

Chapter 2 – Parasites of free-living wildlife, and transmission at the wildlife-livestock boundary

2.1 Introduction

The role of parasites in shaping the ecology of wild animals has received increasing attention in recent years. Once considered to be either a marginal influence of little regulatory importance, or agents of occasional catastrophe, parasites were given a central place in ecology in a series of important publications in the 1970s (Crofton, 1971; Kennedy, 1975; Anderson and May, 1978; May and Anderson, 1978). The quantitative parasitology of wildlife has been the subject of intense subsequent work, reviewed by Grenfell and Dobson (1995) and Hudson *et al* (2002). Parasites can now be considered within the same conceptual framework as other animal populations, subject to and governed through similar forces – on survival, reproduction, dispersal and interaction - as free-living organisms. Furthermore, the influence of parasites on the ecology of their hosts has been acknowledged and given a theoretical basis. This provides a foundation for a logical consideration of the potential effects of parasitism on host population dynamics, and its implications for both wild and domesticated animals.

2.2 Importance of parasites to free-living wildlife

2.2.1 Effects on the individual host

Disease caused by parasites has been recorded in a range of free-living animal species (Davis and Anderson, 1971; Samuel *et al*, 2001). However, it is particularly difficult to gauge the importance of parasitic infection in wildlife because animals are often examined opportunistically, for example cetaceans or seals that become stranded or are caught accidentally in fishing nets (Stroud, 1978; Cerioni and Mariniello, 1996), or killed by hunters. Even animals killed on roads (e.g. Wolfe *et al*, 2001) may not represent an entirely unbiased sample if animals more prone to parasitism, or whose behaviour is affected, are more or less likely to disperse and put themselves at risk. Animals found dead or killed in a non-random manner may therefore carry burdens that are atypical, and such surveys can be used only as a limited reflection of the occurrence of parasitism or disease in the overall population. Equally, it is often difficult to tell whether parasite-associated pathology found in dead animals contributed to death; the impact of parasitic

disease on the individual host may depend on other factors, such as injury or poor body condition (Tompkins and Begon, 1999).

Clinical disease, moreover, is not the only significant consequence of parasitic infection. Sub-clinical effects at lower burdens can be important if they reduce host fitness, either on their own or in combination with other ecological and physiological factors. Much of the information concerning the sub-clinical effects of many parasite groups comes from domestic ruminants, in which losses due to parasitism are of considerable economic importance. Gastrointestinal nematodes, for example, may decrease feed intake, impair gastrointestinal function, alter protein and energy metabolism, and – in some species – cause direct blood loss (Le Jambre, 1995; Fox, 1997). At sub-clinical levels of infection, animals grow more slowly, and may become permanently stunted, while adults show decreased fertility and wool production (Sykes, 1978; Familton, 1991). The negative effects of gastrointestinal parasitism, as well as susceptibility to infection, can be attenuated to some extent by a high protein diet, and conversely are potentiated in malnourished individuals (Wallace *et al*, 1995; Van Houtert and Sykes, 1996; Coop and Kyriazakis, 2001). Wild animals may routinely experience times of nutritional deprivation, during which they may be at greater risk of sub-clinical disease. Free-living ungulates may therefore be susceptible to ill effects from parasite burdens considered insignificant in well-fed domestic livestock (Halvorsen *et al*, 1999).

Some reports of gastrointestinal nematodes in free-living ruminants are presented in Table 2.1. With notable exceptions in Soay sheep and Svalbard reindeer, assessments of the significance of parasites found for the individual host and thence the population have relied on the identification of visible pathological change, and on a subjective comparison with burdens typically associated with disease in domestic ruminants. Thus, Horak *et al* (1983) discarded parasites as the cause of decline in a population of Blue wildebeest on the basis that little pathological change was observed. However, the severity of disease caused by macroparasites is often related to parasite burden, and heavily infected animals may be rarely encountered, either because they are few or because their death can pass unnoticed in the field. Evidence of disease caused by macroparasites may consequently be difficult to find in the population as a whole, and its apparent absence, along with the possibility of sub-clinical effects, does not rule out the importance of parasitic disease to wildlife populations.

Table 2.1. Reports of gastrointestinal nematodosis in wild ruminants. This is not a complete list, but concentrates on studies which make an attempt, objective or otherwise, to estimate effects on the host.

Host species	Nematode species	Number of animals examined for nematodes, and source	Typical mean nematode burden	Effects on host	Source
Soay sheep (<i>Ovis aries</i>)	mainly <i>Teladorsagia circumcincta</i>	69 found dead during cyclical population crash, and examined. 52 treated and 40 untreated animals monitored for survival, and dead animals examined.	8,000-10,000	Abomasal lesions, and pathology indicative of protein-energy malnutrition. Reduced survival demonstrated in infected animals.	Gulland, 1992.
Reindeer (<i>Rangifer tarandus</i>)	mainly <i>Ostertagia grühneri</i>	Experiment on 18 captive, naturally-infected reindeer.	1,600-5,300	Reduced food intake; slowed growth.	Arneberg <i>et al</i> , 1996; Arneberg and Folstad, 1999.
	<i>Ostertagia grühneri</i> and <i>Marshallagia marshalli</i>	18 shot and 6 found dead on the island of Svalbard.	<i>O.g.</i> up to 17,000; <i>M.m.</i> up to 10,000	Effects on performance thought likely, especially on calf growth.	Bye and Halvorsen, 1983; Halvorsen <i>et al</i> , 1999.
Blue wildebeest (<i>Connochaetes taurinus</i>)	mainly <i>Haemonchus bedfordi</i>	55 animals shot to determine the cause of population decline.	mean 400	No evidence of pathology, thought insignificant.	Horak <i>et al</i> , 1983.
Impala (<i>Aepyceros melampus</i>)	mixed trichostrongylid infections	36 impala shot for parasitological investigation.	7,000-20,000	No evidence of pathology; no apparent effect of parasitism on fat deposits.	Horak, 1978.

The effects of parasites on individual hosts, therefore, depend on the number of parasites present and on other factors predisposing hosts to disease. It follows that the impact of the parasite at the level of the host population will be dependent not only on a parasite's inherent pathogenicity, but also on its abundance and distribution, and on the action and distribution of factors predisposing hosts to the negative effects of parasitism.

2.2.2 Effects on host populations: theoretical predictions

If host fitness is decreased as parasite numbers increase, and parasite abundance increases at high host density, this may form the basis for density-dependent regulation of the host population. The way in which parasites affect host fitness is important in determining this, as is the distribution of parasites within the host population. Theoretical studies have laid down conditions that are likely to allow regulation of host populations by parasites. Thus, if parasite-induced host mortality is to be the key regulatory mechanism, this will be successful if parasite fecundity is high relative to that of the host (Crofton, 1971), and parasite mortality, parasite-induced host mortality, and the degree of aggregation are all low (Tompkins *et al*, 2002a). This apparently paradoxical result is explained by the fact that if parasites are concentrated in very few hosts, and these are then killed, a large proportion of the parasite population will be lost and parasites will not affect enough of the host population to be significant in its dynamics. Regulation through parasite-induced reduction in host fecundity, on the other hand, is favoured by a high degree of aggregation, since parasites can continue to exert an effect without ruling out their own perpetuation. Since effects on host fecundity can exert stronger regulatory pressure than those on host mortality (Anderson and May 1978; May and Anderson, 1978), the rather surprising conclusions of this reasoning include (Tompkins *et al*, 2002a):

- (i) Aggregation in parasite distribution, on balance, favours parasite-driven regulation of the host population. Since aggregation can act to decrease observed prevalence and intensity of infection (see chapter 6), and heavily infected hosts are rarely encountered, a parasite present at low prevalence and intensity cannot, therefore, be necessarily ruled out as a potential regulator of the host population;
- (ii) High virulence is not necessary for a parasite to regulate the population of its host. In fact, since morbidity can be a stronger regulator than mortality, and macroparasites more often cause morbidity than mortality, the less lethal macroparasites are more likely candidates for host regulation.

Time delays between the stimulation of a density-dependent response and its effect are predicted to result in overcompensation and instability, which may take the form of damped oscillations, stable limit cycles, or chaos, depending on the intrinsic growth rate of the population and the strength of density dependence (May, 1975; Hassell *et al*, 1976; Bellows, 1981). This instability does not preclude regulation, since fluctuation is still within bounds that are determined by density dependence. Time delays introduced by developmental processes in both host and parasite life cycles, including parasite-induced reduction of host fecundity, may therefore predispose to instability and fluctuation around the equilibrium (May and Anderson, 1979; Dobson and Hudson, 1992).

2.2.3 Evidence for regulation of host populations by parasites

Despite the clarity and robustness of model predictions, evidence of parasite-mediated regulation in natural populations is hard to come by. Proving that parasites have a negative effect on the host through increased mortality or decreased fecundity is a prerequisite for demonstrating regulation, but is in itself insufficient since such effects could merely be additive to other forces that are in fact driving the system. Regulation can only be shown in an experimental setting, where removal of the regulatory factor results in a change in the population at equilibrium, and its reintroduction restores it to its initial level (Sinclair, 1989). This has been achieved in the laboratory for nematodes in mice (Scott, 1987), but is much more difficult in the field, to the extent that only one convincing example exists. Red grouse in northern Britain are commonly infected with the caecal nematode *Trichostrongylus tenuis*, which has been associated with decreased fecundity in its bird host. Nematode infection was shown to be the cause of cyclical changes in grouse populations when cycles were stopped in bird populations that were treated with preventive anthelmintic drugs, but continued in untreated populations (Hudson *et al*, 1998). The cycles and their prevention were predicted by a mathematical model based on parasite-induced reduction in host fecundity (Dobson and Hudson, 1992).

Studies of other host-parasite systems have identified several instances where parasites might play a regulatory role (Tompkins and Begon, 1999). However, these studies have not demonstrated that regulation actually occurs, presumably because the necessary experimental manipulations are so difficult. There is a need for more examples from a wide variety of systems to test the generality of theoretical predictions. Soay sheep on the

Scottish island of Hirta suffer periodic population crashes, which are associated with an increase in parasite burdens (Gulland, 1992). Correlations have been established between high parasite burdens, particularly of the abomasal nematode *Teladorsagia circumcincta*, and both increased host mortality and decreased fecundity. Parasite-induced host mortality may be high in this system relative to the effects on fecundity, and it would be interesting to compare the patterns imposed on host populations by parasites (if shown to occur) with those in grouse. However, high burdens of abomasal nematodes have been associated with suppressed immunity in domestic ruminants (Armour, 1989), and it is possible that high parasite burdens in dead Soay sheep are simply a terminal manifestation of other underlying forces. Conditions that favour high mortality (climatic conditions, for instance, or shorter vegetation) may also coincidentally favour high parasite burdens.

While field experiments in relatively constrained, simple ecosystems may be the key to demonstrating the existence of parasite-induced host regulation in nature, the general effects of parasites on host populations are likely to be a great deal more complex than direct effects on mortality and/or fecundity. Interactions between parasitism and other potentially limiting and/or regulatory factors such as predation, food limitation and aggression, may combine to determine any overall regulatory effect on the population. Thus, snowshoe hares that have been treated to eliminate their parasites are less likely to be caught by predators (Murray *et al*, 1997), and any regulatory effects of parasites through host mortality are, therefore, likely to be magnified by preferential predation. In an experimental setting, predation on American winter moth pupae was shown to be effective in regulating the population only when parasites were present (Roland, 1988).

Interactions between gastrointestinal nematodes and their ruminant hosts are also likely to complicate - and be complicated by - trophic relationships. High ruminant density will tend to increase pasture contamination, larval uptake, and therefore parasite burdens. In addition, uneven vertical distribution of infective larvae on the herbage can result in increased transmission as vegetation is grazed closer to the ground (Sykes, 1987), and increased susceptibility of malnourished and therefore immunosuppressed hosts on overgrazed pasture could further potentiate this effect, so enhancing the negative effects of parasitism at high host density. On the other hand, ruminants with high abomasal parasite burdens have been shown to have a suppressed appetite (Forbes *et al*, 2000), and to avoid pasture heavily contaminated with infective larvae (Kyriazakis *et al*, 1998),

leading to a self-regulatory decrease in larval intake, though it seems that sheep in poorer condition select high quality forage regardless of contamination. Sharing pasture with resistant hosts, meanwhile, may act to remove larvae and therefore reduce transmission (Barger, 1997a; Barger, 1999). Finally, physiological adaptations in parasitised hosts may act to attenuate the effects of parasitism, so reducing negative effects on them and favouring host and parasite survival (Hoste, 2001). Host population density could, in theory, be governed by a combination of forage availability and parasite abundance, and further feedback from host abundance on plant growth and biomass raises the intriguing possibility that parasites are key regulators of entire grazing systems (Grenfell, 1988; Grenfell, 1992). Exact effects are difficult to predict, however, much less investigate with the necessary rigour, and so far this possibility remains a theoretical one.

Competition within and between host species may also be modified by their respective parasites (see section 2.4), and effects of parasitism on host fitness may take other subtle routes. Nematode parasites such as *Trichinella spiralis* can dramatically alter behaviour in mice and make them more liable to social exclusion through conspecific aggression (Barnard, 1990), while levels of aggression in mice may themselves be altered by infection with *Toxocara* sp. (Holland and Cox, 2001). Aggression has been proposed as a proximate cause of population regulation in small mammals (Chitty, 1960), and parasites could, therefore, potentially affect populations through modification of host behaviour. Parasites can also affect their host's social status, which could alter reproductive success (e.g. in reindeer, Folstad *et al.*, 1996), and motility (Barnard, 1990). Dominant individuals may, by their behaviour, be more or less prone to further parasitism. Pheasants which are less parasitised by ticks are more able to defend territories, and expose themselves less to further infection than do more mobile males of inferior standing (Hoodless *et al.*, 2002). Reindeer calves of dominant females, on the other hand, eat more and consequently acquire higher nematode burdens (Halvorsen, 1986). It is easy to see how the interplay between parasitism, social behaviour, and further parasite acquisition could lead to complex effects on host fitness and population regulation, which are at the same time interesting and difficult to test.

The true impact of parasitism on host populations, and their contribution to regulation, are therefore difficult to disentangle from the many other factors that act concurrently. It is possible that parasites are the major regulators of host populations only under very specific circumstances, for example when other regulatory factors such as food limitation

or density-dependent dispersal are removed. On the other hand, they may only become regulatory when other factors such as food supply are also limiting (Murray *et al.*, 1998). Factors important in regulating populations far from their equilibrium may be very different to those acting close to equilibrium, and, also, more than one stable point might exist (Sinclair, 1989).

Even when the regulatory potential of parasites is not fulfilled, and they do not determine the direction of population change, they can still be important in determining the level of population abundance. The interaction between density-dependent and density-independent factors in general, and the effect of covariance in factors that affect host and parasite populations, is discussed further in section 2.3.

2.2.4 Human impact on wildlife parasites

The increasing influence of man over many ecosystems raises the question of how human activities might modify already complex host-parasite relationships in wildlife. Effects on wild populations may include constraining range size, limiting or forcing movement, causing stress, or modifying the physical or biological environment, for example through changing land use patterns or pollution. An early example of the effect of restricting wildlife range on parasite transmission is described by Heinichen (1973), who attributed severe mortality in the recovering bontebok (*Damaliscus dorcas dorcas*) population in the 1930s to increased lungworm infection following confinement to fenced areas. In addition, the introduction of domestic animals and increasing wildlife-livestock contact creates opportunities for disease and parasite transmission between wild and domestic animals (Cleaveland *et al.*, 2002). Current understanding of parasite transmission at the wildlife-livestock boundary is discussed in section 2.4.

2.3 Regulation of parasite populations

Any factor that acts to decrease the number of parasites generated by each individual in the present generation has the potential to limit or control parasite abundance. Population size at each stage of the life cycle is determined by the balance between birth (or development from the previous stage) and mortality: both total population size and the population of adult parasites will therefore depend on the processes of birth, development and mortality at all stages of the life cycle, and transmission between hosts.

2.3.1 Determinants of parasite population change

A parasite population may establish and persist in a given host population if its basic reproduction ratio, R_0 , is greater than unity (Anderson and May, 1991; Roberts and Heesterbeek, 1995). R_0 is the average number of female parasites produced by each existing female parasite in its lifetime, in the absence of density-dependent constraints. An expression for R_0 derived from a simple model of the dynamics of cyathostome nematodes in horses (Smith and Scott, 1994) is:

$$R_0 = \frac{\beta}{(\mu + \beta)} \frac{\lambda}{a} \quad (2.1)$$

where λ is rate of production of larvae from adult worms, μ is larval parasite mortality, a host mortality, and β the rate of ingestion of larvae. R_0 will then tend to be higher if the parasites are more fecund, or larvae live for longer, and will be decreased by host mortality. High transmission rates will also increase R_0 , especially if $\mu \gg \beta$ rather than $\mu \ll \beta$.

In reality, expressions for R_0 become complicated as the number of parameters, life cycle stages and time delays considered increase, and R_0 may then vary considerably over time. However, in the simplest case, R_0 must still exceed unity for parasite persistence, while if $R_0 < 1$ the population will tend to decline towards extinction. It should be noted that R_0 represents the theoretical maximum reproductive output of the parasite in a given situation. If host immunity, or any density-dependent constraint, acts to affect parameters, the appropriate measure is R_e , the effective reproduction ratio. R_e can never exceed R_0 .

2.3.2 Density dependence in parasite populations

Density-dependent constraints have the potential to regulate parasite populations. Density dependence can potentially occur at any stage of the life cycle, but for parasites it is most likely in the constrained and reactive environment of the host. Candidate mechanisms of density dependence in these sites include competition, immunity, and parasite-induced host mortality.

Competition for resources is often invoked as a regulatory factor in free-living populations (Begon *et al*, 1996a,b), and could exist among parasites, particularly if space at the preferred site of infection is limited (Sukhdeo and Bansemir, 1996). Evidence for

competition comes from the displacement of new parasites from their preferred feeding sites when they are already occupied, and from measures of decreased parasite performance at high infrapopulation density (Holmes, 1961, 1962; Keymer, 1982). However, the effects of competition between parasites are difficult to distinguish from those of host immunity. For example, it is well known that liver flukes in sheep are affected by crowding: adult worms in the liver have lower fecundity when present in higher numbers, and new juvenile flukes are less successful at migrating through the liver parenchyma, suffering higher pre-adult mortality. This could be due to competition for limited resources, interference competition – for example through fibrosis, making the migration of late arrivals more difficult – or acquired specific immunity (Smith, 1994). In populations of *Strongyloides ratti* in the gastrointestinal tracts of rats, parasite fitness traits were measurably reduced at high infrapopulation density only in immunocompetent animals (Paterson and Viney, 2002), suggesting that in this system at least, density dependence largely operates through the host response.

Other parasite species present in mixed infections could favour density dependence, by exacerbating intraspecific competition and/or triggering a generally effective host response. Displacement and impaired performance was demonstrated in mixed infections of *Hymenolepis diminuta* and *Moniliformis dubius* in hamsters, and attributed to competition for limiting carbohydrate resources (Holmes, 1962). The number and size of helminths in the alimentary tract of cormorants also appear to be affected by the presence of other species (Dezfuli *et al*, 2002). Infection with *Haemonchus contortus* protected lambs against subsequent infection with *Teladorsagia circumcincta* (Dobson and Barnes, 1995). Again, interference competition through physical disruption of the abomasal mucosa could have been the cause, as well as cross-immunity. Ellis *et al* (1999) found no evidence of interspecific competition among helminths of free-living opossum. Dobson (1985) argues that overdispersed parasite distributions militate against interspecific competition widespread enough to regulate parasite populations.

Evidence for the development of effective immunity to macroparasites, meanwhile, comes from a wide range of sources, experimental and field. It operates in at least some circumstances in laboratory and domestic animals (Michel *et al*, 1973; Keymer, 1982; Smith, 1988; Balic *et al*, 2000), and in man (Quinnell *et al*, 1990; Anderson and May, 1991), though its importance in free-living wildlife is less clear (Wilson *et al*, 2002). In domestic ruminants, acquired immunity to gastrointestinal nematodes is thought to be

most closely linked to cumulative exposure to infective larvae, and can result in reduced establishment of subsequently ingested larvae, increased chance of arrested development, and decreased worm length and fecundity (Coyne and Smith, 1994). Studies on mixed infections are divided on whether cross-immunity occurs among abomasal trichostrongylids (Adams *et al*, 1989; Dobson and Barnes, 1995). The host response to parasitic infection, meanwhile, might both increase and decrease resistance to more distantly related parasites (Szanto *et al*, 1964; Cox, 2001; Clark, 2001; Yacob *et al*, 2002).

Density-dependent parasite mortality may also occur in the absence of an effective immune response, if parasites can cause host death at high infection intensities. Since death of the hosts also results in death of all its parasites (in most cases), parasite-induced host mortality has the potential to be a very strong regulatory factor on the parasite as well as the host population (Crofton, 1971; Anderson and May, 1978).

The success of host immunity and parasite-induced host mortality in regulating parasite populations depends not only on their potential to exist, but also on the extent to which they actually occur, and on their strength. Immune responses to a wide range of parasites have been demonstrated, but they are not always very effective in reducing parasite numbers, and may therefore be irrelevant to parasite populations. Many parasites possess specific mechanisms that enable them to evade or redirect host immune responses (Behnke, 1987), such that they are unaffected even while the energetic cost and pathological consequences of the immune response debilitates the host. If the immune response actually favours host mortality, it may be able to regulate parasite populations independently of its direct effectiveness against them. The benefit of an effective immune response to the host in controlling parasitism, meanwhile, may be outweighed by its energetic or pathological cost (Wakelin, 1994).

The extent to which density dependence operates in parasites of natural animal populations is open to question. This may be because it is difficult to obtain adequate data from free-living populations (see chapter 4). On the other hand, regulation of parasite populations in the wild may be the exception rather than the rule. Wild animals are frequently more thinly distributed than in laboratory, farming or human situations, and parasite burdens may remain below levels at which density dependence is important. The constraints placed on parasite population growth by host density and limitations on the

development and uptake of free-living stages are discussed below and in section 2.4.

2.3.3 Interaction between density-dependent and density-independent factors

When both are acting, the effect of density-independent factors on population change is additive to that of density-dependent factors, resulting in a lower population size at equilibrium (Figure 2.1). Environmental variation in either density-dependent or density-independent factors will lead to greater variation about this point, i.e. regulation will constrain population size less tightly (see Figure 2.2). The region of typical fluctuation will get even broader if density dependence is weak (Sinclair, 1989).

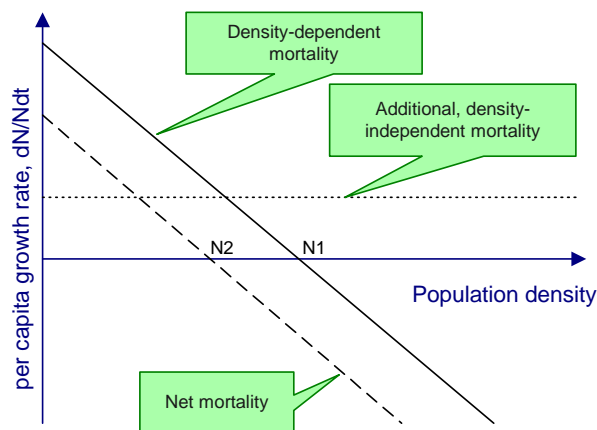


Figure 2.1. Additional density-dependent and density-independent factors. The equilibrium with density-dependent mortality only is labelled N1. Density-independent mortality by definition does not affect the rate of change of population growth rate with population density, but it is additional to density-dependent mortality, and the equilibrium population density decreases to N2. Adapted from Case (2000).

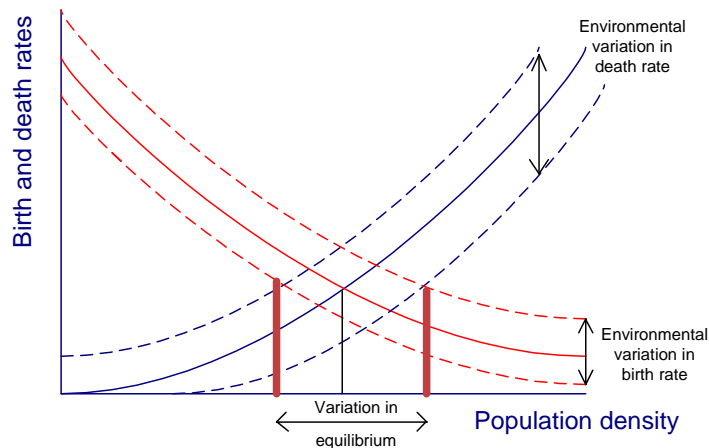


Figure 2.2. Interaction of density dependence and density independence in population birth and death rates. Variation in vital rates in a density-independent manner, through environmental variation, should not change the population size at equilibrium, but will lead to variation in the equilibrium value, and generate a region of typical fluctuation, as indicated. (After Sinclair, 1989.)

The division of limiting factors according to their dependence or otherwise on population density is not entirely straightforward. The same factor may have an effect that has strong, weak or no density dependence at different population abundance, or depending on interactions with other factors (Sinclair, 1989). It would be more accurate to describe factors as acting on a population to generate a combined effect on it that may or may not be density-dependent. Density-independent constraints and environmental variation may maintain the population at a level below that at which density-dependent factors become relevant. A notional scheme of the net effects of constraining factors is presented in Figure 2.3, and Table 2.2 classifies factors acting on gastrointestinal nematodes.

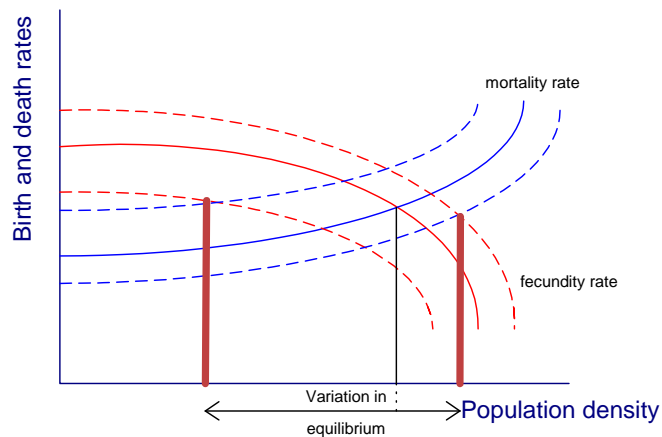


Figure 2.3. A possible pattern of density dependence in parasite populations. Density dependence becomes rapidly stronger as more infrapopulations pass immunogenic thresholds and hosts respond to infection. Regulation is much stronger at high densities than at low densities, and the population size varies further below the equilibrium than above it (c.f. Fig. 2.2).

Table 2.2. Possible sources of density dependence and environmental variation in factors acting on recruitment and mortality rates of adult gastrointestinal trichostrongylid nematodes.

Parameter	Constraint	Density dependence	Environmental variation
Number of larvae ingested	Climate acting on development and mortality of free-living stages	None	High
	Prior host density, when contamination of pasture occurred	May have density-dependent component due to parasite-induced host mortality	May be high, e.g. if climate affects host density
	Parasite abundance and fecundity in previous hosts	Possible	May depend on nutritional status
Larval establishment success	Inherent susceptibility	None	Genetic variation among hosts
	Acquired immunity	Can be strong, above threshold exposure	
Adult parasite mortality rate	'Baseline' natural mortality	None	
	Competition	Yes	
	Acquired immunity		
	Parasite-induced host mortality		

Density dependence that is strong but takes effect only above a certain threshold may provide a 'ceiling', but no 'floor', to population size. The question of what, if anything, governs population persistence at low densities then becomes troublesome. Unregulated populations are predicted to eventually become extinct through stochastic variation (Sinclair, 1989). The rarity with which this seems to occur in nature compared with computer simulation suggests that regulation operates widely in natural populations at low densities. On the other hand, local extinction as a result of climatic stochasticity might be commonplace, stability of free-living populations in the long term being achieved by recolonisation (e.g. Ehrlich *et al*, 1972; Hassell *et al*, 1991; Keeling, 2000). It may be that long time delays in parasite life cycles promote persistence even as they destabilise population regulation, by providing reservoirs of life cycle stages that are each affected by different factors. Thus, extinction of the free-living component of a gastrointestinal nematode population may occur through random climatic effects, but replenishment from the adult population ensures persistence. Conversely, extinction of the adult population through senescence or overcompensating host-induced mortality will be temporary as long as free-living stages remain and there are susceptible hosts and opportunities for transmission. Soil may provide a long-term refuge for infective nematode larvae (Al Saqur *et al*, 1982; though see also Rose and Small, 1985).

Time lags in gastrointestinal life cycles are variable: free-living stages may undergo a prolonged period of dormancy until conditions are suitable for development (Thomas and Stevens, 1960), while ingested larvae can enter hypobiosis, effectively prolonging the pre-patent period (Connan, 1969). The effects of variable time delays on parasite regulation and persistence are interesting, particularly if they vary in a density-dependent manner. In several genera of gastrointestinal nematode, the proportion of ingested larvae entering hypobiosis appears to increase with the number of adult nematodes present (Gibbs, 1986), and emergence occurs as adult worms die. Some workers have suggested that this effectively forms a reservoir for replenishment of the adult stages, maintaining the parasite load at a relatively constant level, though others dispute this (Smith, 1994).

Parasite persistence may therefore owe less to regulation than to local extinction and recolonisation in an environment that is extremely heterogeneous for different life cycle stages. Concepts of heterogeneity and its effects on population regulation are discussed in the next section.

2.4 Environmental heterogeneity and the host-parasite relationship

Heterogeneity in the environment of wild animal populations can drive seasonal variation in their distribution and susceptibility to infection, and therefore in host availability and suitability for parasites. Free-living parasite stages, meanwhile, are exposed to variation in the external environment at a variety of spatial scales, and to changes in climate and weather with time. This spatio-temporal heterogeneity generates a shifting mosaic of opportunities for infection, which parasites must use if they are to persist.

2.4.1 Sources of heterogeneity

Heterogeneity in the environment of the parasite can occur within the host or in the outside world, depending on the stage of the life cycle. Different hosts vary in their inherent susceptibility to infection, and in the way in which they respond to infection. Both are largely a result of genetic variability: the susceptibility of different species to infection is discussed in section 2.5, while there is good evidence that individuals of the same species vary widely in their inherent resistance to gastrointestinal nematodes (Stear *et al*, 1997). Resistance also depends on body condition (Lloyd, 1995; Houdijk *et al*, 2001), and on the strength of any immunity acquired as a result of previous exposure.

Host life history will also generate unevenness in the supply of susceptible individuals. Fish of different ages, for example, may completely change diet and therefore rates of contact with parasite intermediate hosts (Bush *et al*, 2001), while young ruminants may not be capable of developing immunity until they are several weeks old (Balic *et al*, 2000), and are protected from heavy gastrointestinal helminth infection when young because they are not yet grazing heavily. Seasonal birth patterns and host aggregations may present good conditions for infection locally or at particular times of year. The supply of susceptible hosts is critical to the persistence of many microparasites that stimulate complete and lasting immunity (Dobson and Hudson, 1995), and macroparasite transmission could depend on host dynamics in a similar, if less marked, way. The spring turnout of livestock, for example, and concentrated periods of birth such as spring lambing in hill sheep flocks, have long been recognised as critical periods for the transmission of gastrointestinal nematodes (Armour, 1980). Many wild ungulates also give birth over a short period; this may provide an optimal time window for infection, either soon after birth, or later as herbage consumption increases during weaning.

Spatial and temporal changes in climate, meanwhile, profoundly affect the availability of infective stages of gastrointestinal nematodes on pasture (Levine, 1963). In rangelands, climatic variation may also be a major determinant of the distribution of wild and domestic ungulates, both through life history / cultural adaptations such as migration, and by determining food availability in the short term (O'Reagain and Schwartz, 1995). Observed patterns of parasite abundance in livestock in such systems may provide a good model for the role of environmental heterogeneity in determining those of migrating wildlife, all the more so since temporal patterns in body condition and in birth patterns may also approximate those in the wild. Even in intensive livestock production, as practised on fenced pasture in temperate areas, seasonal patterns commonly observed in gastrointestinal nematode abundance may owe as much to seasonality in host population dynamics and susceptibility as to the effect of climate on the free-living stages (Roberts and Grenfell, 1991). In reality, both factors will affect parasite population dynamics, and both effects may be more pronounced in highly variable environments with marked seasonality and extensive host movement. This reasoning is expanded in chapter 3.

Environmental heterogeneity therefore has the potential to shape patterns of parasite abundance in nature through direct effects on parasite populations in the host and in the outside world, and through effects on parasite transmission, for example by determining patterns of host distribution and abundance in time and space. These effects may act together, resulting in complex patterns of parasite distribution.

2.4.2 Implications for parasite distribution

Clumping in the spatial distribution of infective stages in the environment, non-random acquisition of parasites by hosts, and variation in host susceptibility will all favour aggregation in the distribution of parasites among hosts. As already discussed, this aggregation has far-reaching implications for the population dynamics of both host and parasite. Its origins and measurement are of fundamental interest to parasite ecology, and are discussed further in chapter 6.

Variable host presence and susceptibility, and climatic influences on free-living stages, can also generate patterns of parasite abundance with age or season. Examples in free-living wildlife include cycles of abundance in helminths of wood mice (Montgomery and Montgomery, 1988) and snowshoe hares (Keith *et al*, 1985), seasonal variation in

gastrointestinal helminth burdens of white-tailed deer (Waid *et al.*, 1985), and discontinuities in the parasite fauna of fish with age (Bush *et al.*, 2001).

Among gastrointestinal nematodes, spatial heterogeneity at a fine scale is caused by patterns of faecal deposition, since larvae migrate only a short distance from the faeces. If a host encounters one larva near a contaminated site, it is likely to encounter more, and infection therefore proceeds by 'packets' of larvae. Where one area of an animal's range is more frequented than others, repeated contamination will generate patches of high parasite abundance. If these areas are also particularly favourable for parasite development or survival, infection will become further concentrated in them. An obvious example is to be found among the ectoparasites of burrowing animals, whose free-living stages are often concentrated in the burrow (Bush, 2001). Non-random patterns of transmission, and associations between parasite species outside the host, may also give rise to structure in parasite communities (Esch *et al.*, 1990; Poulin, 1998).

2.4.3 Implications for regulation and persistence

The implications of environmental heterogeneity for population regulation and persistence have received much attention with regard to free-living animals. Population dynamic models have been developed for patchy environments where patches differ in carrying capacity or in predation rates (e.g. Murdoch and Oaten, 1975). Unpredictable temporal heterogeneity in factors affecting population growth has been approached using stochastic models or simulations of population dynamics (Renshaw, 1999). The consideration of combined spatial and temporal heterogeneity in forces acting on populations has culminated in the development of metapopulation models, which allow for local extinction and re-colonisation from adjacent habitat patches (Hanski, 1991).

These initiatives in theoretical ecology can be logically extended to a consideration of host-parasite interactions. Hosts provide readily defined, discrete habitat patches for parasites, and parasite infrapopulations therefore have some equivalence to metapopulations of free-living animals. Non-uniform host distribution and movement between and within patches of their own habitat, however, greatly complicate spatial parasite dynamics, and many spatial models of parasite transmission have assumed parasite spread through a uniform and static host population (Keeling, 1999). This may be realistic for plant pathogens, or for microparasites of animals where pathogen spread is

considerably faster than host movement, but it is unlikely to be an adequate description for macroparasites of wildlife. Gastrointestinal nematodes, for example, move with the host, but transmission relies on relatively sedentary infective stages, which become distributed over a spatial plane and are subject to all the spatial and temporal heterogeneity of the environment across that plane, as well as the vagaries of host sampling of the herbage on which they lie.

Existing spatial models of macroparasites have tended to focus on spatial variation in the suitability of the environment for transmission, and are mostly descriptive in nature (e.g. Rogers, 2000; Randolph, 2000; Brooker and Michael, 2000). Our understanding of parasite population persistence, however, might be usefully advanced using some of the more sophisticated approaches of spatial ecology. Metapopulation theory, for example, may provide a way around the problem of predicted extinction of unregulated parasite populations outlined in section 2.2. Regulation may occur at high parasite densities produced in hosts during good years for the development and transmission of free-living stages, but these years could be exceptional, with climatic stochasticity maintaining populations below the region of density dependence most of the time. The ‘floor’ to population size is then extinction and re-colonisation rather than an alleviation of density-dependent constraints. Host population density and movement patterns might then be crucial to long-term parasite persistence in many systems, and they deserve to be considered as factors in the ecology of natural parasite populations.

Co-variation in the spatio-temporal distribution of free-living stages and that of host density may have further implications for parasite population regulation. Transmission will be maximised when peak larval availability coincides with peak density of susceptible hosts. It may therefore be assumed that parasite species that achieve such coincidence will develop a selective advantage and become adapted to that host. Indeed, it seems that among gastrointestinal nematodes of ruminants, *Nematodirus battus* may adopt this strategy, delaying its hatching until the spring when young ruminants are grazing (Boag and Thomas, 1975). However, concentrated intake of large numbers of infective larvae over a short space of time may be more likely to stimulate an immune response in the host, and reduce persistence of adult parasites. A disproportionate immune response may eliminate parasites altogether within weeks of the first infection (Dineen *et al*, 1965). Slower trickle infection may favour longer persistence of adult parasites in the host, without incurring density-dependent penalties, as well as spreading

pasture contamination over a longer period of time and – potentially – a wider area. Such progressive infection requires prolonged host presence, and migrating hosts may be more prone to intense seasonal episodes of infection. If the stimulation of host immunity is inevitable even at low infective doses, concentrated infection and rapid reproduction while there is time might in fact be optimal for the parasite. If parasites can regulate the host population, however, host life history strategy might evolve to decrease the chances of infection. Thus, migration in reindeer has been suggested to be a strategy to avoid infection with *Hypoderma* sp. (Folstad *et al*, 1991).

The duration of any immunity to parasites will also affect the nature of patchy transmission to migratory hosts. Frequent visits to areas contaminated with parasites may serve to boost immunity and enable parasite control in the longer term. The implications of cross-immunity in mixed parasite infections, and the possibility of asynchronous peaks of infection between parasite species as a result of differential impact of climate on their free-living stages, could further complicate the immuno-epidemiology of nematode infections in patchy environments.

2.4.4 Environmental heterogeneity and parasite control

Ideas concerning parasite persistence and regulation are likely to find an immediate and important testing ground in the design of strategies to control or eliminate parasites through manipulation of their environment as it pertains to transmission. An empirical understanding of the effects of climate and host density on parasite abundance has long been used to guide grazing strategies in domestic animals (Gordon, 1948). Thus, climate cannot be controlled, but its effects on free-living stages can be maximised by allowing more time for them to work in the absence of suitable hosts, while heterogeneity in host susceptibility can be used to advantage by grazing mixed ages or species of livestock together or in rotation (Barger, 1997; Barger, 1999).

This approach can be taken a step further by applying emerging ecological ideas. Population viability analysis, for instance, designed largely to help maximise survival probability in threatened populations (Lindenmayer and Lacy, 2002), could be used to minimise survival probability in parasite populations and therefore enhance control and/or aid eradication. Metapopulation theory, meanwhile, has already been used to assess risks of pathogen transfer through movement of endangered wildlife (Hess, 1996).

Increased ecological sophistication in the control of parasite populations in domestic livestock is finding growing enthusiasm as problems of anthelmintic resistance take hold (Barnes *et al*, 1995; Williams, 1997). At the same time, movement of livestock between countries, and the threat of significant climate change, are likely to increase the risk of the introduction and spread of parasites beyond their normal ranges (Harvell *et al*, 2002). Control of parasites in wildlife, and of transmission between wildlife and livestock, might have to use as its principal tool an understanding of the role of heterogeneity in an environment increasingly shaped by man, in the persistence and abundance of parasite populations. Differences in host susceptibility, and patterns of contact between different host species, are all environmental heterogeneities from the point of view of the parasite, and are likely to be prime determinants of parasite transmission at the wildlife-livestock boundary.

2.5 Transmission of parasites at the wildlife-livestock boundary

2.5.1 Host specificity

Adaptations of parasite species that enhance their ability to exploit a given host species may render them incapable of completing their life cycle in other species or types of host. Barriers to infection may be morphological, physiological or immunological. Most infective gastrointestinal larvae, for instance, are enclosed in a chitinous sheath, which must be shed before further development in the host. The stimulus for exsheathing is primarily a high concentration of dissolved carbon dioxide, as found in the rumen (Soulsby, 1982), and natural infection of non-ruminants is therefore unlikely. Natural immunity – e.g. through intestinal mucus and macrophage activity - may likewise prevent infection of unsuitable hosts independently of previous exposure.

In nature, animals become infected only if they come into contact with infective stages in the course of their normal behaviour, and ethology and ecology can therefore provide additional barriers to infection. *Trichinella spiralis*, for example, can infect many species, but natural transmission is largely restricted to predation, scavenging and cannibalism. Successful infection of a host species by inoculation in the laboratory therefore tells us little about host range in the field. Changes in ecological conditions might expose a previously sheltered host, and the relative roles of innate resistance and ecological avoidance in excluding a parasite from a host species or population are therefore

important if we are to predict and control cross-species transmission. Absence of a parasite does not rule out the risk of future infection.

In reality, host specificity is likely to be complicated by the ongoing dynamics of host-parasite co-adaptation. Restricted ecological opportunity for infection might itself be expected in the long term to lead to host specificity. In this respect, mode of transmission may be important. Sexually transmitted parasites, in which interspecific transmission is very unlikely, may well co-evolve with the host and become very specific (Poulin, 1998). Transmission by ingestion, on the other hand, is likely to expose parasites such as gastrointestinal nematodes to a wide range of potential host species. Retention of the morphological and physiological flexibility to infect different host species is then offset against the advantages that might accrue from adapting to a specific host. Speciation of the host might result in speciation of the parasite if geographical or ecological separation results in a break in gene flow between parasite populations. Retained flexibility in the parasite, however, may permit infection of newly encountered hosts.

The existence of more than one possible host for a parasite species, therefore, implies either host switching or continued gene flow between parasite populations in existing and newly evolved host species. Cross-species infection may then be more likely given phylogenetic proximity. The cestode fauna of birds, for example, seems to be similar in closely related species, rather than in those that share ecological traits (Kennedy, 1975). Grazing ruminants are similar in both phylogeny and ecology, and the relative role of each in determining host specificity is unclear.

Long-standing relationships between host and parasite species have in the past been assumed to favour both host specificity and a descent to avirulence (Kennedy, 1975). The 'primary' or 'natural' host can then be identified as the one which shows least immunity to the parasite, while 'foreign' hosts will react and hinder development, in extreme cases preventing establishment altogether. However, the balance of evidence in gastrointestinal nematodes of ruminants is against a lack of intrinsic immunogenicity, even in the presumed primary host (Behnke, 1987). Also, the assumption that host-parasite co-evolution will tend towards avirulence is flawed. Parasites, by definition, have a negative impact on their hosts, and lack of control over the intensity of parasite burdens might not be in the host's best interests. At the same time, a strong immune response is costly in terms of energy and sometimes pathology, and complete elimination of the parasite might

not be optimal in terms of host fitness (Wakelin, 1994; Medley, 2002). From the parasite's point of view, it would seem logical that avirulence and minimal effects on the host would favour parasite survival and thence fitness. However, acute infections with a higher rate of egg production and subsequent elimination might lead to equal or superior total reproductive success, with the energy drain on the host and impact on host fitness – the driving force behind the evolution of immunity- becoming irrelevant to the parasite (Poulin, 1998). Virulence of microparasites may be regulated by a direct trade-off with transmission potential, and a similar situation might exist in macroparasites (Medica and Sukhdeo, 2001). Many questions concerning the evolution of virulence in parasites remain unanswered, but it is clear that we cannot rely on virulence or immunogenicity as indicators of host specificity.

Adaptation to a particular host species might increase the chances of local extinctions associated with host population decline, and the retention of a wide host range might therefore be selected as a trait in itself, especially among parasites of hosts that tend towards instability in population size. Narrow host specificity is also a disadvantage if the uptake of infective stages by unsuitable hosts is common, since they will be removed from circulation and infection of a suitable host becomes less likely. This is the case for ruminants, especially where many species graze together. We might then expect limited host specificity, with retained flexibility to invade ecologically similar hosts, even if parasite fitness is reduced in 'secondary' hosts. In these systems, variation in susceptibility within species may be as important to the fate of ingested larvae as host specificity *per se*.

Many of these ideas are not easily testable, but their consideration is worthwhile, since the dynamic nature of host-parasite co-adaptation is very often ignored when ascribing host specificity to parasite species. Dynamic changes in patterns of host distribution and behaviour, and environmental perturbation, may themselves affect patterns of exposure to parasites, and either create new opportunities for infection or change infection pressure on different hosts. The appearance of parasite species in apparently new hosts as a result of change in the ecological context has been seen in a variety of microparasitic infections, such as influenza in gorillas, and distemper in big cats and in seals (Cleaveland *et al*, 2002). The distinction between specificity and exposure, therefore, takes on central importance when trying to determine the risks of transmission between species amidst changing patterns of host contact. The assessment of such risks must be based on

considerations of parasite abundance, and of host contact, as well as records of parasite host ranges in the past.

2.5.2 Primary natural hosts and cross-species transmission

Characteristic patterns of parasite diversity and abundance in different host species have been widely used in the past to indicate the risk of transmission between wildlife and livestock (Dunn, 1968). Contact between wild and domestic animals, it is assumed, will result in transmission of a shared parasite from the most heavily infected host species to those less heavily infected. This will not necessarily be the case: a species that is ecologically prone to infection may accumulate heavy burdens that rely on ‘topping up’ from the primary host, while low burdens can also be important sources of re-infection. Populations of *Gyrodactylus salaris* (a monogenean parasite of fish) in a Norwegian river were not able to grow in grayling (i.e. $R_e \leq 1$), but could persist in groups of grayling for up to four months, providing a reservoir for re-infection of the primary host, salmon (Sterud *et al*, 2002). The importance of a host species to parasite population persistence also depends on the abundance of the host population itself. A lesser-infected host species may still carry most of the parasite population if it is abundant relative to other hosts.

Another misconception is that similarity in the parasite communities of two sympatric host species or populations implies parasite transmission between them. Even where a number of parasite species are shared, the majority of transmission may be between members of the same rather than different host species, regardless of relative abundance. This is because transmission depends on patterns of contact and susceptibility. Genetic diversity among parasites may also be critical to interspecific transmission.

2.5.3 Population dynamics of parasites in multiple host systems

Given mutual susceptibility and ecological overlap, the risk and predominant direction of parasite transmission will depend on quantitative aspects of parasite population dynamics. Multiple definitive host species can be viewed as contributing to, and drawing from, a common pool of infective stages (Figure 2.4). The relative contribution to the pool will depend on the total abundance of parasites in each population, and on their reproductive output. Relative uptake will depend on host presence and density, and feeding behaviour. The exact nature and timing of contact between host populations will

be of central importance to transmission between them. Contamination of the environment with eggs at times of year when environmental conditions are rapidly lethal will be of little importance, while the order in which species graze a pasture at a favourable time of year will be more critical to the principal direction of spread than will relative parasite burdens.

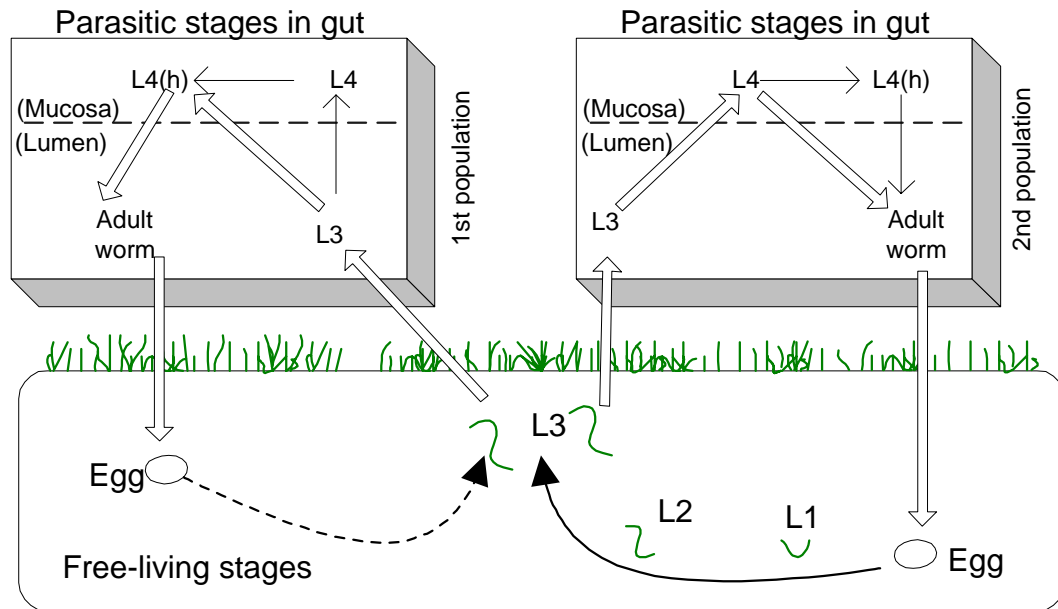


Figure 2.4. Typical life cycle of gastrointestinal trichostrongylid (nematode) parasites of ruminants, illustrating the contribution of multiple host populations to a common pool of free-living infective stages. L1, 2, 3 and 4 represent the first, second, third and fourth larval stages respectively, and L4(h) hypobiotic larvae in the gut mucosa.

Through the shared pool of infective stages, cross-species infection may be important to overall parasite population dynamics and, ultimately, to parasite regulation and control. Larvae that are taken up but fail to develop (in an unsuitable or immune host, for example) are effectively removed from circulation. At the same time, acquired immunity in an important host species may be sufficient to regulate overall parasite abundance, and affect burdens in non-immune hosts. Similarly, an increase in the number of one host species will increase its contribution to the pool of infective stages, and thence the level of parasitism in other host species. If the parasite has effects on the population of these hosts, the parasite will effectively mediate competition between host species. In extreme cases, the existence of shared parasites could lead to extinction or exclusion of competing host species, the surviving species often being that best able to tolerate the highest burdens. Evidence for apparent competition in the field is often confounded by other competitive interactions between species, however, and its general importance has yet to

be established. Recent declines of the grey partridge in the UK have been attributed to apparent competition with pheasants mediated by the caecal nematode *Heterakis gallinarum* (Tompkins *et al*, 2001), while a shared parapox virus has been cited as a factor in the replacement of much of the British population of red squirrels with grey squirrels (Tompkins *et al*, 2002b). The results of infection across a host species boundary might also depend on those parasites already present in the recipient species. The order of infection, as well as relative immunogenicity and pathology, will then contribute to the chances of parasite establishment and to its effect on each host species.

The potential implications of shared parasites for ecological interactions between wildlife species has led to an interest in the modelling of multiple-host, multiple-parasite systems. The algebra of such models, however, rapidly becomes intractable (Begon and Bowers, 1995; Hudson *et al*, 2002). New techniques such as bifurcation theory may provide the tools needed to make sense of complex shared-parasite, multi-host systems. At the moment, theoretical and empirical understanding of such systems is limited, and the few quantitative considerations of them in wild animals (Tompkins *et al*, 1999), have not yet been applied to interactions at the wildlife-livestock boundary.

2.5.4 Control of parasite transmission between wildlife and livestock

Parasites whose main populations are in wildlife hosts may spill over to infect livestock in numbers sufficient to cause production loss. Small but repeated introductions from wildlife can also promote long-term persistence and frustrate attempts to eradicate diseases in livestock. Transmission of foot and mouth disease virus SAT (Southern African Territories) serotypes from buffalo may be responsible for continuing outbreaks in cattle in southern Africa (Thomson, 1995). Persistence of *Taenia hydatigena* in Soay sheep on the island of St. Kilda, meanwhile, is probably reliant on repeated introduction of wind-borne eggs from the Scottish mainland 60km away (Torgerson *et al*, 1995).

The control of parasite transmission between wildlife and livestock can be desirable for a number of reasons. The health of the wildlife population may be important, either for productivity or ecosystem health, or because of conservation concern. Transmission in the other direction, if significant, could have a negative impact on livestock population health and production, with animal welfare as well as economic consequences. Thirdly, a perceived health threat from wildlife may impinge negatively on conservation

programmes designed to preserve or increase wildlife population size and distribution.

Possible methods of parasite control at the wildlife-livestock interface include, most simply, separation of wild and domesticated species, for example by fencing. This is expensive, especially on rangelands where areas are large, and may not fit in with prevailing methods of livestock management. Fences have been extensively used in Africa to prevent transmission of trypanosomosis and foot and mouth disease from wildlife to livestock (Dias *et al*, 1973; Davies, 1981; Suttmoller *et al*, 2000), though their efficacy can be low and economic benefits may not exceed costs (Chilonda *et al*, 1999). Curtailment of movement can also have damaging consequences for the wildlife population (Denney, 1972). Game fences have variously been blamed for genetic separation of the small remaining populations of Prezwalski's gazelles in China (Jiang-Zhigang *et al*, 1995), and for both a decrease in wildlife diversity and abundance, and exacerbation of land degradation, in Botswana (Mordi, 1989; Ringrose *et al*, 1997).

Small wildlife populations threatened with disease may be candidates for medical or immunological intervention. Arctic foxes on the Commander islands were treated with antiparasitic drugs in an attempt to decrease mortality of pups from sarcoptic mange originally introduced by domestic dogs (Goltsmann *et al*, 1996), while an epidemic of morbillivirus in the last known wild colony of black-footed ferrets was controlled by removing animals into a captive breeding programme (Williams *et al*, 1988; Thorne and Williams, 1988); vaccination was also considered (Williams *et al*, 1996). In other cases, such as the risk posed to African wild dogs by epidemics of canine distemper virus in neighbouring domestic dogs (Alexander and Appel, 1994), options may be limited to control of the disease in the domestic hosts. In many cases, the origin of wildlife disease outbreaks is not known, such as the seal morbillivirus epidemic in the North Sea in 1988-89 (Kennedy, 1990; Jauniaux and Coignoul, 2001), and long term monitoring of wildlife is the only 'control' strategy.

In the face of a disease outbreak, culling of source animals or a general reduction in host density have been used to control the spread of infectious diseases within livestock (Thrusfield, 1995), and its spread from wildlife (e.g. Dias *et al*, 1973). Culling is rarely likely to be appropriate for control of macroparasites, since the extent of morbidity does not often justify it, and long-lived free-living stages can act as a reservoir for re-infection.

More practical methods for the control of interspecific transmission of gastrointestinal nematodes of ungulates in particular are likely to be based around strategic movement or treatment of livestock. These are discussed in the next chapter. Logical consideration of the population dynamics of shared parasites, including the effects of host distribution, and the timing and extent of wildlife-livestock contact, will be cornerstones of successful strategies to control transmission of many diseases between wildlife and livestock.

2.6 Synthesis

2.6.1 Priorities for further work

The past three decades have seen parasites emerge from ecological obscurity to become recognised as important members of many ecosystems. Enthusiasm for their potential role as key regulators of wildlife populations, and strong theoretical support for the idea, however, has not yet been matched by extensive and incontrovertible evidence from the field. A major problem is the combination of logistical, ethical, and methodological problems inherent in gathering evidence from free-living populations. The conditions that provide good testing grounds for theoretical predictions – a constrained, accessible, well monitored and easily manipulated population, with well studied dynamics and few confusing extraneous factors acting on it – are the very ones that make them so different to truly natural populations. Of the two most cited field studies of host-parasite interactions at the population level, Soay sheep form a feral island population with no predators or means of dispersal, while grouse are extensively managed through land use and hunting. Both are in the temperate British Isles. Ease of data collection might also skew the weight of evidence for different hypotheses, and with it our perceptions. Tompkins and Begon (1999) suggest that much of the evidence for parasite-mediated density dependence in host populations comes from the effects of ectoparasites on bird fecundity because fecundity is more easily measured than survivorship, and because birds make convenient subjects.

While the opportunities for analytical precision and for manipulation of relatively constrained systems makes them of key importance in furthering our understanding of the processes discussed in this chapter, therefore, there is also room for looking at host-parasite systems in more ‘natural’ circumstances, and in those that bring out a different emphasis. The role of host movement and environmental heterogeneity, and their spatial

scale, in parasite dynamics, and parasite regulation and persistence in shared-host, multi-parasite systems, are both areas where theoretical exploration has begun relatively recently, and as a result has found little empirical resonance to date. There is also a related need to devise more refined ways of analysing imperfect data from wild populations. Improved or repeated data collection from wildlife is not always possible, and analysis should aim to make the most of limited or problematic data sets.

Many wildlife populations, furthermore, are increasingly ‘unnatural’, and affected by human activities, through changes in habitat, hunting, or farming. Hudson *et al* (2002) acknowledge that there is a growing gap between the theory of parasite ecology and the practice of wildlife management. Disease transmission between wildlife and livestock is a problem that threatens to severely undermine conservation efforts and agricultural production in both developed and developing countries. The population dynamics of disease in wildlife and livestock, and, in particular, the effect of changing patterns of contact between them, require greatly improved understanding if transmission at the wildlife-livestock interface is to be approached in a rational way. Preliminary studies of wildlife populations that are suffering natural or anthropogenic perturbation on a large scale are needed, in order to observe the effects on the whole system as they occur. Such approaches can give early indications of the generalities of knowledge gleaned from smaller, neater settings, and provide an opportunity to assess how rapidly advancing ecological understanding can be brought to bear on emerging problems of conservation and disease control.

2.6.2 The saiga-nematode system as a model for study

The saiga antelope (*Saiga tatarica*) is a nomadic herding antelope of the steppes and deserts of Central Asia. In central Kazakhstan, winters are extremely cold and summers hot and dry, and saigas undergo long annual migrations in search of food. Despite the harsh climate and low population density, parasitism is common. The long migrations and harsh climate provide a good opportunity to study the effects of host movement and environmental variation at a variety of spatial and temporal scales on the dynamics of the host-parasite relationship.

Many parasite species of saigas are known to be shared with domestic livestock in Kazakhstan. A comparative approach will help to determine the extent to which observed

patterns of parasite abundance and distribution in saigas are shaped by their life history, as opposed to by general epidemiological patterns in the region. Careful examination of parasite distribution will shed light on the host specificity of the parasites concerned, and on the risks of transmission between saigas and livestock.

There have been dramatic changes in patterns of contact between saigas and livestock in recent years, with the overall density of all hosts plummeting, and movement of livestock decreasing. The ways in which parasite species with different life histories respond to this change will provide insights of general ecological interest. Archives spanning several decades include records of parasite occurrence, climatic measurements, and saiga and livestock population sizes, and analysis of these records could shed light on factors determining parasite abundance in the past. An understanding of these factors will help us to predict likely future patterns of parasitism, and plan control strategies.

The saiga-livestock-nematode system, therefore, combines a range of hosts and parasites whose basic population dynamics are well studied, and an ecological context that is both novel and relevant to wider questions of host-parasite dynamics. The system is not new to study. However, past efforts have paid little reference to developing western thinking, and remain relatively inaccessible in the Russian literature and in unpublished reports. This body of work provides an opportunity to confront recent ideas in parasite ecology with independent data, and compare them with independently derived local understanding.

In the next chapter, existing work on parasites of the Saiga is reviewed, and set in the context of saiga ecology, known epidemiological characteristics of the parasites in Kazakhstan, and their distribution among different wild and domestic hosts. This is used as the basis for a discussion of possible parasite transmission between saigas and livestock, and for the development of an approach to further investigation of the problem.