0.23 ± 0.08 $p < 0.01$	-0.31 ± 0.13 $p < 0.05$
Weight	$+0.80 \pm 0.02$ $p < 0.001$	–	-0.30 ± 0.25 ns	-0.39 ± 0.19 $p < 0.05$
FEC(summer)	-0.26 ± 0.02 $p < 0.001$	-0.05 ± 0.04 ns	–	$+0.71 \pm 0.09$ $p < 0.001$
FEC (spring/ autumn)	-0.22 ± 0.04 $p < 0.001$	-0.14 ± 0.04 $p < 0.001$	$+0.28 \pm 0.04$ $p < 0.001$	–

[a]Values above the diagonal are for males, those below the diagonal are for females.
[b]FEC spring/autumn refers to faecal egg count in spring (for females only) and autumn (for males only).
Source: For details see Coltman *et al.* (2001a).

are removed from Soay sheep survival rate is enhanced. Prior to the population crash of 1988–9, Gulland (1992) administered slow-release intra-rumenal anthelmintic boluses to 52 Soay sheep (19 male lambs, 12 female yearlings, 14 male yearlings and 7 male two-year-olds) to chemically remove their worm burdens. These boluses are designed to release 42 mg of albendazole per day for at least 100 days, but field observations suggest that their efficacy extends well beyond this time. The idea was to determine whether treated animals survived better than the controls. During the late winter of 1989, over 70% of the sheep died and there was no detectable difference in the proportional mortality of sheep in the treated (44/52–85%) and control (34/40–85%) groups. However, the daily survival rate of the treated animals was significantly higher than that of the controls (Gulland 1992).

A second bolus experiment was conducted prior to the winter of 1991–2 (Gulland *et al*. 1993), when the overall mortality rate was much lower (44%). This time, anthelminthic boluses were given to 55 sheep

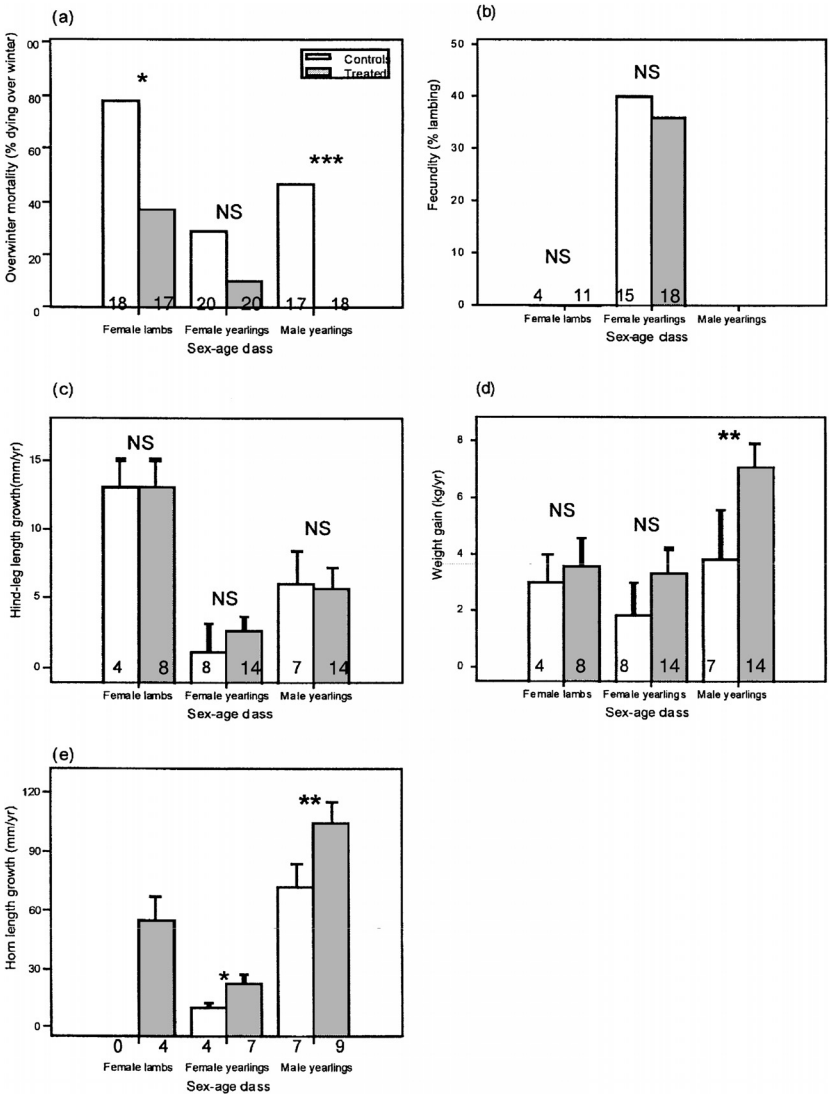
(17 female lambs, 20 female yearlings and 18 male yearlings). In all three sex–age classes, the mortality rates of treated animals was lower than that of the controls and, for female lambs and male yearlings, this difference was statistically significant (Fig. 5.12a). The most interesting comparison here is between the male and female yearlings; in females there was a relatively small reduction in mortality, from 6/21 (29%) animals in the control group to 2/20 (10%) in the treated, whereas in males there was a massive reduction in the number of animals dying, from 8/17 (47%) in the controls to 0/18 (0%) in the treated. Thus, it appears that parasites are a much more important mortality factor for males than females, and parasites might explain at least part of the male-biased mortality observed in adult Soays (Chapter 3 and section 5.5; see also Wilson *et al*. 2002).

The impact of parasites on fecundity is less clear. Although the probability of females lambing in spring tends to decrease as their FEC in the previous August increases (logistic regression analysis: yearlings: $b = -1.081$, $\chi^2_1 = 7.30$, $p = 0.007$; two-year-olds: $b = -0.441$, $\chi^2_1 = 0.54$, $p = 0.462$; adults: $b = -0.777$, $\chi^2_1 = 4.33$, $p = 0.037$), it appears that the main impact of parasites on fecundity is probably via their effect on female survival, since parasites disappear as an important

FIG. 5.12. Experimental analysis of the effects of parasites on fitness. The fitness components examined are (a) over-winter mortality, (b) fecundity, (c) skeletal (hindleg length) growth, (d) weight gain and (e) horn growth. The figure shows the results of an experiment in which treated animals (closed bars) were given an anthelminthic bolus to remove their gastrointestinal parasites and control animals (open bars) were matched for age and sex. Sex–age classes included in the experiment were female lambs, female yearlings and male yearlings (Gulland *et al*. 1993). Over-winter mortality is defined as the percentage of animals failing to survive until the following year; fecundity is defined as the percentage of females that survived and produced a lamb that lived to weaning age. Growth rates (means ± SE) were calculated for animals which survived for at least a year following bolusing and were captured and measured in the summers of both 1991 and 1992 (growth is defined as the difference between these two measurements). Numbers at the base of each bar are the number of animals in each group (note that in (e), only measurements for normal-horned animals are included). Significance levels: – analysis not possible, NS $p > 0.05$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

correlate of fecundity when we consider only those females that survived throughout the following year (yearlings: $b = -0.756$, $\chi_1^2 = 2.36$, $p = 0.124$; two-year-olds: $b = -0.009$, $\chi_1^2 = 0.001$, $p = 0.991$; adults: $b = -0.054$, $\chi_1^2 = 0.012$, $p = 0.911$).

A better way of examining parasite-induced reductions in fecundity is to manipulate parasite burden experimentally. Whilst a large-scale experimental manipulation of female parasite loads has yet to be



undertaken on St Kilda in a year of low mortality, we can examine the impact of parasite removal on the fecundity of those ewes that survived the population crash of 1991–2 (see above). This indicates that the fecundity of surviving yearlings was marginally enhanced by parasite removal, but not significantly so, and none of the ewes that were bolused as lambs produced offspring that survived to weaning age (Fig. 5.12b). Thus, in this year of high over-winter mortality, there was no evidence that parasites affected fecundity, except via their effects on ewe mortality. These results contrast with those from a recent study of Svalbard reindeer (*Rangifer tarandus platyrhynchus*) infected with gastrointestinal nematodes, in which it was found that the removal of parasites with anthelminthics resulted in a significant increase in fecundity of between 5% and 14% (Albon *et al*. 2002; Stien *et al*. 2002). Associated with this increased fecundity were significant increases in body mass and back-fat depth. Interestingly, however, there was no effect of anthelminthic treatment on the survival of female reindeer. It remains to be determined whether parasite-mediated reductions in fecundity occur on St Kilda in years of low over-winter mortality.

Across all age classes, there were significant negative phenotypic correlations between summer FEC and both body weight (males: $r = -0.15 \pm 0.05$, $p < 0.001$; females: $r = -0.20 \pm 0.04$, $p < 0.001$) and hindleg length (males: $r = -0.16 \pm 0.05$, $p < 0.001$; females: $r = -0.16 \pm 0.04$, $p < 0.001$); phenotypic correlations with autumn FEC (males) and spring FEC (females) were also negative and generally statistically significant (Coltman *et al*. 2001a). Genetic correlations between FEC and body weight/size were also significantly negative (Table 5.5 and above). When broken down by age class, a similar picture emerges, with strong negative correlations between August FEC and both hindleg length and body weight, particularly in young animals (Table 5.6). However, the empirical evidence that parasites affect sheep growth rates is poor, with only the correlation between FEC and male lamb hindleg growth proving statistically significant (Table 5.6).

Experimental evidence for an impact of parasites on growth and development comes from a comparison of the growth patterns of control and treated animals in the year following the bolus experiment

Table 5.6. *Correlations between log faecal egg count and (a) absolute size and (b) growth rate of hindleg length, body mass and horn length in female and male Soay sheep*

Measurement[a] and age-class	Females			Males		
	r	df	*p*	*r*	df	*p*
Absolute size						
Hindleg length						
Lambs	**−0.400**	**85**	**<0.001**	**−0.166**	**182**	**0.025**
Yearlings	−0.217	41	0.161	**−0.313**	**93**	**0.002**
Adults	−0.111	136	0.194	−0.096	94	0.354
Body weight						
Lambs	**−0.335**	**84**	**0.002**	**−0.163**	**178**	**0.029**
Yearlings	−0.214	40	0.173	**−0.321**	**91**	**0.002**
Adults	**−0.255**	**136**	**0.003**	−0.153	94	0.135
Horn length[b]						
Lambs	**−0.331**	**84**	**0.002**	−0.001	178	0.989
Yearlings	0.001	41	0.999	**−0.341**	**93**	**<0.001**
Adults	0.035	135	0.685	−0.019	95	0.850
Growth rate[c]						
Hindleg length						
Lambs	0.128	26	0.516	**−0.305**	**43**	**0.041**
Yearlings	0.235	23	0.258	−0.168	38	0.299
Adults	−0.032	59	0.808	−0.285	21	0.187
Body weight						
Lambs	0.176	26	0.370	−0.203	41	0.190
Yearlings	0.175	22	0.412	0.089	37	0.588
Adults	−0.203	58	0.120	−0.126	21	0.564
Horn length[b]						
Lambs	0.187	26	0.341	−0.242	43	0.108
Yearlings	−0.096	23	0.648	−0.234	38	0.146
Adults	0.133	59	0.308	−0.226	21	0.299

[a]Data cover measurements taken in the years 1985–95. Numbers in bold are significant at the 5% level.

[b]To ensure that correlations are comparable, only normal-horned individuals were included in the analysis.

[c]Growth rates are calculated as the difference between the trait size in the summer when the faecal egg count was taken and the trait size the following summer.

of 1991–2 (see above). This showed that although there was no significant effect of parasite removal on hindleg growth in any of the sex–age categories (Fig. 5.12c), yearlings given an anthelminthic bolus gained nearly twice as much body weight over the following year as animals in the control groups (Fig. 5.12d). For males, this difference was statistically significant (means $\pm$ SE: treated: 7.14 $\pm$ 0.65, control: 3.86 $\pm$ 0.67; $t = 3.148$, df $= 19$, one-tailed $p = 0.0026$), but for females it was not (treated: 3.36 $\pm$ 0.84, control: 1.75 $\pm$ 0.56; $t = 1.348$, df $=$ 20, one-tailed $p = 0.096$); female lambs that survived the winter had similar weight gains regardless of treatment. The greater weight gains of treated yearlings cannot be due to the effects of parasite-induced host mortality because this is likely to truncate the lower end of the weight distributions, and hence mask any relative weight gains in the treated group, rather than accentuate them. The effects of parasites on the growth of domestic sheep are well documented: reductions in weight gain of 20–60% have been recorded in domestic sheep (Barker 1973; Sykes and Coop 1976; Sykes *et al.* 1977), and a reduction of 37% was reported in sheep experimentally infected with just 1500 *T. circumcincta* per day (Coop *et al*. 1985). This reduction in weight gain is due to a combination of anorexia (which can result in a reduction in food intake of up to 20%) and decreased utilisation of ingested food.

Horn size is an important determinant of male mating success in Soay sheep (Chapters 7 and 9). In normal-horned animals, the horns are more than three times longer in adult males (mean $\pm$ SD: 392 $\pm$ 103 mm) than females (126 $\pm$ 62 mm). Both theoretical and empirical studies indicate that the expression of sexually selected traits, such as horns, may be commonly condition-dependent, such that animals in good condition invest relatively more sexually selected traits (Andersson 1994). As expected, we found that in all age classes, males with high faecal egg counts tended to have shorter horns and slower horn growth rates (Table 5.6), though only the correlation between horn length and FEC in yearlings was statistically significant. The 1991–2 bolus experiment showed that yearling males in the treated group grew their horns 25% longer than males in the control group (means $\pm$ SE: treated: 106.0 $\pm$ 7.1, control: 78.1 $\pm$ 7.7; $t = 2.64$, df $= 14$, one-tailed $p = 0.0096$; Fig. 5.12e). Within the treated group, horn

growth rate was significantly negatively correlated with FEC at the time of dosing ($r = -0.708$, df = 9, $p = 0.0147$; the equivalent correlations for hindleg growth and weight gain were non-significant, $p > 0.2$). Since parasite establishment rate in Soays is repeatable and males appear to be predisposed to high or low infection rates (K. Wilson, unpublished data), it seems likely that this result is a consequence of variation in amount of time males remained parasite-free following bolusing. An increase in horn growth was also observed in yearling females treated with anthelminthics (treated: 23.0 ± 4.8, control: 8.3 ± 1.5), though statistically this difference was less significant ($t = 2.23$, df = 9, one-tailed $p = 0.026$), and there was no correlation between horn growth rate and FEC at dosing ($r = -0.387$, df = 6, $p = 0.343$). Thus, parasites appear to restrict the opportunities for horn growth, particularly in males, but the magnitude of their effect is possibly not as great as it is for weight gain (in treated yearling males, weight gain increased by 85% whereas horn growth increased by only 25%). There are no comparable data for domestic sheep.

Another important determinant of male mating success in Soays is their ability to locate and defend oestrous ewes (Chapter 9). These activities are likely to require considerable stamina and a reduction in the amount of time allocated to maintenance activities such as feeding and resting. The effect of parasites on reproductive behaviour was determined by examining the correlation between FEC and male time-budgets during the 1996 rut. Male behaviour was classified as sexual, aggressive, feeding, moving or resting (Chapter 9). In both juveniles and older age classes, heavily parasitised males tended to spend less time engaged in sexual activity, fighting and moving, and spent more time feeding and resting. Although the overall correlations between FEC and the proportions of time engaged in sexual behaviour, feeding and aggression were all statistically significant ($|r| > 0.45$, $p < 0.002$), when time-budgets were analysed separately for each age class (so reducing sample sizes), only the correlations between sexual activity and FEC proved to be statistically significant (Fig. 5.13). Thus, it appears that males with high parasite loads spend less of their time performing sexual behaviours and more of their time feeding and resting, suggesting that parasites constrain opportunities for matings and may limit reproductive success. However, without manipulating

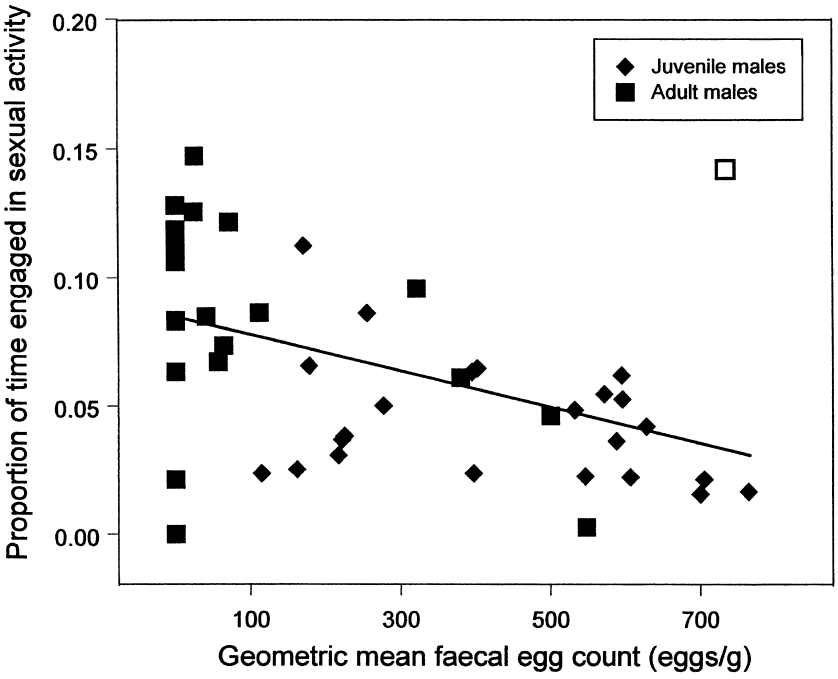


FIG. 5.13. Effects of parasites on male sexual behaviour. Data are for juvenile males (diamonds) and adult males (squares) for the period mid-October to mid-December 1996. The proportion of time engaged in sexual activity is averaged over the eight weeks of the rutting season, based on weekly averages (see Chapter 9). Geometric mean faecal egg counts were calculated over the same period. The overall correlation is statistically significant ($r = -0.458$, df = 43, one-tailed $p = 0.0008$), as are the correlations for juveniles alone ($r = -0.363$, df = 21, one-tailed $p = 0.044$) and adult males alone ($r = -0.213$, df = 19, one-tailed $p = 0.0231$), after excluding the outlier indicated by the open square.

parasite loads directly, the impact of parasites on sexual behaviour must remain speculative.

5.5 Discussion

ST KILDA AS A MODEL SYSTEM FOR PARASITIC INFECTIONS OF WILDLIFE

With one or two notable exceptions (e.g. Elton *et al.* 1931), the study of wildlife diseases was not really taken seriously by ecologists until the

mid to late 1970s, when theoretical and empirical studies demonstrated that parasites were not simply passive passengers in their hosts, but had the potential to be an important force in their evolution and ecology (Anderson and May 1978, 1979; May and Anderson 1978, 1979). Since then, studies of host–parasite interactions in the wild have slowly yielded interesting and important new insights into how animals cope in parasite-rich environments. One of the reasons why progress in this field has been slow is that the study of wildlife diseases presents a number of logistical problems that are often difficult or impossible to overcome. Paramount amongst these is simply being able to quantify parasite abundance. There are two main approaches available: either hosts are destructively sampled and the parasite burden is counted directly (e.g. Hudson 1986; Stein *et al*. in press) or parasite numbers are estimated using indirect measures such as faecal egg or larval counts (e.g. Wilson *et al*. 2002). The obvious downside of destructively sampling hosts is that it is not possible to collect longitudinal data using this method, and it is precisely this kind of information that is so useful for quantifying epidemiologically important attributes, such as parasite establishment rates and the strength of acquired immunity. Indirect measures of parasitism can be used to collect longitudinal data, but often their relationship to the actual parasite burden is unknown or inconsistent (e.g. lungworm larval counts in bighorn sheep; Festa-Bianchet 1991b), making their interpretation difficult or impossible.

Fortunately, there is a strong positive relationship between faecal egg count and worm burden in Soay sheep (Fig. 5.2) and this, combined with the fact that most of the sheep on St Kilda are individually identifiable, has allowed us to track the parasitism trajectories of known animals virtually from the day they are born until the day they die. These long time series are virtually unique in wildlife epidemiology and have revealed a number of important new insights. For example, it is now clear that the characteristic sex difference in parasite burdens seen in adult sheep first appears in lambs when they are just ten weeks old, and the difference in parasite loads between the sexes magnifies with age as females, but not males, develop a strong acquired immune response to their parasites (Figs. 5.6 and 5.10). These

longitudinal data have also allowed us to determine that the characteristic spring rise in faecal egg counts that occurs in pregnant ewes reaches its peak at almost exactly the time of parturition (fig. 5.9a). Both of these examples illustrate the power of individual-based parasite data to shed light on temporal epidemiological patterns. Similar patterns probably occur in other mammal populations elsewhere, but their detection relies on a level of data resolution that is rarely achievable in other systems because of logistical constraints.

Our studies on St Kilda have revealed that parasites may impact on the weight gain, weapon growth and mortality of their hosts (Fig. 5.12). They may also impact on the stamina of rutting males (Fig. 5.13) and ultimately limit the reproductive success of both sexes. However, the density of Soay sheep on St Kilda is unusually high and their parasite burdens tend to be large. So, are the costs of parasitism exerted on Soay sheep unusually high or are similar costs observed in other vertebrate–macroparasite systems? On the available evidence, it appears that in many instances the costs of parasitism are significant (Grenfell and Gulland 1995; Gulland 1995; see below). However, most of these studies are correlational, showing that individuals with high parasite loads tend to die sooner, produce fewer young and/or grow at a slower rate. Therefore, in general, these types of study may yield biased or incomplete estimates of the true costs of parasitism, because they are potentially confounded by variation in the quality of animals – an association between high parasite loads and low fitness may be due to the detrimental effects of parasites on host fitness, but may also be due to the fact that animals in poor condition not only die sooner and reproduce less but also have increased susceptibility to parasites. Studies on St Kilda, and elsewhere (e.g. Albon *et al*. 2002; Stien *et al*. in press), suggest that reliable estimates of the costs of parasitism may be gained only by experimentally reducing or enhancing parasite loads and observing the subsequent effects on the life-history characters of interest.

Anthelminthic dosing of sheep on St Kilda has also shown that the costs of parasitism may be revealed only under conditions of 'intermediate stress'. For example, the chemical removal of parasites had little impact on overall mortality during the 1988–9 population crash,

when over 70% of the sheep died regardless of parasite burden, and it is unlikely that parasite removal would have had much impact on the level of mortality that occurred during a low-density winter like 1989–90, when less than 5% of the sheep died. Only in 1991–2, a year of intermediate mortality (44%), was parasite removal reflected in enhanced survival (Fig. 5.12). The effects of parasites on fitness may also be expressed differently in different sex–age classes. For example, parasite removal improved the survival chances of female lambs, but not female yearlings (Fig. 5.12). Thus, as with studies of the cost of reproduction, it is becoming increasingly clear that the costs of parasitism are not fixed, but are 'context-specific' (Chapter 10). It is likely that similar context-specific costs are prevalent in other host–parasite interactions.

UNDERSTANDING NEMATODE INFECTIONS OF SOAY AND DOMESTIC SHEEP

A unique attribute of our studies of the epidemiology of nematode infections of Soay sheep is that we are able to draw parallels with comparable studies on their domestic counterparts. The seasonal pattern of parasitism in Soays on St Kilda is remarkably similar to that observed in domestic hill sheep in Scotland (Fig. 5.8). The major difference in their dynamics of infection is in the magnitude of the first spring rise in faecal egg counts, which is substantially larger in Soays than in Scottish hill sheep. However, this difference is probably explicable in terms of a difference in the life-histories of the two breeds of sheep (i.e. the precocial sexual maturity of the Soays). A similarity between the epidemiological patterns of wild and domestic sheep means that reasonable extrapolations can be made from one situation to the other. For instance, it is often extremely difficult to obtain reliable data on worm burdens from wildlife hosts because of restrictions on culling. Because there are fewer such restrictions for domestic animals, it is possible to use data on worm numbers from domestic animals to determine the likely patterns for wildlife hosts. Thus, it has been possible to construct epidemiological models for Soay sheep parameterised using FEC and worm burden data from Scottish hill sheep (Boyd 1999; B.T. Grenfell and K. Wilson,

unpublished data). Whilst such models allow us to analyse the early development of parasitism in Soays with a reasonable degree of accuracy, they fail to predict seasonal variation in FEC for animals older than about six to seven months. Understanding the deficiencies of the models is clearly a priority for future research.

Epidemiolgical studies of domestic animals may also gain valuable insights from studying the infections of wildlife since controlled studies of domestic animals are rarely able to simulate the same degree of stress on their subjects that is often observed in wildlife populations. For example, the spring rise in FEC observed in male Soays in high-density years (Gulland and Fox 1992) (Fig. 5.6) strongly indicates that a major factor generating the spring rise in females is nutritional stress. Most studies of domestic animals tend to concentrate on either young animals (less than one year old) or well-fed adult females (breeding stock). The absence of studies of adult male domestic sheep means that an apparent 'peri-parturient rise' in males has never been recorded in them. This, combined with the understandable reluctance to impose extreme food rationing on pregnant ewes, has meant that the importance of nutrition in generating the spring rise in the FEC of domestic sheep has probably been underestimated.

Concurrence of the epidemiological patterns observed in wild and domestic sheep is not restricted to the parasite stages living within their hosts; they also extend to the free-living stage on the pasture. The Soays provide good evidence for two patterns regularly observed in studies of domestic sheep parasites: first, the biphasic pattern in L3 counts, with one peak in late spring and the other in midsummer (Fig. 5.4a); second, the positive relationship between stocking density and the magnitude of the midsummer peak (Fig. 5.4b). As observed in these other studies, the relationship between stocking density and L3 density becomes less clear at other times of year, probably due to the effects of temperature, rainfall and humidity. On St Kilda, the density of infective larvae on the pasture shows high spatial repeatability both within and across years (Fig. 5.5). However, the relationship between L3 density and faecal egg count is not consistent (Fig. 5.5), due to spatial variation in other factors, such as vegetation structure, body condition, genetic make-up, and so on. Untangling these

interacting factors, and their importance in determining spatial variation in sheep mortality and life-history variation, is clearly a challenge for the future.

SEX BIASES IN PARASITISM AND MORTALITY

One of the most striking patterns to emerge from our studies on St Kilda is the consistent difference between the sexes in their susceptibility to parasitic infection. Within six weeks of picking up their first infection, males have significantly higher faecal egg counts than females, and by their first summer (aged just four months old) their FEC is more than double that of females. The faecal egg counts of the two sexes continue to diverge throughout their lives (Fig. 5.10), except in high-density springs when they temporarily converge (Fig. 5.6). Parasite establishment rate appears to be approximately twice as great for male lambs as for females (Fig. 5.7) and, whereas females develop long-lasting acquired immunity in response to nematode infection, males fail to develop any further protective immunity beyond their second year of life (Fig. 5.10). Thus, males appear to have poorer acquired immunity than females and higher parasite loads.

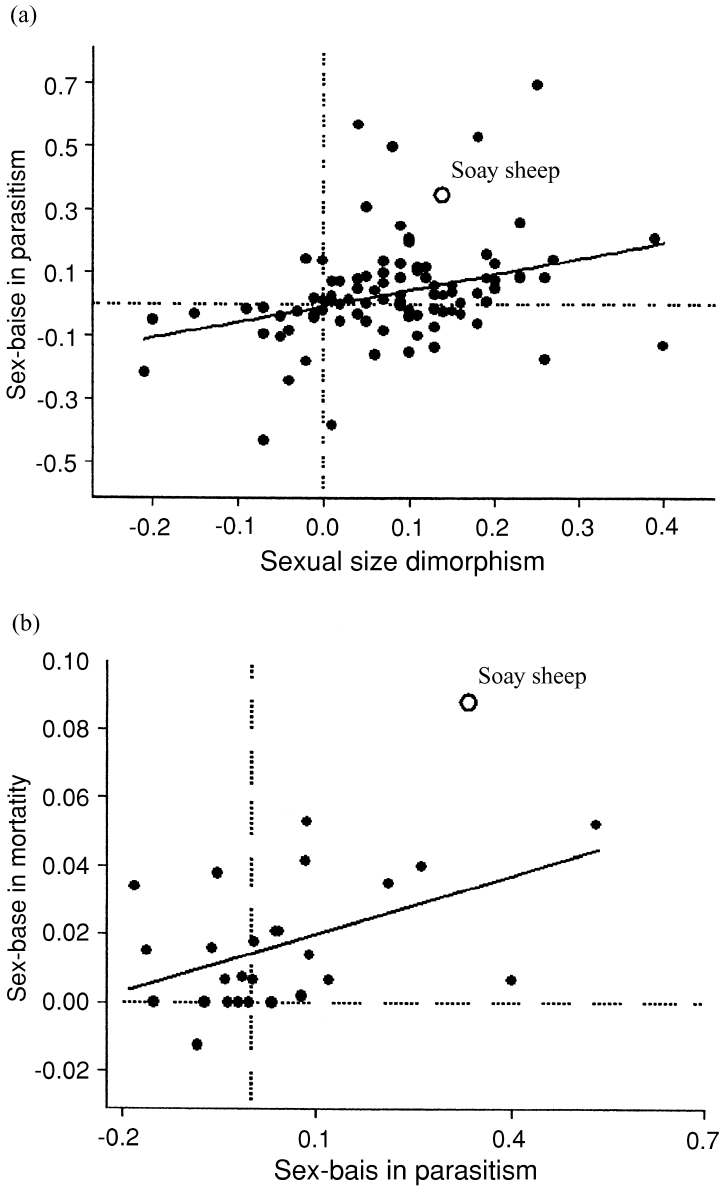
This trend, of males being more heavily parasitised than females, is not unique to the Soay sheep of St Kilda. It is also observed in a number of other ungulate species, including bighorn sheep infected with lungworm (Festa-Bianchet 1991b), white-tailed deer infested with ticks (Kollars *et al.* 1997) and red deer infested with bot flies (Bueno-de la Fuente *et al*. 1998). Indeed, a number of recent meta-analyses have found that across a range of host taxa, including mammals, the two sexes often differ in their susceptibility to parasitism (Poulin 1996; Schalk and Forbes 1997; McCurdy *et al*. 1998; Moore and Wilson 2002). These analyses were prompted by the observation that male and female sex hormones differ in their effects on the immune system; in females, oestrogens stimulate humoral immunity and inhibit cell-mediated responses, whereas male androgens (including testosterone) depress both humoral and cell-mediated immune responses (Grossman 1985; Schuurs and Verheul 1990). As a consequence, parasites may often fare better inside male than female hosts, possibly generating a sex bias in parasitism rates. The two sexes also differ in

their behaviour and this might lead to variation in their exposure to the parasites' infective stages, as might differences in body mass, since large animals may offer larger targets to the infective stages of parasites or their vectors. All of the comparative analyses conducted thus far indicate that, in mammals, males tend to be more heavily parasitised than females. Moreover, the extent of the male bias in parasitism tends to be greater in polygynous than monogamous species, and is positively correlated, across species, with the degree of sexual size dimorphism (Moore and Wilson 2002). Thus, species in which males are significantly larger than females tend to exhibit male-biased parasitism, whereas the opposite trend is observed in species where females are the larger sex (Fig. 5.14a).

In humans (Owens 2002), as well as in other mammal species (Promislow and Harvey 1991; Promislow 1992), males tend to suffer not only from higher levels of parasitism than females, but also greater mortality. A recent comparative analysis indicates that these two phenomena may be related since, across mammal species, the extent of male-biased mortality was positively correlated with the

FIG. 5.14. Relationship between sex-biased parasitism and (a) sexual size dimorphism and (b) sex-biased mortality in natural populations of mammals. Each of the closed symbols represents mean values for a mammal species, using data extracted from the literature (see Moore and Wilson (2002) for details). The data for the Soay sheep (open symbols) are based on the sex-specific mean prevalence data displayed in Fig. 5.10, sex-specific body weights from Illius *et al*. (1995), and sex-specific life-expectancy data (unpublished analysis). For each host species, sex-biased parasitism is calculated as the difference between mean prevalence of infection in males and females. Thus, positive values represent species in which males tend to be more heavily parasitised than females. Sexual size dimorphism is calculated as the logarithmically transformed ratio of mean male to mean female body mass, such that positive values represent those species in which males are typically larger than females. Sex-biased mortality is calculated at the log-transformed ratio of female life expectancy to male life expectancy. Thus, positive values represent species for which males typically outlive females. For Soay sheep, sex-biased mortality estimates range between 0.09 and 0.44, for cohorts born between 1980 and 1990. For illustrative purposes, only the minimum value is shown here. The lines are the least-squares regressions to the data published by Moore and Wilson (2002), which exclude the Soay sheep data.

extent of sex-biased parasitism (Moore and Wilson 2002) (Fig. 5.14b), even after the extent of sexual size dimorphism had been controlled for. Thus, it appears that differential susceptibility to parasitism may contribute to the widespread phenomenon of male-biased mortality in mammals (Promislow and Harvey 1991; Promislow 1992). To test



these ideas further, empirical studies of host–parasite systems are required, and St Kilda provides an ideal model system for doing this. Indeed, to date, Soay sheep on St Kilda provide the best data so far in support of the hypothesis that parasites contribute to sex-biased mortality (Moore and Wilson 2002; Wilson *et al.* 2002). Not only do male Soay sheep have consistently higher parasite loads than females, but they also appear to suffer greater parasite-induced mortality. In all age classes, over-winter mortality is significantly higher in males than females, being up to 100% higher in some years (Clutton-Brock *et al.* 1997). The observation that this sex-biased mortality was eliminated in a group of yearling males that had their parasites chemically removed (Fig. 5.12) suggests that much of the 'additional' mortality suffered by males is a consequence of their higher parasite loads, at least within the yearling age class. Further experiments will be required before we can determine the extent to which sex-biased susceptibility to parasitism is responsible for generating male-biased mortality in older age classes, and whether this mechanism can help explain male-biased mortality in other host taxa.

Parasites impinge not only on the survival of male Soays, but also on their reproductive success. On St Kilda, where male–male competition is the primary form of sexual selection, mating success is determined by relative stature (as measured by body size and mass), horn size (used in fights with other males) and stamina (required to pursue rival males and oestrous females, and a function of body condition) (Preston *et al.* 2003; Chapter 9). Although parasites do not appear to affect the skeletal growth of either sex, they do reduce the rate at which males gain body mass and grow their horns, and these effects are greater in males than females (Fig. 5.12). Parasites also appear to reduce male sexual activity (Fig. 5.13). Thus, for males, at least, parasites are an important constraint not only on male survival, but also on male reproductive success. Similar results have been observed in other ungulate populations. For example, in reindeer (*Rangifer tarandus*), antler asymmetry but not size was correlated with parasite load (Lagesen and Folstad 1998), and in white-tailed deer (*Odocoileus virginianus*), body size and total number of antler points were significantly reduced in individuals heavily infected with liver fluke, particularly

for animals in the youngest age classes (Mulvey and Aho 1993). All of these studies suggest that parasites may impinge on the reproductive success of individual males and that weapons (antlers and horns), in particular, may act as sensitive indicators of a male's ability to resist parasitic infection. As such, it has been suggested that visual attributes of weapons may be used in mate choice to indicate information about parasite burden (Markusson and Folstad 1997). To date, there is little evidence for female mate-choice in Soays (see Chapter 9).

REGULATION OF VERTEBRATE POPULATIONS BY MACROPARASITES

The mathematical models of Anderson and May (e.g. Anderson and May 1978; May and Anderson 1978) clearly demonstrate the potential of parasites to regulate their host population via density-dependent reductions in host fitness. These models show that stable regulation of the host population is particularly likely when a large proportion of the parasite population is aggregated in a small proportion of the hosts (i.e. when k is small; see section 5.3). However, if parasite aggregation is too great, then the host population will escape regulation by the parasite. Since the majority of macroparasites, including *T. circumcincta*, exhibit aggregated distributions (Shaw and Dobson 1995; Shaw *et al.* 1998), it is possible that population regulation by macroparasites is quite common (Tompkins and Begon 1999).

By far the best evidence that parasites may regulate the size of a wild vertebrate population comes from studies conducted in the north of England on the red grouse (*Lagopus lagopus scoticus*) and its parasite *Trichostrongylus tenuis* (Hudson *et al.* 1985, 1992a; Hudson 1986; Hudson and Dobson 1989, 1997; Dobson and Hudson 1992). The red grouse is a medium-sized game bird that inhabits the heather moorlands of Britain, and *T. tenuis* is a nematode parasite that lives in the caecum of its host. Unlike Soay sheep, red grouse do not produce an effective immune response against their main parasite and this appears to have a major impact on the host–parasite dynamics. Populations of red grouse exhibit cycles with periods ranging between four and eight years (Potts *et al.* 1984; Hudson *et al.* 1985; Dobson and Hudson 1992). As with the Soays, high population densities are associated with high parasitism rates and high mortality. Experimental reductions

of parasite burdens suggest that *T. tenuis* not only reduces the body weight and survival of red grouse, but also has direct effects on their reproductive success (clutch size, hatching success and chick survival) (Hudson 1986; Hudson and Dobson 1989). Mathematical models indicate that it is the parasite-induced reductions in host reproduction that enhances the ability of *T. tenuis* to destabilise the red grouse population dynamics and to generate population cycles (Dobson and Hudson 1992). These models predicted that if the parasite population could be reduced to a sufficiently low level, then the population cycles would be stopped. The results of a replicated field experiment over ten years clearly showed that when cyclical grouse populations were administered with anthelminthics to chemically remove their parasites, the amplitude of population cycles was severely reduced, whilst control populations continued to cycle as before (Hudson *et al.* 1998). This strongly suggests that parasites are primarily responsible for the instability observed in these populations of red grouse, though the results remain controversial (Hudson *et al.* 1999; Lambin *et al*. 1999; Tompkins and Begon 1999).

So, do parasites have a similar effect on the population dynamics of Soay sheep and other ungulate populations? For the vast majority of ungulate populations, the role of parasites in host dynamics has not been examined. However, a recent study of the Svalbard reindeer (*Rangifer tarandus plathyrynchus*) living in the high Arctic strongly suggests that the negative impact of nematode parasites on host fecundity may be sufficient to regulate population densities around their observed levels (Albon *et al*. 2002). The evidence that parasites are necessary and sufficient for the regulation of Soay sheep dynamics is less convincing. Experimental manipulations of parasite burdens on St Kilda have clearly demonstrated that trichostrongylids reduce Soay sheep survival during population crashes, particularly in young animals and males of all ages, and there is also some evidence that parasites may reduce the fecundity of females. Thus, it seems likely that the parasites are at least contributing to the depth of the crashes, if not the frequency of their occurrence. However, unlike red grouse, which lack an effective immune response against *Trichostrongylus tenuis*, the Soay sheep acquired immune response is

usually extremely effective against *Teladorsagia circumcincta* (in females, at least) and breaks down only in late winter or early spring during years of high population density. Thus, the effects of parasites on growth, survival and reproduction are much more 'focussed' in Soay sheep than in red grouse.

Population crashes on St Kilda are generated by a complex interaction between the sheep, their food supply, their parasites and climate (Grenfell *et al*. 1992, 1998; Coulson *et al*. 2001; Chapter 3). Thus, mortality episodes coincide with periods when the sheep have become malnourished, due to a reduction in: the amount of food available (a function of sheep density and weather), feeding opportunities (limited by weather and parasite-induced anorexia), and gut absorption (reduced by parasite damage). However, the only way to clearly demonstrate the nature of this interaction would be to conduct a factorial experiment in which nutrition and parasite load were independently manipulated and subsequent mortality monitored. Even then, this would not conclusively demonstrate that the parasites are necessary for the generation of population crashes. To do this, we would need to conduct a large-scale experimental reduction of parasite loads similar to that performed by Hudson *et al.* (1998). Although such a manipulation is logistically feasible, the inability to reliably predict crashes in advance (Grenfell *et al*. 1998; Coulson *et al*. 2001; Chapter 3), the lack of suitable control populations, and the inability to replicate the experiment, would severely undermine the usefulness of such a manipulation. Indeed, a large-scale experiment would be inconsistent with the management objectives that The National Trust for Scotland have for St Kilda and its sheep (see Chapter 1). It seems likely, therefore, that progress in determining the importance of parasites in generating population crashes will be made only by combining long-term analyses of the vegetation dynamics (Chapter 4), with small-scale experiments (similar to that conducted by Gulland *et al*. 1993) and mathematical modelling (Grenfell 1988, 1992; Grenfell *et al*. 1995).