8 Parasites and pathogens

8.1 Introduction and definitions

This chapter introduces parasitism and disease within wildlife populations. It addresses how an infection affects a population's dynamics and how it spreads through a population. The veterinary aspects of infection, special to each parasite and host, are not dealt with here. Instead we look at examples of how parasites and disease regulate populations, structure communities and affect conservation of endangered species, reduce the potential yield of harvested populations, and are of use in controlling pests.

Parasites feed on living hosts and (unlike predators) do not always kill them. Some parasites have many hosts, others are species-specific. Parasites and pathogens can best be divided into two classes: *microparasites*, which include viruses, fungi, and bacteria, and *macroparasites*, such as arthropods (e.g. fleas, ticks) nematodes, and cestodes (e.g. tapeworms). Microparasites and macroparasites have a roughly equivalent kind of effect upon their hosts and so can be lumped together as *parasites*. The debilitating effect of the parasite upon the host is termed *disease*. At the end of the book we include a glossary of terms often used in parasitology and epidemiology.

8.2 Effects of parasites

All animals support many species of parasites. For example, the American robin (*Turdus migratorius*) has at least 62 macroparasite species, the European starling (*Sturnus vulgaris*) 126 helminth species alone, the African buffalo over 60 species, and we ourselves (*Homo sapiens*) as many as 149 species (Windsor 1998). Many of these species live with their hosts through a substantial portion of the host lifespan, causing some minor debilitation. These parasite species are adapted to their hosts, and the hosts are adapted to the presence of the parasites. Such parasites are said to be *endemic*. The disease caused by this type of parasite is called *enzootic*. (Note the special use of the term "endemic" in this context. In another context, a species is endemic when it is confined naturally to one location, such as an island or a habitat).

Endemic parasites cause *chronic* impacts on a host; that is, low-level, persistent, nonlethal debilities or diseases. Other parasites cause *epizootic* disease (in animals) or *epidemic* disease (in humans). These cause relatively short-term, major, and often fatal debilities. As a result of human impacts and global climate change on ecosystems, we are experiencing the appearance of new diseases, sometimes termed *emerging infectious diseases (EIDs)*. Enzootic and epizootic diseases have different effects on ecosystems, endangered species, and introduced pests. Parasites may lower the standing biomass

Wildlife Ecology, Conservation, and Management, Third Edition.
John M. Fryxell, Anthony R. E. Sinclair and Graeme Caughley.

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of a host population. Hence, they are disadvantageous if the host population is to be conserved or harvested, and advantageous if the host population is to be controlled.

The role of disease in mammals can be generalized to all vertebrates (Yuill 1987). Parasites can be expected in all wildlife species in every ecosystem. Death of the host is unusual and occurs only if: (i) serious illness facilitates transmission, as in rabies; (ii) the parasite does not depend on the infected host for survival and can complete its life cycle after the host dies; and (iii) the pathogen moves through host populations over a wide geographic area and over a long period of time. Disease may have a drastic effect on the survival of wildlife, but more commonly its effects are subtle. It can adversely affect natality or normal movement. Brucellosis in caribou has both effects. A caribou cow infected with brucellosis may abort her fetus, and the same disease can also cause lameness from degenerative arthritis in the leg joints. Infective agents can also affect the host's energy balance by reducing energy intake or increasing energy costs through higher body temperature and metabolic rate.

8.3 The basic parameters of epidemiology

8.3.1 *Simple compartment models of parasite–host interactions*

Simple models for describing the way a disease establishes and spreads through a population start by assuming a constant host population size. This assumption allows us to understand transmission processes over short time intervals. More complex models can also account for changes in parasite and host populations.

For directly transmitted infections of microparasites such as rinderpest, we can divide the host population (N) into three groups: *susceptibles* (S), *infected* (I), and *recovered* (R). The dynamic relationships between these groups are illustrated in a simple compartment model called the SIR model (Fig. 8.1; Anderson and May 1979). Host population size is determined by birth and death rates. Death rates arise from disease and other causes. The effects of disease are described by: (i) the per capita rate of mortality (α); (ii) the per capita rate of recovery (γ); (iii) the transmission rate or coefficient (β); and (iv) the per capita rate of loss of immunity.

The rate of change of the susceptible population is given by the rate of transmission of disease from infected to susceptibles. Thus:

$$\frac{dS}{dt} = -\frac{\beta SI}{N}$$

where N and β are assumed to be constant.

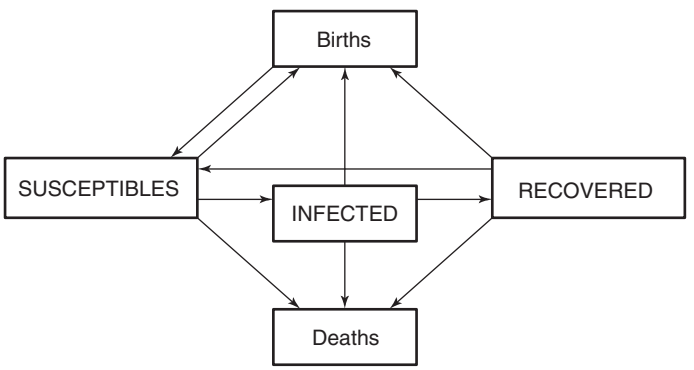

Fig. 8.1 The SIR model, showing the relationships between susceptibles, infected, and recovered components of the population.

The rate of change of the infected population is given by the rate of transmission from infected to susceptibles minus the rate of recovery of infected animals. Thus:

$$\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I$$

where we assume γ is a constant and that transmission is directly related to the proportion of infected individuals in the population (I/N) times the size of the susceptible population (S).

The rate of change of the recovered population is given by the rate of recovered infected individuals. Thus:

$$\frac{dR}{dt} = \gamma I$$

8.3.2 *Thresholds of infection and the transmission coefficient*

When does a disease become epidemic; that is, start to spread through a population? The answer to this question depends on the net reproductive rate R_0 of the pathogen. For microparsites, R_0 is the average number of secondary infections produced by one infected individual, and for macroparasites it is the average number of offspring per parasite that grows to maturity. If the parasite has two sexes, it can also be defined as the average number of daughters reaching maturity per adult female.

If R_0 is less than unity, the initial inoculum of parasites will decay to extinction. R_0 is not a constant for a parasitic species but is determined by the varying characteristics of both the parasite and the host populations, particularly the density of the host. The conditions leading to persistence of the infection are given by Anderson and May (1986) and Anderson (1991) as the ratio of the rate at which new infectives are generated (β) to the rate at which they are lost:

$$R_0 = \frac{\beta}{\gamma}$$

An epidemic occurs if $R_0 > 1$, meaning that more infectives are generated than are lost. An epidemic stops when $R_0 = 1$. We can stop an epidemic by vaccinating a proportion (C) of the susceptible individuals. We can then reduce R_0 by:

$$R_0 = \frac{(1 - C)\beta}{\gamma}$$

The proportion that must be vaccinated in order to prevent an epidemic (i.e. to keep $R_0 = 1$) is:

$$C > 1 - \frac{\gamma}{\beta} = 1 - \frac{1}{R_0}$$

Thus, the proportion to be vaccinated is critically dependent on R_0. If $R_0 = 2$ then 50% of susceptibles must be vaccinated; if $R_0 = 10$ then 90% must be vaccinated (Krebs 2001).

The relationship can also be expressed in terms of a threshold host density N_T below which the infection will die out:

$$N_T = (\alpha + b + \gamma)/\beta > N$$

where b is the mortality rate of uninfected hosts. This makes the point that R_0 is dependent upon host density. Note that if the parasite is highly virulent (large α), or if recovery is rapid (large γ), or if the parasite transmits poorly between hosts (small β), then a dense population (large N_T) is needed to stop the infection dying out. This equation can be elaborated to take into account the effect of an incubation period and post-infection immunity (both of which increase N_T) and "vertical" transmission of the infection, whereby a fraction of the offspring of an infected female is born infected (which lowers N_T).

These equations encapsulate two important concepts of epidemiology:

1 Persistence or extinction of an infection is determined by only a few traits of the host and parasite.
2 The density of the hosts must exceed some critical threshold to allow the infection to persist and spread.

We next examine two examples of disease persistence in wildlife populations.

Swine fever

An example of the study of epidemiology involves classical swine fever (CSF) in wild pigs in Pakistan (Hone *et al*. 1992). This is a viral disease of pigs spread primarily by close proximity of hosts. It is widespread in Europe, Asia, and Central and South America. Understanding of its epidemiology is relevant to efforts to keep it out of North America and Australia.

CSF was introduced to a population from wild boar (*Sus scrofa*) in a 45 km^2 forest plantation in Pakistan. The known starting population (all of which were susceptibles) was 465. One infected animal was released into this population. After 69 days, 77 deaths had been recorded and it was assumed there were no deaths of uninfected animals. The regression of cumulative mortality over time provided a deterministic estimate of the transmission variable β as 0.00099/day. The threshold population of pigs (N_T) below which the disease cannot persist was estimated by:

$$NT = \frac{\alpha + \gamma}{\beta}$$

where α is the mortality rate from infection and γ is the recovery rate. Animals were infective for 15 days over this period. The mortality rate was 0.2/day and the recovery rate was 1/15 or 0.067/day. Thus, N_T was $(0.2 + 0.067)/0.00\,099 = 270$ animals.

The number of secondary infections (R_D) is the ratio of the number of susceptibles (S; in this case the starting population of 465) to the threshold population N_T. Thus:

$$R_D = S/N_T = 465/270 = 1.7$$

A disease establishes when R_D is unity or greater, but this is valid only for the initial population and not a prediction for persistence.

In general, six pieces of information are required from an epizootic to make predictions about the transmission of a disease:

1 the initial abundance of hosts;
2 the number of infectives initially involved;
3 the number of deaths during the epizootic;

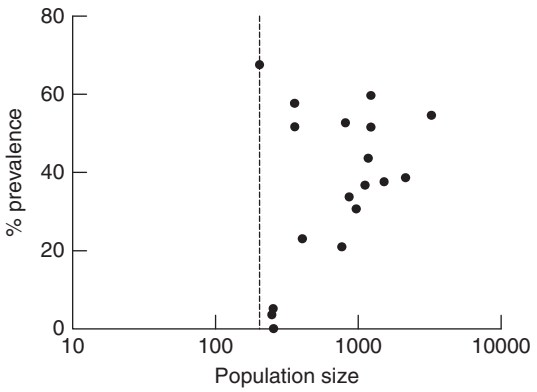

Fig. 8.2 The threshold for establishment of brucellosis in bison of Yellowstone National Park is around 200 animals. (Data from Dobson and Meagher 1996.)

4 the incubation period;
5 the recovery rate;
6 the disease-induced mortality rate.

Brucellosis in Yellowstone National Park

Brucella abortus is a bacterium of the reproductive tract. It causes abortions and is transmitted by animals licking aborted fetuses and grazing contaminated forage. It is common in many ungulates of Africa and has been present in the elk and bison of Yellowstone National Park since the introduction of domestic stock to North America. There are species-specific differences in the effects of the disease on hosts. In elk, over 50% of females abort their first fetus, whereas in bison few if any do so (Thorne *et al*. 1978; Meyer and Meagher 1995).

Bison can acquire brucellosis from elk where the two species mix. Initially healthy bison in Grand Teton National Park acquired the disease from elk on the adjacent National Elk Refuge when the two species fed together in winter at Jackson Hole (Boyce 1989). Modeling of the epidemiology (Dobson and Meagher 1996) shows a threshold population for establishment in bison around 200 animals (Fig. 8.2), and the proportion of the host population infected increases directly with population density. The threshold population, however, is so low that it is very difficult to eradicate the disease – the population would need to be reduced below 200, a cull deemed to be unacceptable in a National Park.

8.4 Determinants of spread

Rate of spread c of an infection is determined, as is persistence, by traits of both the parasite and the host, particularly the rate of mortality α caused by the disease and the net reproductive rate R_0 of the pathogen. Källén *et al*. (1985) give the relationship as:

$$c = 2(D\alpha(R_0-1))^{1/2}$$

where D is a diffusion coefficient more or less measuring the area covered by the wandering of an infected animal over a given period of time. Dobson and May (1986a) calculated the constants of that equation for rinderpest in Africa from the observed radial spread of 1.4 km/day. Pech and McIlroy (1990) used a more elaborate version the other way around, estimating from knowledge of the equation's constants a potential spread of foot-and-mouth disease of 2.8 km/day through a population of feral pigs in Australia.

8.5 Endemic pathogens

8.5.1 *Birth rates*

Parasites usually take some of the energy and protein eaten by the host, and so the host suffers some loss. Such losses, if severe enough, can affect the reproductive ability of the host. When the nematode *Capillaria hepatica* was experimentally introduced to laboratory mice it resulted in reduced number of live young born and higher mortality of young before weaning. Such a reduction of natality and early survival might prevent the plagues of mice that are a feature of Australian wheatlands (Singleton and Spratt 1986; Spratt and Singleton 1986).

The bacterium *Brucella abortus* can reduce both conceptions and births in some ungulate species. In birds, parasites can reduce reproduction through forced desertion of nest sites, as in cliff swallows (*Hirundo pyrrhonota*) and many seabirds, or can reduce clutch size (barn swallow, *H. rustica*), cause delays in mating (great tit, *Parus major*), and lower body condition (house wren, *Troglodytes aedon*) (references in Loye and Carroll 1995). Red grouse (*Lagopus lagopus*) in northern England produced larger clutches of eggs and showed higher hatching success when the nematode *Trichostrongylus tenuis* was reduced with anthelmintic drugs (Hudson 1986). In general, there are still few data on the effect of parasites on host birth rates.

8.5.2 *Mortality rates*

Laboratory mice infected with the nematode *Heligmosomoides polygyrus* exhibited mortality rates in proportion to the intensity of infection (Scott and Lewis 1987). Soay sheep (*Ovis aries*) on the North Atlantic island group of St. Kilda exhibit population crashes every 3 or 4 years. Mortality is highest towards the end of winter, and dead animals have high nematode worm burdens. Live animals that were experimentally treated with anthelmintic drugs had higher survival rates (Gulland 1992). Other studies of rodents and hares show that mortality is often associated with high parasite burdens (e.g. helminths in snowshoe hares, Keith *et al*. 1984; bot flies in *Microtus* voles, Boonstra *et al*. 1980).

8.6 Endemic pathogens: synergistic interactions with food and predators

The great majority of parasites and diseases coexist with their hosts over long periods and do not exhibit wide fluctuations in prevalence over time. Direct mortality from these parasites is usually low. In contrast, they can have important indirect effects by (i) responding to the nutritional state of the host and becoming pathogenic or otherwise increasing vulnerability to predation and (ii) altering the behavior of the host.

8.6.1 *Interactions of parasites with food supply*

There is much evidence that the pathogenicity of parasites is influenced by the nutritional status of the host. In one experimental study, Keymer and Dobson (1987) repeatedly infected mice every 2 weeks for 12 weeks with larvae of the helminth *Heligmosomoides polygyrus*. Mice on a low-protein diet accumulated parasites in direct proportion to the infective dose. In contrast, those on high-protein diets had worm burdens that reached a plateau and even declined over time, and overall the worm burdens were lower for the same dose.

In a field study of snowshoe hares in Manitoba, Murray *et al*. (1997) reduced the natural burdens of sublethal nematodes using antihelmintic drugs. On three of six study areas, hares were provided with extra high-quality food during the winter, when food is normally limiting. The researchers found that survivorship of hares depended on a synergistic interaction of food and parasites. Overwinter survival was 56% in control animals (unfed and normal worm burdens). In unfed but parasite-reduced animals, survival was 60%, while in untreated but fed animals it was 73%. However, in fed and parasite-treated animals survival reached 90%.

Field experimental studies of the effects of parasites are rare, and most information comes from descriptive studies, where animals dying in poor nutritional condition also have high parasite burdens. Studies of the periodic mortality of Soay sheep on St. Kilda indicate that animals were emaciated and malnutrition was the cause of death. However, dead animals also had high nematode counts, indicating an interaction between food and parasites (Gulland 1992).

8.6.2 *Interaction of parasites with predators*

Parasites and pathogens can increase a host's vulnerability to predation by changing its ability to escape the predator. Snowshoe hares with high nematode burdens in spring were more likely to be caught in live traps than those with lower worm burdens (Murray *et al*. 1997). (Wood bison (*Bison bison*) populations may be held at low densities by predators only when there is a high prevalence of diseases such as tuberculosis (TB) and brucellosis (Joly and Messier 2004)).

In the red grouse (*Lagopus lagopus*), there is a complex interaction between the nematode *Trichostrongylus tenuis* and predators such as red fox (*Vulpes vulpes*). These game birds, being ground nesters, are vulnerable to predation while incubating eggs. Normally, grouse emit scent in the feces that can be detected by trained dogs (and presumably by foxes) up to 50 m away. However, during incubation female grouse stop producing caecal feces and dogs cannot locate the birds more than 0.5 m away. The parasite *T. tenuis* burrows into the caecal mucosa and disrupts its function so that the bird cannot control its scent (Dobson and Hudson 1994). Hudson *et al*. (1992a) demonstrated experimentally the effect of these worms on the detectability of incubating red grouse by dogs, treating some birds with anthelmintic drugs to reduce their worm burdens. Trained dogs found many fewer treated birds than untreated birds with naturally high worm burdens (Table 8.1). Thus, parasites increased the susceptibility of grouse to predation.

Parasites may also increase predation on hosts by altering the behavior of the host either as an incidental consequence of debility or as a specific adaptation to enhance transmission; the latter occurs when the predator is the final host in the life cycle of the parasite. In the former case, a disease that causes debility of the host makes it more conspicuous to predators through abnormal behavior, and especially flight behaviors. Other general responses are unusual levels of activity, disorientation, and altered responses to stimuli.

Three lines of evidence support the hypothesis that modified behavior of hosts is a strategy adapted to increase transmission (Lafferty and Morris 1996). First, hosts infected by transmissible stages of parasites often behave differently. Second, experimentally infected prey are more readily eaten by predators in laboratory experiments. Third, infected prey are eaten by predators more frequently than is expected in field

Table 8.1 Red grouse nests found by dogs (scent) and random search (researchers) with respect to treatment of the female with an anthelmintic to reduce burdens. (Source: Hudson *et al*., 1992. Reproduced with permission of John Wiley & Sons Ltd.)

		Number found	
Year	Treatment	Dog scenting	Human search
1983			
Treated	Low worm burden	6	7
Untreated	High worm burden	37	10
1984			
Treated	Low worm burden	9	7
Untreated	High worm burden	29	7

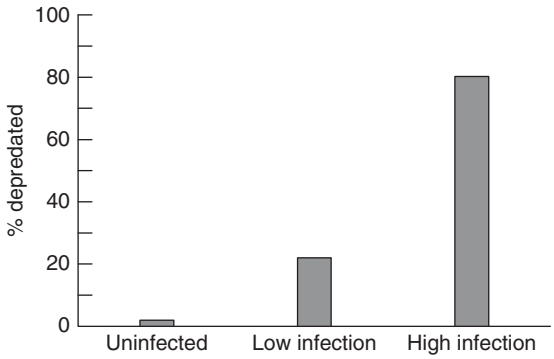

Fig. 8.3 Parasitized killifish are more heavily depredated by birds than are unparasitized fish. (Adapted from Lafferty and Morris 1996.)

studies. We have mentioned that parasitized snowshoe hares were more likely to be eaten by predators in spring (Murray *et al*. 1997). Conspicuous behaviors exhibited by killifish (*Fundulus parvipinnis*) were linked to parasitism by larval trematodes. Field experiments showed that parasitized fish were heavily depredated by birds, the final hosts (Fig. 8.3).

8.7 Epizootic diseases

Unlike enzootic diseases, epizootic ones have intermittent effects on host populations. They have outbreak phases, with rapid spread and high mortality, followed by periods of quiescence, when they lie dormant in host species. The great majority of the pathogens that cause epizootics are microparasites such as viruses and bacteria. Some case studies illustrate their behavior.

8.7.1 *Rinderpest*

Rinderpest is a virus from the Morbillivirus group (genus *Morbillivirus*, family Paramyxoviridae) that produces measles in humans and canine distemper in dogs, cats, and hyena. It is probably the oldest member of the group, from which others evolved. Predators can develop cross-immunity to distemper by feeding on herbivores infected with rinderpest (Rossiter 2001). Its natural host is cattle and it is endemic to Asia. It is highly contagious through droplet infection via licking and sneezing. It causes high fever and inflammation and lesions of the alimentary and respiratory passages.

Rinderpest, as far as evidence goes, was absent from Africa until it was introduced during the 1880s via Egypt to southern Sudan and Ethiopia. By 1889 it was causing epidemics in East Africa, where it killed 95% of the cattle and similar proportions of closely related wildlife, especially African buffalo but also wildebeest, and of the less closely related giraffe, warthog, greater and lesser kudu, and other antelope species (Rossiter 2001). By 1896 the epidemic had reached the tip of South Africa and the West African coast, causing similar mortality. Thereafter, rinderpest reappeared at roughly 20-year intervals, producing slightly less virulent epizootics. Mortality of susceptible animals was at least 50%.

From 1961 to 1976 an Africa-wide cattle vaccination campaign (called JP-15) aimed to eradicate the disease from cattle and thereby from wildlife. The latter, being unnatural hosts, could not maintain the disease by themselves; they obtained it by contact with cattle (Sinclair 1977, 1979a, 1995; Plowright 1982). Although the campaign largely succeeded, a few foci of infection remained in the remote regions of southern Sudan and Mali. By 1979, new outbreaks appeared in Mali, Mauritania, and Senegal,

and in 1981 it appeared in East Africa, dying out in 1984. Lax vaccination programs contributed to the spread, and it now appears that vaccination of cattle is required indefinitely (Walsh 1987; Rossiter 2001).

8.7.2 *Myxomatosis*

The *Myxoma* virus is endemic to rabbit species in South America. It was deliberately introduced to Australia in 1950 as a biological control agent for the European rabbit, which had become a serious exotic pest. The initial spread from the source infection on the upper Murray River was via mosquito vectors, which had increased as a result of recent floods. The first wave of the epidemic took 6 months to cross Australia and mortality was 99%. The virus has remained in the population since 1950 and every 2 years or so outbreaks occur, although mortality has declined to about 87%. Initial abundance of rabbits dropped considerably, but over the decades rabbit numbers have increased as they have become resistant to the virus and virulence has declined. Rabbit fleas (*Spilopsyllus cuniculus*) were introduced to augment the spread of the disease in wetter regions of Australia, and they now act as major vectors of the virus (Fenner and Fantini 1999).

8.7.3 *Rabbit hemorrhagic disease*

Rabbit hemorrhagic disease (RHD) is caused by a virus (*Lagovirus*, family Caliciviridae) that first appeared in domestic rabbits in China during the 1980s. It has since caused heavy mortality of wild rabbits throughout Europe. It is closely related to a disease killing European hares (*Lepus europaeus*).

It was being tested on Wardang Island, South Australia as a possible biological control agent for rabbits in Australia when it escaped and established in wild rabbits on the mainland in 1996 (Mutze *et al*. 1998). Although mechanisms of transmission and spread are not fully understood, blowflies (*Calliphora* spp.), a psychodid fly, the rabbit flea (*S. cuniculus*), and culicine mosquitoes are all carriers. Initial mortality of rabbits was high (about 90%) and rabbit numbers have remained depressed since its introduction (Bowen and Read 1998; Fenner and Fantini 1999).

8.8 **Emerging infectious diseases of wildlife**

The rinderpest in Africa is an early example of a number of diseases that have recently appeared in wildlife and human populations. The phocine distemper virus of grey seals (*Halichoerus grypus*) has spread along the coast of Europe (Kennedy 1990), while in Australia two orbiviruses cause blindness of eastern grey kangaroos (*Macropus giganteus*) (Hooper *et al*. 1999) and the *Chlamydia* bacterium causes blindness and urogenital disease in koala (*Plascolarctos cinereus*). These are wildlife cases, while acquired immunodeficiency syndrome (AIDS), ebola virus, tickborne spirochetal bacteria causing Lyme disease (*Borrelia burgdorferi*), and the virus causing severe acute respiratory syndrome (SARS) are human examples.

These EIDs are associated with a range of underlying causal factors. They can be classified on the basis of three main pathways of infection:

1 EIDs associated with a jump from domestic to wildlife populations living nearby.
2 EIDs connected with direct human intervention through translocation of host or parasite.
3 EIDs with no human or domestic animal associations.

The rinderpest is a clear case of the transfer of a virus from cattle to susceptible wildlife hosts that had not before met the disease. Similarly, canine distemper has spread into wild dog (*Lycaon pictus*) populations of the Serengeti, causing the local extinction of

that species (Ginsberg *et al*. 1995); into lions, causing a 40% mortality; and also into hyena (Roelke-Parker *et al*. 1996). Most likely the rapidly expanding human population surrounding the Serengeti ecosystem, with its associated domestic dogs, which carry the disease, is the source of these new outbreaks. Another example is brucellosis (Meagher and Meyer 1994). This was introduced to North America with the import of cattle and then jumped to elk and bison in Yellowstone in the United States and Wood Buffalo National Park in Canada.

The translocation of wildlife for agriculture, hunting, and conservation has increased exposure to novel diseases. Translocation of fish and amphibians may have caused the ranavirus epizootics now threatening many amphibian populations (Daszak *et al*. 2000). Rabies epizootics in the eastern United States developed from translocations of infected racoons (*Procyon lotor*) from the southern part of the country, where the disease was enzootic (Rupprecht *et al*. 1995).

Zoos and captive feeding programs may inadvertently expose animals to novel diseases due to the close proximity of neighboring hosts. Asian elephants (*Elephas maximus*) in zoos have contracted a lethal herpes virus from neighboring African elephants (*Loxodonta africana*) (Richman *et al*. 1999). There is considerable concern that the agent for bovine spongiform encephalopathy (BSE) could be transferred to zoo-held wildlife through contaminated food, and thereby to free-living wildlife (Daszak *et al*. 2000).

A classic case of epidemic outbreak triggered inadvertently through translocation caused by humans is the catastrophic disease known as white-nose syndrome (Fenton 2012). Massive die-offs of small brown bats were discovered at a number of bat hibernacula in New York State in 2006. Many of the dead individuals had a white powdery infection of their nasal region. Subsequent laboratory analyses confirmed that the fungus *Geomyces destructans* was present in all of the dead specimens. The cause of death is still somewhat unclear, but is currently thought to stem from increased frequency of arousal from hibernation during the fall and winter. As each arousal event burns up a significant fraction of fat reserves, the disease causes death through increased risk of starvation.

Genetic data suggest that the fungus originated in Europe, where it occurs at low, nonpathogenic levels. It is speculated that infected European bats found their way across the Atlantic in a container or some other form of shipment. Subsequent laboratory trials verified that North American bats infected with the European fungal strain developed the full set of symptoms associated with white-nose syndrome (Warnecke *et al*. 2012), confirming that this is a novel disease for North American bats, with characteristically pathogenic properties.

Die-offs rapidly spread to neighboring states (Fig. 8.4), probably aided by bat dispersal during mating swarms in the late summer and fall, with similarly disastrous consequences. The disease has decimated whole bat colonies, with an average mortality rate of 73% per year (Frick *et al*. 2010). While bat survival rates tend to improve in subsequent years following introduction, the population growth rate can still remain negative for a number of years. Population viability analysis (Chapter 16) based on a Leslie matrix model with disease-mediated survival rates suggests that white-nose syndrome may be capable of causing regional if not complete extinction of the small brown bat, once one of the most common bat species in North America (Frick *et al*. 2010). The ecological consequences could be enormous, given the important role that bats play in feeding on aerial insects.

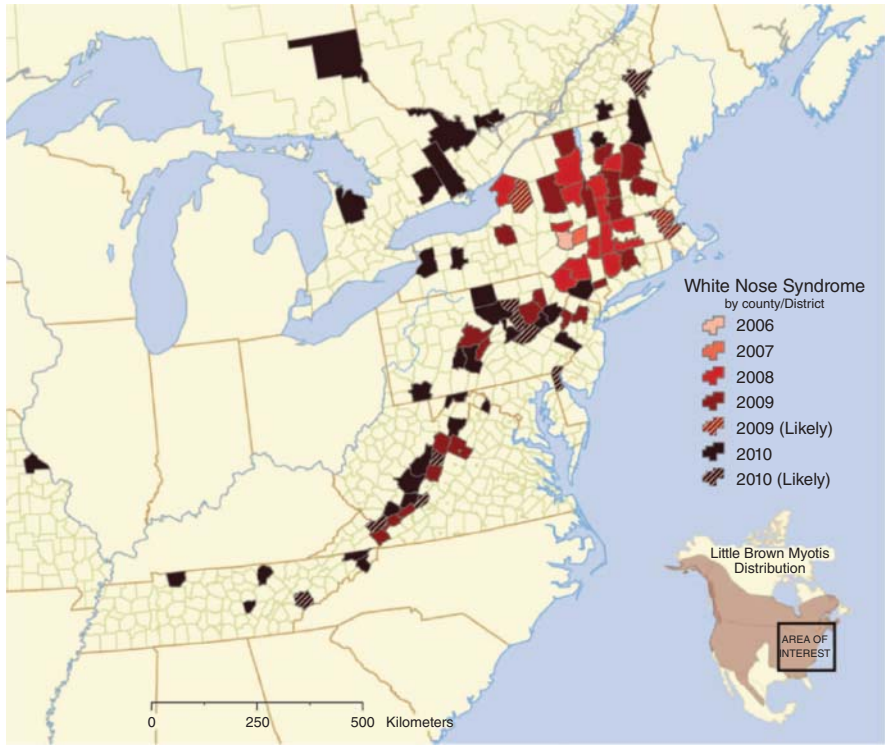

Fig. 8.4 Spatial spread of white-nose syndrome in small brown bat colonies in the northeastern United States during 2006–2010. (Source: Frick *et al*., 2010. Reproduced with permission of the American Association for the Advancement of Science.)

Climate change may be having an effect on the emergence, frequency, and intensity of epizootics. The chytrid fungus (*Batrachochytrium dendrobatidis*) causes mortality and decline in amphibian populations in many parts of the world via the fungal disease cutaneous chytridiomycosis (Berger *et al*. 1998; Pounds *et al*. 2006; Wake and Vredenburg 2008). The synchronous emergence of this novel disease, only discovered in the 1990s, in widely spaced sites (North, Central, and South America and Australia) could stem from global climate change (Pounds *et al*. 1999, 2006). Chytridiomycosis has been linked to massive die-offs of multiple frog species, through disruption of gas exchange at the moist skin surface of amphibians. It usually infects tadpoles, but does not kill them. Unfortunately, juveniles or adults are often reinfected following metamorphosis, with deadly consequences (Wake and Vredenbrug 2008). Movement of infected adults from pond to pond is thought to aid in disease spread (Wake and Vredenbrug 2008). Close correspondence between unusually warm years, chytridiomycosis outbreaks, and disappearances of multiple frog populations in montane study sites in the Central American tropics suggest that climate warming is a trigger for deadly disease outbreaks. Climate change has been similarly implicated in outbreaks of African horse sickness in South Africa (Bayliss *et al*. 1999) and various aquatic diseases (Marcogliese 2001).

In general, the causes of EIDs are largely ecological. These are: (i) movement and migration of hosts and pathogens to new environments; (ii) the change of environment *in situ* through global climate change; and (iii) a change in agricultural and forestry practices that bring species into contact. Changes in genetic characteristics

of the pathogens play little if any part in EIDs, except perhaps in their ability to jump to new hosts (Krause 1992; Schrag and Wiener 1995).

8.9 Parasites and the regulation of host populations

As we have seen, most endemic parasites interact with other factors such as food and predators to reduce host population numbers. There are few examples where parasites on their own regulate a host population; that is, act in a density-dependent way. One clear example comes not from an endemic parasite but from an emerging epizootic disease. The poultry pathogen *Mycoplasma gallisepticum* has entered a previously unknown host, the house finch (*Carpodacus mexicanus*), in North America. The decline in finch population caused by the disease has been proportional to the initial density of finches, with the result that 3 years after the start of the epizootic most finch populations had stabilized at similar densities (Fig. 8.5). Thus, the mortality is density-dependent and the disease has regulated the finch population (Hochachka and Dhondt 2000).

We have already mentioned the emerging epizootic RHD, which was released in Australia in 1996 and has caused major declines in European rabbit numbers (Mutze *et al*. 1998). The disease now appears to be keeping rabbit numbers at very low levels. Bighorn sheep (*Ovis canadensis*) populations regularly experience pneumonia outbreaks caused by the bacterium *Pasteurella*. This has caused declines of bighorn sheep throughout western North America. Pneumonia can regulate bighorn sheep numbers, particularly in Idaho, keeping populations well below those determined by food resources. The source of the disease is domestic sheep, which are less susceptible to mortality from the pathogen than are the bighorns (Monello *et al*. 2001). More anecdotally, the rinderpest virus was probably regulating the African buffalo and wildebeest populations of the Serengeti, Tanzania before its removal through vaccination of cattle in 1963 (Sinclair 1977).

The role of endemic pathogens, particularly macroparasites, in regulating hosts is not clear. The nematode *H. polygyrus* regulated laboratory mouse populations (Scott 1987; Scott and Lewis 1987; Scott and Dobson 1989). We have yet to find examples from the field. However, recent studies suggest that macroparasites may at least be causing population cycles. For example, red grouse populations in Britain exhibit 7-year cycles, and it appears that these might be produced by the nematode *T. tenius*. Winter mortality

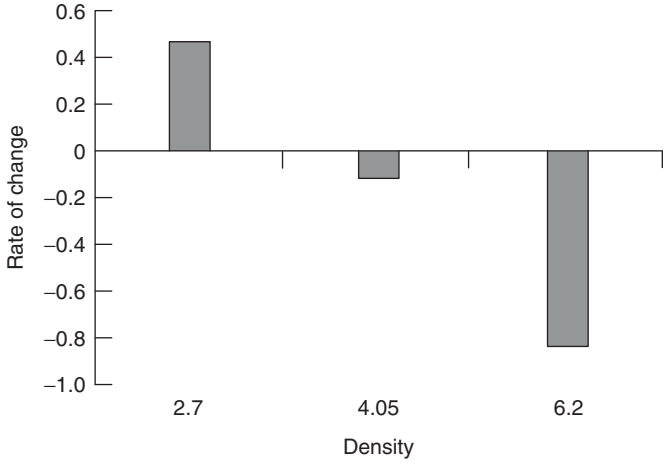

Fig. 8.5 The rate of change of a house finch population due to mortality by the pathogen Mycoplasma gallisepticum is density-dependent. (Data from Hochachka and Dhondt 2000.)

is the major factor determining changes in grouse numbers, although breeding losses are also important. Both winter loss and breeding loss are correlated with the intensity of parasite infection. Cycles may be resulting from time delays in the recruitment of parasites, so that they are partly out of phase with host numbers (Hudson *et al*. 1992b; Dobson and Hudson 1992). This idea was tested experimentally by reducing parasite burdens with anthelmintic drugs. Treatment of the grouse population prevented the normal decline in numbers, demonstrating that parasites were the cause of the decline phase of the cycle (Hudson *et al*. 1998).

8.10 **Parasites and host communities**

As more research on parasites is carried out, we are becoming aware of the role they play in structuring the diversity and abundance of host communities. This is a new area and much remains to be done (Minchella and Scott 1991; Poulin 1999). Most parasites have shorter life cycles and much faster rates of increase than their hosts. These features are the opposite to those of predators and, therefore, parasites can have different impacts on the structure of host communities.

8.10.1 *Altering species interaction*

Parasites can have three types of impact on host communities (Poulin 1999):

1 *Competition* They can affect competitive interactions between two species by having a greater effect on one of the pair. A superior competitor may become an inferior competitor in the presence of the parasite. The northward spread of white-tailed deer in the hardwood forests of North America was accompanied by its meningeal nematode parasite *Parelaphostrongylus tenuis*. This worm is lethal to both moose and caribou, the original inhabitants of the forest, and populations of these species have declined (Anderson 1972; Nudds 1990; Schmitz and Nudds 1994). Thus, the parasite has altered the relative abundance of the three host species by affecting one less than the others. Schall (1992) shows that competition in *Anolis* lizards is altered by the presence of the malaria parasite (*Plasmodium azurophilum*). On the Caribbean island of St. Maarten, the normally dominant *A. gingvivinus* excludes the subordinate *A. wattsi*, which is found only in the central hills. However, the parasite is common in *A. gingvivinus* and rarely so in *A. wattsi*. In the presence of the malaria, the two coexist.

2 *Reducing predation* They can reduce the efficiency of predators or herbivores in obtaining prey so that the prey increase at the expense of their competitors. In other words, parasites can alter the effect of "apparent competition" (see Chapter 9). Little has been documented at the carnivore trophic level. In herbivores, reduced food intake in reindeer is induced by gastrointestinal nematodes (Arneberg *et al*. 1996), allowing heavily grazed, palatable plants to increase in abundance. The presence of rinderpest in the Serengeti ecosystem (Section 8.7.1) reduced the dominant herbivore, wildebeest, by some 80%. One consequence of this reduction of wildebeest was to increase the biomass of grasses on the Serengeti plains and decrease both the diversity and the abundance of small dicot species, which are overshadowed and outcompeted by the grasses.

3 *Increasing prey susceptibility* They can increase the availability of prey for a predator and so alter the competitive relationships between predators. We have already mentioned that parasites alter prey behavior to the benefit of their predators (Section 8.6.2). There are no data on how altered prey behavior affects the community of predators.

8.10.2 *Complex ecosystem effects*

Red grouse in northern England have declined in numbers due to an increase in prevalence of the tickborne virus louping ill, which affects the central nervous system.

The increase of the disease was produced by a change in the relative abundance of two plant species of the heath communities inhabited by grouse: heather (*Calluna vulgaris*), which is the major food for grouse, and bracken (*Pteridium aquilinum*), which produces a humid mat layer, the habitat for ticks. Bracken is increasing at the expense of heather because it can invade when heather is burned. Sheep ticks (*Ixodes ricinus*) are maintained by domestic sheep and mountain hares (*Lepus timidus*). The spread of bracken has increased the exposure of grouse to ticks and hence to louping ill virus (Dobson and Hudson 1986; Hudson *et al*. 1995).

Myxomatosis was introduced to rabbits in England in 1953. It caused severe mortality and resulted in several indirect effects on the ecosystem (Ross 1982). The normally closely-grazed grass lawns on the chalk downs changed to tussock grassland, with *Festuca rubra* and heather invading. Indeed, rabbits, which were introduced to Britain from Europe a thousand years ago, had maintained the species composition of these grasslands for so long that no one knew of any alternative state. There was an initial increase in diversity of flowering herbs, followed by dominance of tussock grasses, and eventually some areas turned to woodlands. Plant succession affected animal diversity: European hares (*L. europaeus*), voles (*Microtus agrestis*), and ants increased, while the sand lizard (*Lacerta agilis*) decreased. Predators that depended on rabbits, such as ermine (stoats, *Mustela erminea*) and buzzards (*Buteo buteo*), also declined. Similar changes were recorded in South Australia after RHD reduced rabbits there in 1996.

Dutch elm disease, caused by the fungus *Ceratocystis ulmi*, decimated elm trees (*Ulmus* spp.) in Britain in the early 1970s. These were among the most abundant trees in agricultural areas and their removal changed the physical structure of the habitats for birds. Death of the trees had less effect than their removal, because dead trees provided nesting and feeding sites. Bird diversity was reduced by eight species (from 36 to 28) as a consequence. Later, as the dead trees disappeared, increased light levels changed the herbaceous plant community (Osborne 1985).

These examples illustrate how the presence or absence of a disease can have complex indirect effects that filter down even to the plant community.

8.11 Parasites and conservation

Parasites and pathogens can be important in all three components of wildlife management. They can cause conservation problems by reducing the densities of species targeted for conservation, they can reduce the potential yield of harvested populations, but, on the positive side, they can be used to control pest species. This section provides examples in each of these areas to give a feel for the range of effects.

The long period of natural selection over which a parasite and its obligate host sort out an accommodation with each other ensures that a persistent infection has little influence on the density of the host. If, however, the specific characters of the host and parasite are such that usually $R_0 < 1$ then the infection is likely to be sporadic and may have a large but temporary depressing effect on the density of the host. Bubonic plague and (until recently) smallpox acted in this way against humans.

As we have already mentioned, parasites can reduce both birth and survival rates and, hence, affect population size. Therefore, they are relevant to the conservation of small populations and can be a cause of population decline (see Chapters 17 and 20). There are several ways in which threatened species may be exposed to parasites.

8.11.1 *Introduction of domestic or exotic species*

Microparasitic diseases are now implicated in the decline and extinction of several wildlife species, particularly carnivores (Ginsberg *et al*. 1995; Kat *et al*. 1995; Tompkins and Wilson 1998; Murray *et al*. 1999). Thus, African wild dog (*Lycaon pictus*), Ethiopian wolf (*Canis simensis*), and Blanford's fox (V*ulpes cana*) in Israel have all been decimated by rabies or canine distemper contracted from domestic dogs. The arctic fox (*Alopex lagopus semenovi*) on the Aleutian Islands has likewise contracted mange from dogs.

Parasites, especially microparasites, have their greatest effect when they jump from one species of host to another. This process is also a major source of evolutionary opportunities for parasites. For example, the knee worm (*Pelecitus roemeri*) has been "captured" by wallabies and kangaroos from the parrot family. The effect of the worm is unknown in parrots, but in macropods it induces a fibrous capsule up to the size of a cricket ball on the knee. Trans-specifics are the parasites and pathogens to watch out for. They can cause significant conservation problems, but they can also sometimes be used to control pest species. Other trans-species parasites must be guarded against because they cause considerable additional mortality. We have already mentioned the myxoviral rinderpest epidemic that swept the length of Africa in the 1890s and killed large numbers of wild ungulates, particularly African buffalo. Asian cattle were its original host, but it jumped across to wild ungulates when it reached Africa. The decline of moose populations in Nova Scotia and New Brunswick is associated with infestation by the nematode brainworm *Parelaphostrongylus tenuis*, which jumped from its original host, white-tailed deer. The infestation in moose is fatal, but there is little evidence that the parasite can maintain itself in the moose except by reinfection from white-tailed deer (Anderson 1972). However, the relationship between meningeal worm, white-tailed deer, and moose has not been studied experimentally and not all the circumstantial evidence is consistent (Samuel *et al*. 1992).

The translocation of domestic or exotic wildlife may lead to parasites and pathogens jumping to a new suite of species. In Australia, native animals such as kangaroos and wombats became infected with common liver flukes (*Fasciola hepatica*) acquired from sheep and cattle. The liver flukes cause severe lesions in the liver of the wombat (Spratt and Presidente 1981).

We have already seen how the presence of sheep brought the sheep tick to English moorlands and the louping ill virus to red grouse, causing their populations to decline. Similarly, high mortality of monkeys (*Presbytis entellies*, *Macaca radiata*) occurred after cattle were introduced to India, increasing the numbers of the tick *Haemophysalis spingera*. This tick carried the flavivirus causing Kysanur Forest Disease (Hudson and Dobson 1991).

Perhaps the most well known example of parasites introduced by wild exotic species is that of avian malaria in the Hawaiian Islands (Dobson and Hudson 1986; van Riper and van Riper 1986; McCallum and Dobson 1995; Cann and Douglas 1999). Early extinction of lowland native bird species (12th–19th centuries) resulted from agricultural clearing of forests, and later the introduction of rat and Indian mongoose (*Herpestes auropunctatus*), which depredated their highly vulnerable nests. The mosquito *Culex quinquefasciatus* was introduced in 1826. It spread across all of the islands but it did not carry avian malaria – no cases were detected from blood samples in the early 1900s.

Many species of exotic bird were introduced to the islands between 1900 and 1930 as a response to the damage caused by the clearing of forests for agriculture. As in New

Zealand and Australia previously, there was an organization (the Hui Manu) committed to introducing exotics. It is now clear that these exotics were responsible for bringing in the avian malaria (*Plasmodium relictum capistranoae*). Native birds were highly susceptible to the parasite and many species became extinct because of it. Now native species are restricted to habitats above 600 m, where both the mosquito and exotic birds are at low density. Reintroduction of native species in lowlands is not feasible in the presence of the parasite.

The species-jumping process may also operate the other way, with wildlife acting as reservoirs of parasites and pathogens transmitted to domestic stock. The controversies over brucellosis in bison (and its transmission to domestic stock in both the United States and Canada) and the transfer of TB (*Mycobacterium bovis*) from European badgers to cattle are obvious examples (Peterson *et al*. 1991; Clifton-Hadley *et al*. 2001). The appearance of SARS in humans in 2002 is thought to have arisen because people in southern China keep civets (*Viverra zibetha*) in captivity and eat them.

8.11.2 *The alteration of habitat*

We explore in Chapter 21 the general consequences of degraded habitats and fragmentation for conservation. One particular effect of habitat fragmentation is increased exposure to parasites. In birds, and perhaps in other animal groups, Loye and Carroll (1995) suggest three mechanisms: (i) increased edge habitat due to fragmentation increases the contact rate between species in adjacent habitats and exposes those in fragments to new vectors and new parasites to which they are more susceptible; (ii) loss of habitat could force birds to reuse old nests, exposing them to higher numbers of fleas, ticks, and other nest-living parasites; (iii) as a special case in birds, fragments expose them not only to predators commuting from the surrounding agriculture but also to brood-parasite birds such as the brown-headed cowbird (*Molothrus ater*) of North America.

Some of these mechanisms are illustrated by Loye and Carroll (1995) for the Puerto Rican parrot (*Amazona vittata*). This species is restricted to a single fragment of high-elevation forest. Habitat degradation, harvesting for the pet trade, and novel parasites have been factors in its decline. In particular, fatal parasitism of nestlings by muscid botflies became a problem after the pearly-eyed thrasher (*Margarops fuscatus*) invaded the forest fragment in about 1950. The thrasher nestlings are host to abundant blood-feeding botfly maggots. The introduced native thrasher therefore brought in its endemic parasite, which then spread to a new but rare host, the parrot.

8.11.3 *Captive breeding and reintroductions*

Parasites and pathogens can be a factor driving the decline of an endangered species and can become an issue in the recovery of endangered species generally. Parasites and pathogens can hinder or thwart attempts to establish captive-breeding populations. Thorne and Williams (1988) review the well-known example of the first attempts to establish a captive-breeding colony of black-footed ferrets (*Mustela nigripes*) in the United States. A previous attempt in the early 1970s failed because canine distemper virus (CDV) killed the only two litters. The source colony also disappeared. The extreme susceptibility of the black-footed ferrets to CDV became apparent when four of six died after being vaccinated for CDV in the 1970s. The vaccine had previously been shown to be safe in domestic ferrets. In 1981 the species was rediscovered in Wyoming, United States and the colony's vulnerability to disease was quickly realized. Precautions were taken to minimize human introductions of disease, especially CDV and influenza. The population declined from an estimated peak of 128 in 1984 to only

16 in 1985. The decline spurred an attempt to start a captive-breeding colony, but the first six ferrets captured rapidly succumbed to DCV. Despite all precautions, CDV infected the colony and most of its members died from the virus. The few survivors eventually formed a breeding population. Nonetheless, as Thorne and Williams (1988) note, "The captive breeding program went from a carefully planned approach with ideally selected, unrelated founder animals to a crisis situation with related animals, a poor sex ratio, and few mature, experienced breeder males."

Captive-bred animals released into the wild may spread disease or pick up parasites and pathogens from endemic wildlife. A potential example of the former is Jones's (1982) report of the release of Arabian oryx captive-raised in the United States for a national park in Oman, which was delayed when the animals tested positive for antibodies to bluetongue disease. The failure of the reintroduction of woodland caribou to an island within their historic range in Ontario, Canada is an example of the problems that can be encountered when reintroduced animals become infected with a disease from the endemic wildlife. The area had been colonized by white-tailed deer and the caribou became infected with meningeal worm via a gastropod secondary host (Anderson 1972).

Another example comes from the captive breeding of whooping cranes (*Grus americana*). An eastern equine encephalitis (EEE) virus fatally infected 7 of the 39 captive-bred population at the Patuxent Wildlife Research Centre in Maryland, United States. At that time in 1985, the captive population accounted for about 25% of the world's population. EEE virus causes sporadic outbreaks of disease in mammals and birds in the eastern United States and is spread by mosquitoes. No deaths are usually seen in endemic hosts, but introduced game birds such as ringneck pheasants (*Phasianus colchicus*) are vulnerable. Among the approximately 200 sandhill cranes in pens neighboring the whooping cranes, some were serum positive for EEE virus, but no clinical signs were found. The discovery of the vulnerability of the whooping cranes to a common pathogen was seen as an unrecognized risk and an obstacle to the species' recovery (Carpenter *et al*. 1989).

8.12 Parasites and control of pests

Spratt (1990), in reviewing the possible use of helminths in controlling vertebrate pest species, pointed out the marked contrast between the numerous successes in biological control of insects and the almost universal failure of such methods in controlling vertebrates. The one unequivocal success has been the use of the myxoma virus to control European rabbits in Australia (Fig. 8.6):

> Myxomatosis is a benign disease in *Syvilagus* (cottontail) rabbits in South America which is transmitted mechanically by mosquitoes. In the European rabbit (*Oryctolagus*), which is a pest in Australia and England, the virus from *Sylvilagus* produced a generalized disease that is almost always lethal. Myxomatosis was deliberately introduced into Australia in 1950 and into Europe in 1952. It was first spectacularly successful in controlling the rabbit pest, but biological adjustments occurred in the virulence of the virus and the genetic resistances of the rabbits. After 30 years of interaction, natural selection has resulted in a balance at a fairly high level of viral virulence. Fenner (1983)

The initial annual mortality rates were very high in Australia – over 95% – but they dropped progressively over the next few years. There is a widespread perception that

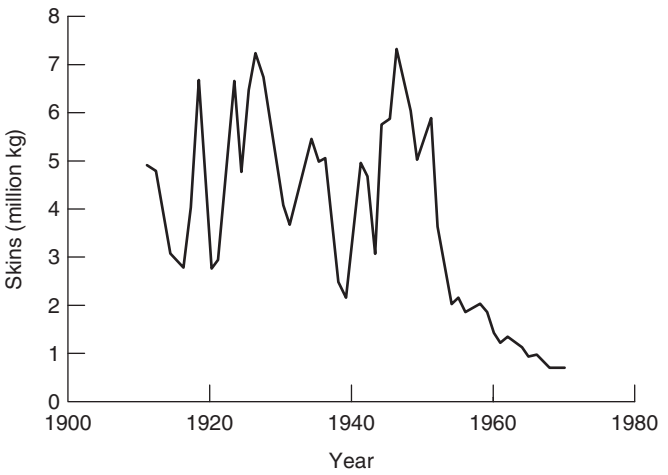

Fig. 8.6 The number of rabbits trapped in Australia (million kilograms of skins) shows a rapid decline after 1950, when the myxoma virus was introduced to control them. (Data from Fenner 1983.)

the rabbits and the disease accommodated to each other and, therefore, that myxomatosis provided only a temporary respite. This is not so. The rabbit density at equilibrium with the disease is considerably lower than the mean density in the absence of it.

Parer *et al*. (1985) demonstrated the controlling effect of this virus. They used a relatively benign strain of myxoma to immunize rabbit populations against the more virulent field strains that swept through the study area in most years. Rabbit densities increased by a factor of 10 under this treatment. Even after the rabbits and the virus had reached an accommodation with each other, the disease was apparently holding the mean density of rabbits to about 10% of that prevalent before its introduction.

8.13 Summary

Most parasites and pathogens have little effect on their hosts. When a parasite jumps from one host species to another, it is the "naivety" of the new host to the parasite or pathogen that is responsible for the reaction of the new host individual or population to the parasite. In both meningeal worm in cervids (other than white-tail deer) and liver fluke infection in wombats, it is the dramatic host immunologic response to the new parasite that is responsible for the debilitation in the animals. Such a response has been dampened over time as the meningeal worm has evolved in white-tail deer and liver fluke in sheep, so that we no longer see the same level of debilitation in the "normal" host species.

The key points from the epidemiology of parasites and pathogens are that the fate of an infection is determined by only a few traits of the host and parasite and that there is a critical density of the host that allows the infection to persist and spread. Efforts to reduce the effects of parasites and pathogens can be at their most important in the management of small populations of endangered species, be they in the wild or in captivity. Diseases of harvested wildlife are more rarely controlled, unless they present a potential hazard to people. Few attempts to use parasites and pathogens to control pest wildlife have been successful.

9 Consumer–resource dynamics

9.1 Introduction

In this chapter we explore those things an animal needs to eat so as to survive and reproduce: resources. This leads to a description of the structure and dynamics of consumer–resource systems, in which the consumers and their resources can interact in complex ways. We show how to analyze such systems by breaking them down into their dynamic components. This approach is used to compare several different systems: kangaroos and plants in Australia; trees, moose, and wolves in North America; small mammals in northern Europe; and snowshoe hares and lynx in Canada.

9.2 Quality and quantity of a resource

A *resource* is defined as something that an animal needs whose consumption by one individual makes it unavailable to another. The most obvious example is food, and to that may be added shelter, water, and nesting sites. By definition, a resource is beneficial. As the availability of resources rises, an individual's fecundity and probability of survival are enhanced.

Food resources are often characterized by two attributes: the amount of food available to an animal and the suitability of that food to the animal's requirements. For example, quality may be described as the percentage of digestible protein in the food, whereas quantity may be measured as dry mass of food per hectare. This often leads to a discussion on whether the quality or the quantity of the food is the most important to the animal. In most cases the distinction is meaningless. It indicates that the resource is being measured in the wrong units. If the resource is in fact digestible protein then that is what should be measured. The availability of the resource should be expressed as dry weight of digestible protein per hectare. Its measurement may entail measuring dry weight of herbage as an intermediate step, but that does not make herbage the resource.

9.3 Kinds of resource

It is necessary at this stage to give a classification of resources, because the interaction between a resource and the animals that depend upon it can take several forms. These in turn influence the dynamics of the population in different ways.

The use of a resource may be *preemptive*. An example is the use of nesting holes by parrots: individuals are either winners or losers. On the other hand, the use of a resource may be *consumptive*. All individuals have access to the resource and each individual's use of it reduces the level available to others. An example is the use of plants by herbivores. We see that both preemptive and consumptive uses of a resource remove a component of that resource from use by other individuals. Consumptive use removes the component permanently, whereas preemptive use removes it temporarily.

Wildlife Ecology, Conservation, and Management, Third Edition.
John M. Fryxell, Anthony R. E. Sinclair and Graeme Caughley.

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To complete the classification, there may be an *interactive relationship* between the population and the resource in that the level of the resource influences the rate of increase of the population, and reciprocally the level of the population's density influences the rate of increase of the resource. The dynamics of the animals interact with the dynamics of the resource. Examples are the relationship between a herbivore and its food supply and the relationship between a predator and its prey. In a *reactive relationship*, however, the rate of increase of the animal population reacts to the level of the resource (as before) but the density of the animals has no reciprocal influence on the rate of renewal of the resource. The relationship between a scavenger and its food supply or between a herbivore and salt licks are examples of reactive relationships.

We start by developing a general theoretical framework that applies in principle to all consumer–resource relationships, regardless of whether they focus on plants and herbivores, carnivores and their herbivorous prey, or all three.

9.4 Consumer–resource dynamics: general theory

The origin of consumer–resource theory can be traced directly to the contributions of two early ecologists: Alfred Lotka and Vito Volterra (Kingsland 1985). Starting from very different backgrounds, these two men simultaneously developed a similar framework for thinking about interactions between consumers and their resources (Lotka 1925; Volterra 1926b), a general framework that is still in common use, albeit with considerable change in biological details. The framework is a set of mathematical expressions for simultaneous changes in the density of consumers (symbolized here by N) and their resources (symbolized by V):

$$\frac{dV}{dt} = \textit{growth of resource} - \textit{mortality due to consumption}$$
$$\frac{dN}{dt} = \textit{growth of consumer due to consumption} - \textit{mortality}$$

In the case of resources, mortality is largely due to consumption, whereas for consumers there is a constant background level. For example, one might model reproduction and mortality by the following equations (Rosenzweig and MacArthur 1963):

$$\frac{dV}{dt} = r_{\max} V \left(1 - \frac{V}{K}\right) - \frac{aVN}{1 + ahV}$$
$$\frac{dN}{dt} = \frac{acVN}{1 + ahV} - dN$$

where $r_{\max}$ is the maximum per capita rate of resource recruitment, K is the resource carrying capacity in the absence of consumers, a is the area searched per unit time by consumers, h is the handling time for each resource item, c is a coefficient for converting resource consumption into offspring, and d is the consumer per capita mortality rate. The particular form of the equations used in this example is based on the most commonly observed patterns. In the absence of consumption (e.g. when $N = 0$), the resource population has a logistic pattern of growth (see Chapter 5). In other words, the resource population is self-regulating. Consumption rates and per capita rates of growth by predators are presumed to increase and level off according to Holling's (1959) Type II functional response (see Chapter 7).

Depending on the parameter values that one uses, this model is capable of a variety of dynamics. In Fig. 9.1, we demonstrate one possible outcome: cyclic fluctuations of both consumers and resources over time.

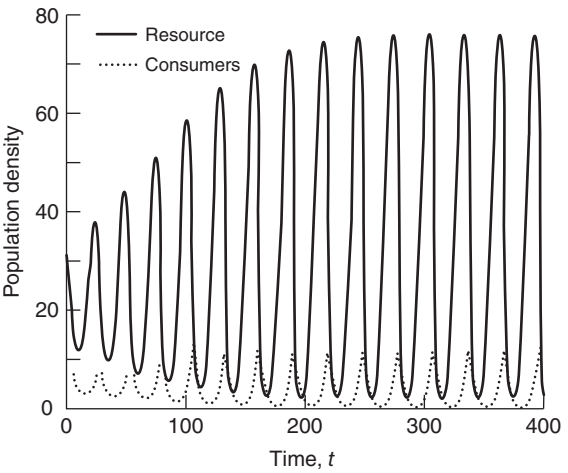

Fig. 9.1 Cyclic dynamics over time for the general consumer–resource model with the following parameter values: $a = 0.1, h = 0.2,$ $c = 0.2, d = 0.3, r_{max} =$ 0.4, and $K = 120$.

Rather than plot densities of both resource and consumer populations against time, ecologists often plot the density of consumers against that of resources. This is known as a phase-plane diagram. For example, in Fig.9.2 we replot the data shown in Fig. 9.1 as such a phase-plane diagram. The phase-plane trajectory in this figure displays a pattern spiraling outward from the starting point until it converges on a repetitive pattern known as a *stable limit cycle*. For most realistic consumer–resource models, this is a common outcome (Rosenzweig 1971; May 1972, 1973). If we had started at values outside the stable limit cycle, we would observe a spiral inward until the trajectory converged once again on the stable limit cycle.

There are some useful additional lines displayed in Fig. 9.2: the null isoclines (sometimes termed "nullclines") for consumers (the vertical line) and resources (the hump-shaped curve). Each of the null isoclines identifies a combination of consumer and resource densities at which $\frac{dV}{dt} = 0$ or $\frac{dN}{dt} = 0$ (i.e. one or the other population is unchanging). By rearranging our equations for simultaneous change in consumer and

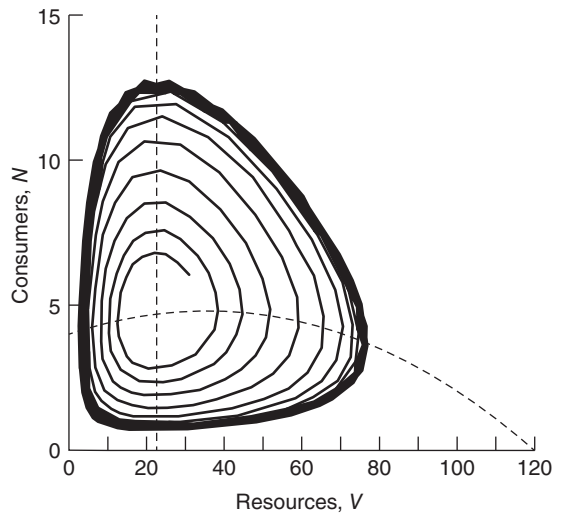

Fig. 9.2 Data from Fig. 9.1 replotted as a phase-plane diagram of consumers versus resources.

resource density, we can show that this will be the case when:

$$N = \frac{r_{\max} V \left(1 - \frac{V}{K}\right)}{\frac{aV}{1+ahV}}$$

In other words, at any of the consumer and resource combinations lying on the hump-shaped isocline, consumption exactly matches the rate of resource production, so resource density will be unchanging.

Similarly, at the resource density shown by the vertical dotted line, the consumer population acquires just enough resources to allow it to balance mortality with offspring production. This occurs when:

$$V = \frac{d}{a(c - dh)}$$

In this case, there is only one sustainable combination of consumer and resource densities at which both are unchanging: the point of intersection of the two null isoclines. If we somehow set both populations to this equilibrium point, they would stay there. Slight deviation from the equilibrium leads to spiraling outwards of the consumer–resource trajectory until the stable limit cycle is reached (Fig. 9.2). Hence, the coexistent equilibrium is dynamically unstable, at least for these parameter values. Other trivial equilibria are also present: when both N and $V = 0$ and when $N = 0$ and $V = K$. These equilibria are also unstable for the parameter combination shown in Fig. 9.2.

For other parameter values, a second sustainable outcome is possible: a stable equilibrium for both consumers and resources (Fig. 9.3). The only difference between the models plotted in Fig. 9.2 and Fig. 9.3 is the carrying capacity of resources. A decrease in the resource carrying capacity tends to be stabilizing, while its enrichment tends to be destabilizing. This has been termed the "paradox of enrichment," whereby provision of a better resource environment leads only to destabilization of consumers (Rosenzweig 1971).

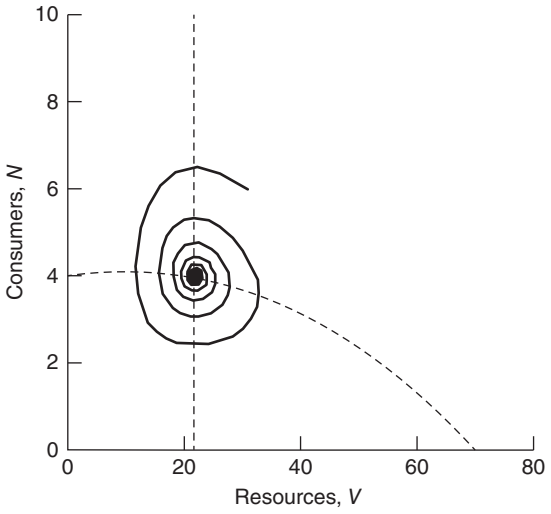

Fig. 9.3 Phase-plane diagram of the dynamics over time for a stable form of the consumer–resource model with the following parameter values: $a = 0.1, h = 0.2, c = 0.1, d = 0.3, r_{\max} = 0.4$, and $K = 70$.

Although the complete explanation for this phenomenon is complex, the system can be usefully viewed as reflecting a dynamic tension between stabilizing influences (such as self-regulation by resources) and destabilizing influences (such as consumption of resources). The reason that consumption tends to be destabilizing is that the per capita risk of resource mortality for a given consumer density is inversely related to resource density (see Chapter 5). Hence, an increase in resource levels leads to diminished risk of death, which imparts a positive, rather than negative, feedback on population dynamics. When the carrying capacity is small, the consumer null isocline lies close to the resource carrying capacity, to the right of the hump in the resource null isocline. This is a region where stabilizing influences are stronger than destabilizing ones. In contrast, when the carrying capacity is large, the consumer null isocline lies far from the carrying capacity, where destabilizing influences hold sway.

With other parameter combinations, consumers would be unable to persist, simply because the intake of resources at any resource level would be unable to compensate for mortality. The possible outcomes of this consumer–resource model depend entirely on the parameter values. Predicting the outcome of even this highly simplified representation requires detailed knowledge of the magnitude of ecological parameters. We now go on to illustrate how this approach can be applied to a well-studied system: red kangaroos and their food plants in Australia.

9.5 Kangaroos and their food plants in semi-arid Australian savannas

9.5.1 *Plant dynamics*

The dynamics of a renewable resource can be quite complicated, containing elements of seasonality, intrinsic growth pattern, and the modification of the two by the animals using the resource. To clarify some general issues, we shall consider in detail a well-studied example: the growth of the herbage layer fed upon by kangaroos in the arid zone of Australia.

Fig. 9.4 shows Robertson's (1987) estimate of the *plant growth response*: growth by ungrazed herbacious plants in response to rainfall. He sampled growth rates on a kilometer grid over 440 km^2 of the arid zone of Australia. The measurements were repeated every 3 months for 3.5 years and rainfall was recorded for each 3-month interval. The curve labeled 60 mm indicates that the higher the biomass at the start of the 3-month period, the lower the increment of further biomass added over the next 3 months. This is to be expected as plants compete for space, water, light, and nutrients.

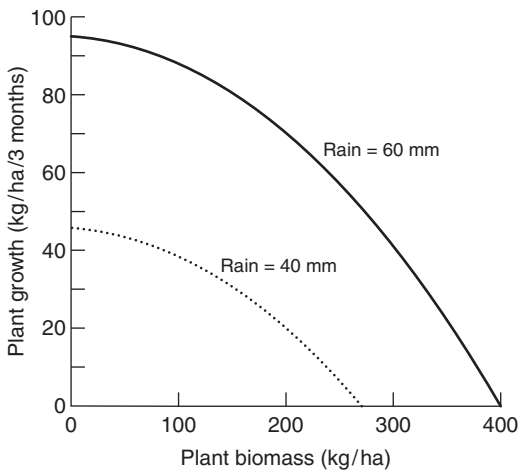

Fig. 9.4 Plant growth rate as a function of plant biomass at the beginning of the interval and rainfall during the interval, for pastures in Kinchega National Park. (After Robertson 1987.)

Table 9.1 100 years of rainfall statistics at central Australian study site.

	Mean (mm)	Standard deviation (mm)
Dec–Feb	62	59
Mar–May	57	47
Jun–Aug	59	34
Sep–Nov	61	44
Annual	239	107

The 60 and 40 mm curves shown in Fig. 9.4 are part of a family of curves each representing that trend for a given rainfall over 3 months. We can summarize the figure by saying that the higher the rainfall, the higher the growth increment, but that for a given rainfall, the higher the starting biomass, the lower the growth increment. Hence, the rate of plant growth is influenced by both rainfall and plant biomass at the beginning of the period.

Fig. 9.4 is a graphical representation of a regression analysis that estimated the function $\alpha(V, R)$, showing how incremental plant growth (in kg/ha) measured over the next 3 months is related to vegetation biomass (V, in kg/m^2) and rainfall (R, in mm):

$$\alpha(V, R) = -55.12 - 0.01\,535V - 0.00\,056V^2 + 2.5R$$

Unlike the logistic model, plant growth in the Australian study was highest at low levels of abundance, rather than at intermediate levels (Chapter 5). This is probably due to there being an ungrazeable plant reserve belowground. At low levels of plant abundance, rapid regrowth is enabled by translocation from these belowground tissues. Such an ungrazable refuge tends to lend a stabilizing influence to the interaction, as we shall shortly see.

Table 9.1 summarizes 100 years of rainfall statistics for the central Australian study site. There was no significant correlation of rainfall from one quarter to the next, nor between consecutive years.

9.5.2 *The functional response of kangaroos to plant abundance*

Having established how fast the resource grows in the absence of grazing and browsing, we now need to know what happens to it when a herbivore is present. The amount a herbivore eats per unit time is a constant only when it is faced by an *ad libitum* supply. Herbivores are seldom so lucky. The trend of intake against food availability is therefore curved, being zero when the level of food is zero and rising with increasing food to a plateau of intake. From there on, no increase in food supply has any effect on the rate of intake, because the animal is already eating at its maximum rate. Such a curve is called a *functional response* or *feeding response*: the trend of intake per individual against the level of the resource (see also Chapter 7). It can be represented symbolically by an equation such as:

$$\beta(V) = c(1 - \exp(-bV))$$

where $\beta(V)$ is plant consumption as a function of plant biomass, c is the maximum (satiating) intake, V is the plant biomass, and b is the slope of the curve, a measure of grazing efficiency. The last has another meaning: its reciprocal $1/b$ is the level of the resource V at which 0.63 (i.e. $1 - e^{-1}$) of the satiating intake is consumed.

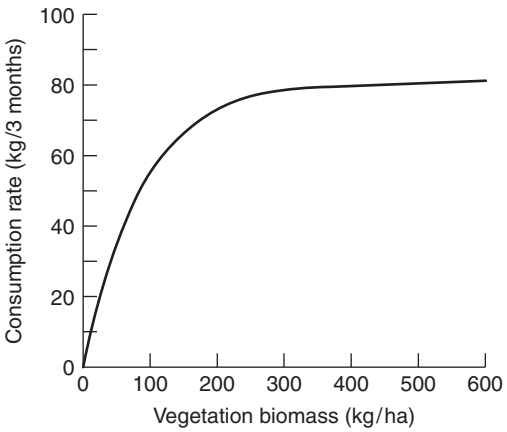

Fig. 9.5 Food intake per individual red kangaroo per day at varying levels of food availability. (After Short 1987.)

Fig. 9.5 shows the dry-weight food intake (I) of a red kangaroo at various levels of pasture biomass when it is grazing annual grasses and forbs interspersed with scattered shrubs (Short 1987). The equation for a 35 kg kangaroo is:

$$\beta(V) = 86(1 - \exp(-0.029V)$$

where I is the intake of food per red kangaroo measured in kilograms dry weight over 3 months, assuming no shrubs in the pasture layer (Short 1987). The satiating intake is 86 kg/3 months, occurring when pasture biomass exceeds 300 kg/ha.

Short (1987) estimated these two functional responses by allowing high densities of kangaroos and rabbits to graze down pasture in enclosures, the offtake per day being estimated as the difference between successive daily estimates of vegetation biomass corrected for trampling. Daily intake could be estimated for progressively lower levels of standing biomass because the vegetation was progressively defoliated during the experiment. We scale up this daily intake rate to intake per 3 months in order to maintain a similar time frame as for the plant growth data.

Although the functional response has been discussed here in the context of a plant–herbivore system, all of this discussion carries over to prey–predator systems. They are exactly analogous. The only difference lies in the difficulty of measuring a predator's food intake. The ability to measure intake by way of radioactive tracers has greatly simplified this problem. A good example is Green's (1978) use of radio-sodium to estimate how much meat a dingo eats in a day.

9.5.3 *The numerical response of kangaroos to plant abundance*

The functional response gives the effect of the animal upon a consumable resource. In contrast, the *numerical response* gives the effect of the resource on the change in animal numbers. If the resource is used in a preemptive rather than a consumptive way (e.g. nesting holes used by parrots) then it may be adequate to represent the numerical response by consumer density of the animals against the level of the resource (e.g. nesting holes per hectare). If the animals' use of the resource is consumptive, however, then the relationship between the animals and the resource is best portrayed as the instantaneous rate of population increase against the level of the resource.

Fig. 9.6 shows the numerical response relationship between the rate of increase of red kangaroos and the biomass of pasture. Bayliss (1987) estimated rates of increase

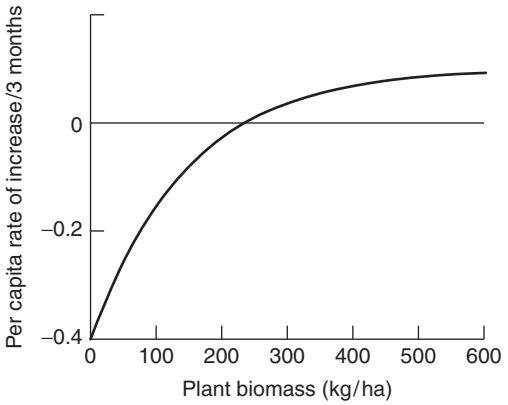

Fig. 9.6 Rate of increase of red kangaroos on a 3-monthly basis in relation to food availability. (After Bayliss 1987.)

from successive aerial surveys and pasture biomass from ground surveys. As with the functional response, the numerical response has an asymptote: there is an upper limit to how fast a population can increase and no extra ration of a resource will force that rate higher. The numerical response differs from the functional response in that negative values are both possible and logically necessary. If they were not, the population would increase to infinity.

The numerical response can usually be described by an equation of the form:

$$r(V) = -d + a(1 - \exp(-fV))$$

where r is the exponential rate of increase of the animals, d is the maximum rate of decrease, and a is the maximum extent to which that rate of decrease can be alleviated. Hence $a - d = r_{max}$ is the maximum rate of increase. Demographic efficiency, the ability of the population to increase when resources are in short supply, is indexed by f. For the present example, the constants were estimated (Bayliss 1987, modified by Caughley 1987) as:

$$r(V) = -0.4 + 0.5\ (1 - \exp(-0.007V\ ))$$

The maximum rate of increase (i.e. when vegetation abundance is maximal) on a 3-monthly basis is $0.5 - 0.4 = 0.1$. Note that we have calculated the parameters for growth over 3 months, in order to remain consistent with the timeframe for other parameters used in the model. On an annual basis, r_{max} can be scaled up by simply multiplying by the four quarters in the year: $r_{max} = 0.4$. Hence, the population's maximum finite rate of increase over a year $\lambda = \exp(0.4) = 1.49$, a 49% increase per year.

9.5.4 *Plant–kangaroo dynamics*

So far we have taken a plant–herbivore system and dissected it into its component processes: plant growth, the herbivore functional response to changes in plant biomass, and the numerical response of the herbivore, in terms of its rate of increase, to the biomass of the plants.

The evaluation of these component influences upon a population's dynamics provides two bonuses. First, it furnishes a tight summary of the dynamic ecology of the system. Second, it provides that summary in terms of causal relationships rather than correlations.

The numerical response of the herbivore allows us to calculate the equilibrium level to which plant biomass will converge in a constant environment under the influence of an unrestrained population of herbivores; that is, the null isocline. It is the x -intercept of the regression of rate of increase of the herbivores against plant biomass or, put another way, the plant biomass at which rate of increase of the herbivore is zero (see Fig. 9.6). The level of plant biomass V at which this occurs is:

$$V = (1/0.007)\ln(0.5/(0.5 - 0.4))$$

which equals 230 kg/ha dry-weight plant biomass.

This value is immensely important ecologically. It is the equilibrium level of plant biomass imposed by grazing in a constant environment. This is of some theoretical interest but of limited practical importance, because environments are not constant. However, it is also the level of plant biomass above which the herbivore population will increase and below which it will decrease (the *critical threshold*), and that is true whether the environment is constant or variable and whether the density of herbivores is high or low.

Using similar logic, we can calculate the combination of kangaroo and plant densities at which consumption exactly matches regrowth by plants. This will occur when $N\beta(V) = \alpha(V, R)$. We can rearrange the terms to isolate kangaroo density on the left-hand side of the equation, $N = \alpha(V, R)/\beta(V)$. Both null isoclines for the kangaroo–plant system are plotted as dotted lines in Fig. 9.7.

We can now reassemble the response functions of the system in their proper relationships to examine dynamics in the absence of stochastic variability in rainfall. We shall see what these in combination reveal about the system's dynamic behavior:

$$\frac{d}{dt}V(t) = \alpha(V, R) - N(t)\beta(V)$$
$$\frac{d}{dt}N(t) = N(t)r[V(t)]$$

Under a constant-rainfall regime (in this case, 60 mm per 3-month period), the system converges on the equilibrium: the point of intersection of both null isoclines (Fig. 9.7).

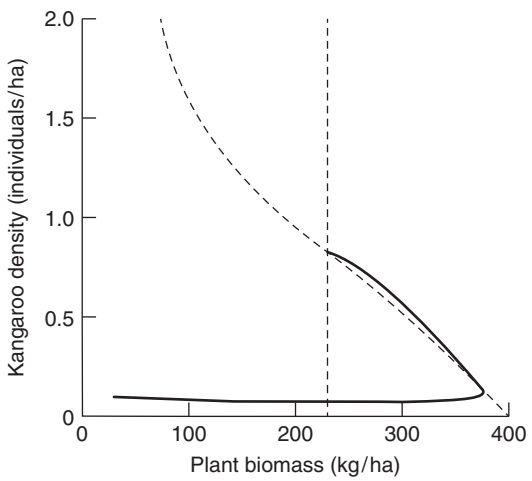

Fig. 9.7 Dynamics of kangaroos and plants over time, based on the Australian model discussed in the text.

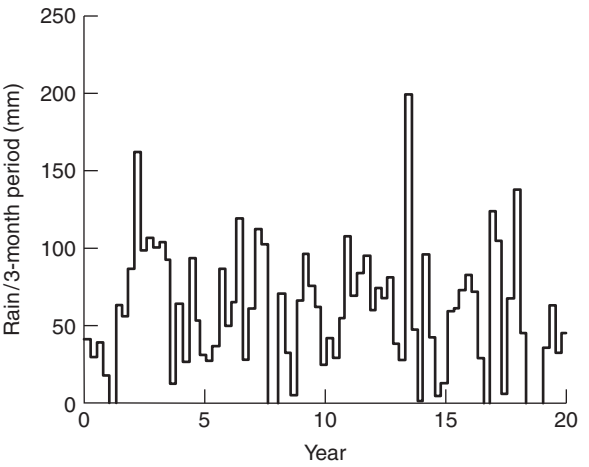

Fig. 9.8 A typical stochastic series of rainfall amounts per 3-month period drawn from a normal distribution with mean and variance equivalent to the Australian data. (After Caughley 1987.)

This shows that the equilibrium is stable, as one might have guessed based on the negative slope of the plant null isocline. Convergence on the equilibrium is circuitous, involving a burst of plant growth followed by plant decline as the kangaroo population stabilizes.

Rainfall can also be simulated as a sequence of random events from a normal distribution with a mean and standard deviation identical to the Australian data (Fig. 9.8), and the consequent changes in plant biomass and kangaroo numbers can be calculated accordingly.

Fig. 9.9 demonstrates a typical time trend for kangaroos, as generated by the equations describing the unpredictable rainfall (Fig.9.8) and the responses to it of the plants and herbivores. The only external input other than starting conditions is the random values from the 3-monthly rainfall distributions whose observed means and standard deviations are given above. The kangaroo population trajectory is a mathematical consequence of that rainfall, as its effect feeds through to plant growth, herbivore population growth, and grazing pressure.

The rainfall of this region takes the form of high-amplitude, high-frequency fluctuations (Fig. 9.8). The herb layer, whether grazed or ungrazed, generates a similar trace of high-amplitude, high-frequency fluctuations as it reacts speedily to rainfall or the lack

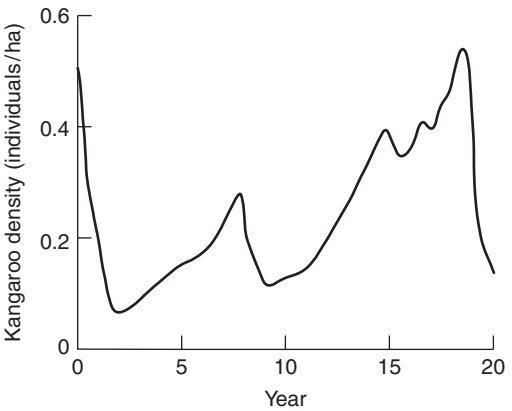

Fig. 9.9 A typical stochastic time series for kangaroos, using the model discussed in the text, for the rainfall sequence shown in Fig. 12.8.

thereof. The fluctuations are paralleled by similar but more constrained fluctuations in the kangaroos' rate of increase as the population reacts dynamically to variations in food supply. The trend of kangaroo density differs from the rainfall regime, comprising fluctuations of high amplitude but of low frequency. This result might have been predictable from first principles: present density is an integration of past rates of increase, not of present conditions. Initial conditions are not highly influential: the system remembers previous plant biomass for only 3 years, but the memory of kangaroo density can linger for 10. As a consequence of the slow tracking of resources by kangaroos, there is a substantial time lag in their response to changing climatic conditions. This lag imparts an irregular fluctuation over time, rather than constancy in abundance, despite the stability of the system under deterministic (constant climatic) conditions. Caughley (1987) has christened systems that show slow convergence on stochastically shifting equilibria as "centripetal."

9.6 Wolf–moose–woody plant dynamics in the boreal forest

9.6.1 *Models of the tri-trophic system*

Few natural systems have been studied in sufficient detail to supply all the necessary parameters that we observed in the Australian kangaroo and plant system. Fortunately, it is often possible to estimate plausible parameter values from allometric reasoning or historical data from a variety of sources, allowing us to make educated guesses about system dynamics in a generic sense (Yodzis and Innes 1988; Turchin 2003). We shall demonstrate this approach for moose, wolves, and woody plants in the boreal forests of North America. This is an important system to understand, because it occurs across much of the extensive boreal forest biome spanning North America. We use Turchin's (2003) parameter estimates for the interactive model.

First, we recognize from the outset that this is fundamentally a tri-trophic system, meaning that there are three trophic levels that interact in the food chain. The framework we shall use simply expands the consumer–resource model outlined at the beginning of the chapter to a third trophic level (P, for wolves), which feeds on the second (N, for moose), which itself feeds on self-regulating plant resources (V). In all cases, we shall measure density in biomass (plants) or numerical abundance (for animals) per square kilometer. Mathematically, we can represent this interaction with the following system of equations:

$$\frac{dV}{dt} = r_{\max}\left(1 - \frac{V}{K}\right) - \frac{aVN}{b+V}$$
$$\frac{dN}{dt} = \frac{aeVN}{b+V} - dN - \frac{ANP}{B+N}$$
$$\frac{dP}{dt} = \frac{AENP}{B+N} - DP$$

where a is the maximum rate of plant consumption by a single moose, b is the plant biomass at which plant consumption is half of the maximum, d is the rate of plant consumption at which moose just sustain themselves, e is the efficiency of conversion of food intake into new moose, and A, B, D, and E represent the same set of parameters with respect to wolves.

We should note the similarity between the tri-trophic equations and the simpler consumer–resource model outlined at the beginning of the chapter. Resources have a self-regulating growth term, where the density-dependent term $(1 - V/K)$ reduces the growth rate proportionately with plant biomass. Plant consumption by moose is balanced against this positive contributor to resource abundance, with plant consumption

expressed as the Michaelis–Menten form of the Type II functional response. Moose have a per capita growth function that depends on their intake of plants. Balanced against this is moose consumption by wolves. Finally, wolves have a per capita growth function that depends on their intake of moose, balanced against a constant per capita rate of mortality (presumably due to things like accidents, disease, and old age).

9.6.2 *Parameter estimation for the wolf–moose–woody plant system*

For a large part of the year, moose browse on the leaves and twigs of woody plants. Many species of plant contribute to the food supply of moose (Belovsky 1988). However, we know little about the web of ecological interactions within this plant guild, so we shall consider woody browse during winter (the period of the year when food is most often limiting to moose) as a single category. Edible biomass (measured in Mg/km^2) is symbolized by V. Field data suggest that it is rare to observe higher browse availability than $100\,g/m^2$, which is equivalent to $K = 100\,Mg/km^2$. Maximum moose density is thought to be 2 moose/km^2 (Messier 1994) and woody plant r_{max} is estimated as $3.33\,Mg/km^2$/year (Turchin 2003). We see that the growth term for the edible plant biomass is maximized at low biomass, not at intermediate biomass as it would be for a logistic growth function. The rationale for low edible biomass is that moose have access only to regrowing tissues, such as twigs and leaves, so that the rest of the plant functions as an ungrazeable reserve. Regrowth capacity should not be inversely affected by herbivory so long as it does not jeopardize plant survival. The indigestible component is the same kind of refuse demonstrated in Robertson's study of food plants fed upon by kangaroos in semi-arid Australian grasslands.

The maximum rate of plant consumption by moose is set at 2 Mg/individual/year, based on maximum values quoted in the feeding studies literature (Crête and Bédard 1975). Fitting various curves to Vivås and Sæther's (1987) studies of moose foraging in Norway suggests a foraging efficiency of $b = 40\,Mg/km^2$. Moose can just meet their metabolic requirements at a level of intake of half the maximum, providing an estimate of $d = 1\,Mg/ind/km^2$. Given a maximum exponential rate of increase of 0.2 for moose (Fryxell *et al*. 1988a) and values for all the other parameters, one can solve for e using the following relationship:

$$e = \frac{r_0}{\frac{aK}{b+K} - d}$$

yielding $e = 0.467$.

Rates of wolf consumption of moose are modeled as a Type II functional response, based on Messier's (1994) review of several moose–wolf studies throughout North America. Each of these studies provides one or more estimates of rate of consumption by wolves at a given moose density. By combining all of the recorded data together in a single graph (Fig. 9.10), Messier was able to illustrate one of the most difficult kinds of ecological relationship, the functional responses of large organisms under free-living conditions. Such patterns are essential to our understanding of consumer–resource interactions, yet are prohibitively costly to gather in a single study. Use of aggregate data is a very useful way of solving this problem.

Scaling the wolf consumption rate to a yearly timeframe yields estimates of $A =$ 12.3 moose/wolf/year and $B = 0.47$ moose/km^2. According to Fuller and Keith (1980), each wolf needs to eat 0.06 kg of meat per day to meet maintenance requirements, whereas a population whose individuals eat 0.13 kg each day can grow at the maximal rate. This yields estimates of $D = 0.6$ and $E = 0.1$.

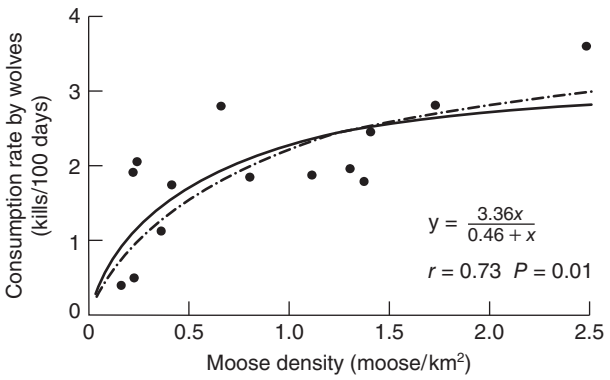

Fig. 9.10 The consumption rate by wolves of moose in relation to moose density. (After Messier 1994.)

9.6.3 *Dynamics of the wolf–moose–woody plant system*

Combining these parameter values together, the outcome is a complex series of oscillations in moose and wolf abundance, which never quite repeat themselves (Fig. 9.11). This is a mild form of deterministic chaos, common in tri-trophic systems (Hastings and Powell 1991; McCann and Yodzis 1994). Even though the fluctuations are

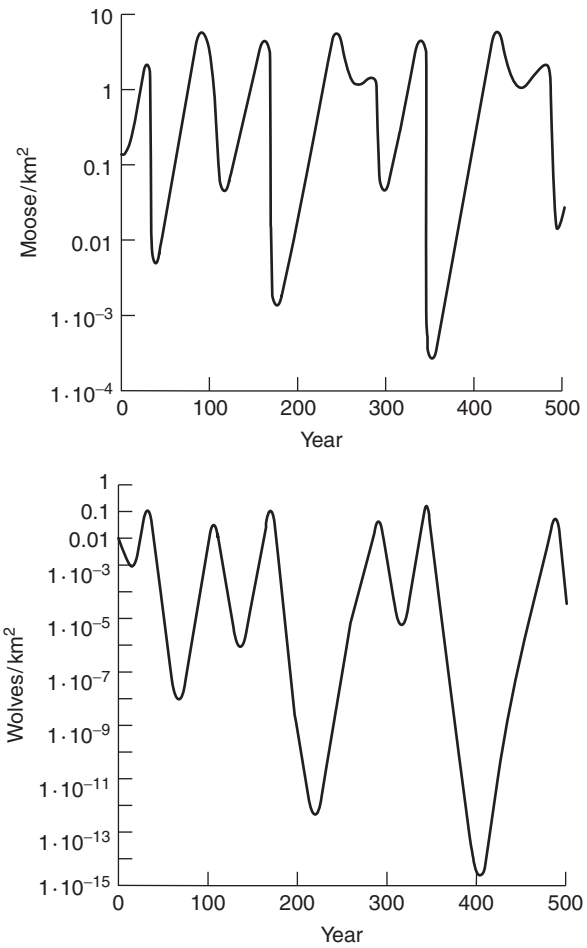

Fig. 9.11 Predicted population dynamics of moose (top) and wolves (bottom) based on Turchin's (2003) tri-trophic model, described in the text.

nonrepetitive, the time between successive peaks tends to be several decades – a very protracted pattern of fluctuation.

The manner by which parameters for the wolf–moose–woody plant model were derived, using a set of observations gathered around the globe, makes it fairly unlikely that we can predict the dynamics of any given system. It does suggest, nonetheless, that this system should exhibit an inherent tendency towards protracted fluctuations that recur over a decade-long timescale. Moreover, the model suggests that these fluctuations will not necessarily converge on a stable limit cycle, as do consumer–resource models with only two trophic levels. Rather, we may expect to see inconsistency as each population progresses from peak to peak.

One obvious objection to this model is that it ignores the role of wolf territoriality. In most landscapes, wolves form communal packs that partition the available habitat among themselves. Territorial strife among wolf packs can be intense, leading to substantial levels of mortality (Peterson *et al*. 1998). At least we should expect that the risk of this mortality should climb with wolf density, if only because of increasing frequency of encounters between members of different packs. One way to incorporate this effect is to make wolf mortality explicitly density-dependent:

$$\frac{dV}{dt} = r_{\max}\left(1 - \frac{V}{K}\right) - \frac{aVN}{b+V}$$
$$\frac{dN}{dt} = \frac{aeVN}{b+V} - dN - \frac{ANP}{B+N}$$
$$\frac{dP}{dt} = \frac{AENP}{B+N} - DP - \frac{s_0 P^2}{\gamma}$$

where the maximum density of wolves (recorded from field studies) $\gamma = 0.1$ and the maximum per capita rate of wolves $s_0 = 0.4$. This modification imposes an additional per capita mortality term that increases by s_0/γ with each unit increase in wolf density P.

Territorial effects of this sort often have a stabilizing influence. Such is the case with the wolf–moose–woody plant model: the addition of density-dependent mortality due to territorial strife changes the dynamics of the system from deterministic chaos to a stable limit cycle (Fig. 9.12). The level of strife is insufficient, however, to completely stabilize the system.

The best long-term data set available on both moose and wolves is from Isle Royale, a small island 40 km off the coast of Canada in Lake Superior that supports a mix of deciduous and coniferous vegetation species typical of the boreal forest on the mainland. Moose apparently invaded Isle Royale a century ago, while wolves arrived by ice in the 1940s. Estimated patterns of abundance on Isle Royale certainly suggest protracted fluctuations over time (McLaren and Peterson 1994; Peterson 1999; Post *et al*. 1999), with moose populations fluctuating slowly with 25 years between successive peaks (Fig. 9.13).

It is difficult to conclusively tell from the Isle Royale time series data whether the system is cyclic or chaotic, because they are simply insufficient to evaluate even such a well-studied system. Such will nearly always be the case in slow-changing wildlife species. Nonetheless, the tri-trophic model seems to capture the fluctuating tendency of the Isle Royale system.

There are many other factors that could also contribute to the apparent instability of the Isle Royale populations. For example, complex changes over time in the age

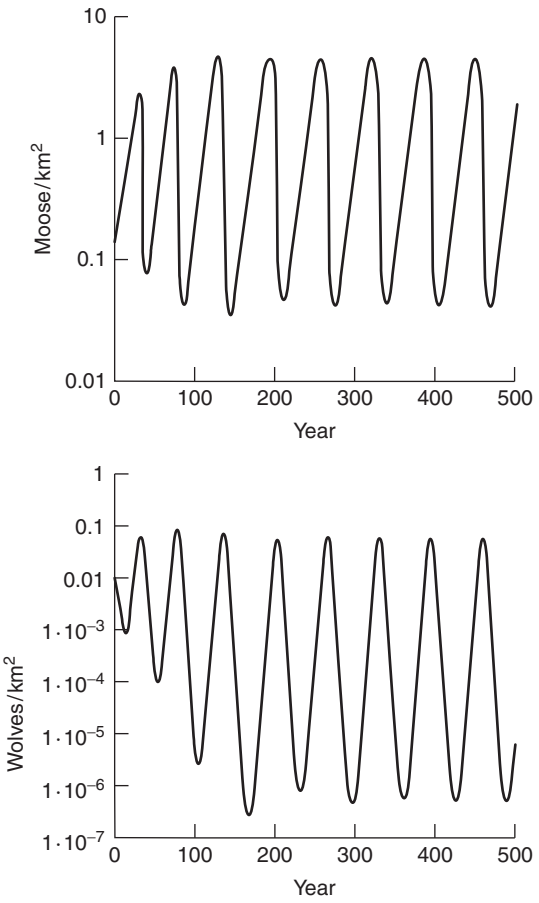

Fig. 9.12 Predicted dynamics of the moose (top) and wolf (bottom) system with woody plants when wolves have additional density-dependent mortality due to territorial aggression, as described in the text.

structure of moose could contribute to the propensity for fluctuations (Peterson and Vucetich 2003). Wolves are highly selective for specific age classes of prey, so changes in age distribution could translate into substantial changes in predation risk. As we discuss in Chapter 13, it can take many years for age distributions to stabilize in long-lived organisms. When age distributions are shaped by dynamic interactions with predators, this can be even more destabilizing. We also know that the wolf population on Isle Royale has much lower levels of genetic variability than do populations on the mainland (Wayne *et al*. 1991). This could influence wolf demographic parameters in unknown ways. Finally, there is evidence that complex interactions among climatic conditions, social grouping patterns of wolves, and predation risk of moose could contribute to instability. In years of deep snow, wolves form larger packs, which leads to increased rates of mortality on moose (Post *et al*. 1999). Nonetheless, the instability of this system seems to be intrinsic to the basic consumer–resource interactions (moose–vegetation and wolf–moose).

Truly long-term data for temperate-zone carnivores (wolves and coyotes) and ungulates (moose or white-tailed deer, depending on location) are scarce. Data from the Hudson's Bay Company probably represent the lengthiest data set. They suggest very slow oscillations in the abundance of wolves and coyotes during 1750–1900,

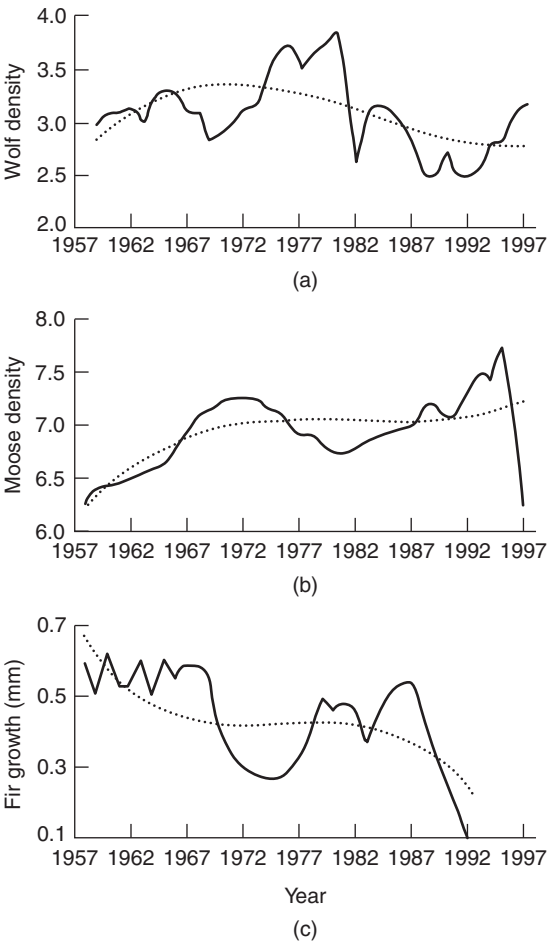

Fig. 9.13 Population dynamics of wolves (a) and moose (b), as well as annual growth of balsam fir trees (c), on Isle Royale. The solid lines represent observed values, the dotted lines polynomial regressions. The densities represent total abundance of each species recorded over the entirety of Isle Royale. (After Post *et al*. 1999.)

with roughly two cycles per century (Turchin 2003). Although the Hudson's Bay data on deer skins are more fragmentary, they too suggest long-term cycles in abundance (Turchin 2003). Slow oscillations by white-tailed deer in Canada (Fryxell *et al*. 1991) and moose in Finland (Lehtonen 1998) suggest that long-term oscillations are an important feature of some large mammal species.

9.7 Other population cycles

Long-term data for a number of other wildlife populations show pronounced cycles, first identified by Charles Elton (1924). Such cycles are sometimes regular, such as the 10-year cycle of snowshoe hares (Sinclair *et al*. 1993), sometimes erratic, such as the 3–6-year cycle of voles (*Microtus agrestis* and *Clethrionomys rufocanus*) in northern Europe (Turchin and Hanski 1997). They can be explained in many ways: unstable behavioral polymorphisms in cyclic populations (Chitty 1967; Krebs and Myers 1974); maternal effects transferred to offspring, imparting lagged density dependence (Inchausti and Ginzburg 1998); coupled interactions between plants, herbivores, and/or carnivores (Hansson 1987; Turchin and Hanski 1997; Turchin and Ellner 2000; Turchin and Batzli 2001). We shall review the northern European vole and

North American snowshoe hare populations here and consider the logic underlying consumer–resource explanations for population cycles.

Some of the longest continuous studies of vole populations come from sites in Scandinavia, Finland, and Russia (Turchin 2003). These data point to a fascinating geographical pattern: populations at southern latitudes show little evidence of repetitive, cyclic dynamics, whereas populations from more northerly latitudes exhibit repetitive cycles or perhaps even chaotic dynamics over time (Fig. 9.14).

Many ecological variables change as one progresses from the Arctic Circle to more southerly latitudes, including temperature maxima and minima, precipitation, vegetation cover and composition, primary productivity, mammalian and avian community diversity, and human population density. Most important among these variables, however, is the transition from a suite of generalist predators (red foxes, feral cats, badgers, and various owls, hawks, and other raptors) in the south to a narrow range of specialist predator species (primarily the least weasel, *Mustela nivalis*) in the most northerly areas. The abundance of generalist predators declines in northern latitudes because of the duration and depth of snow cover (Hansson and Henttonen 1985).

Least weasels exhibit a Type II functional response to changes in vole density (Erlinge 1975). As we have already discussed, this pattern of foraging tends to destabilize prey populations, because the per capita risk of mortality due to predators is inversely related to prey density. However, generalist predators switch feeding preferences to favor voles when they reach high density but ignore them when they collapse to low density (Korpimäki and Norrdahl 1991; Erlinge *et al*. 1983). As we show in Chapter 10, switching behavior can stabilize prey numbers, because the per capita risk of mortality for prey due to predation increases with prey density, at least over some prey densities. Because generalist predators can feed on a wide variety of other species, they may not be dependent on vole numbers (Turchin and Hanski 1997). In the absence of predators, vole population growth is self-regulating, due to density-dependent resource limitation and territorial spacing among individual voles.

Turchin and Hanski (1997, 2001) linked specialist predation by weasels, generalist predation, and the self-regulating population dynamics of voles with seasonal changes in vole logistic growth. In keeping with the empirical data, their model predicts (better than alternative models) complex cycles or chaos when generalist predators are rare but much more stable dynamics when they are common.

Data on the cyclical variation in abundance of snowshoe hares comes from fur records of the Hudson's Bay Company in Canada (see Fig. 5.7). These data show a regular oscillation of numbers, with a period of 10 years. Like the other examples we have discussed, snowshoe hares interact not only with their food supplies but also with a suite of carnivores that feed upon them (Krebs *et al*. 2001b). Some of these carnivores, especially the lynx, which is a specialist predator on hares, also display a 10-year cycle in abundance, slightly lagged behind that of the snowshoe hare. These characteristics suggest that the tri-trophic model might be a useful starting point in modeling the dynamics of hare and lynx populations. King and Schaffer (2001) estimated parameters in order to model dynamics of the woody plant–hare–lynx interaction. They found that realistic parameter values generated cycles in hare and lynx abundance of 8–12 years, consistent with the historical data.

Unlike in the other examples we have discussed, however, there is an inherent environmental cycle, the 11-year sunspot cycle, that apparently plays a crucial role in generating the hare–lynx cycle (Sinclair *et al*. 1993). Snow depth is strongly

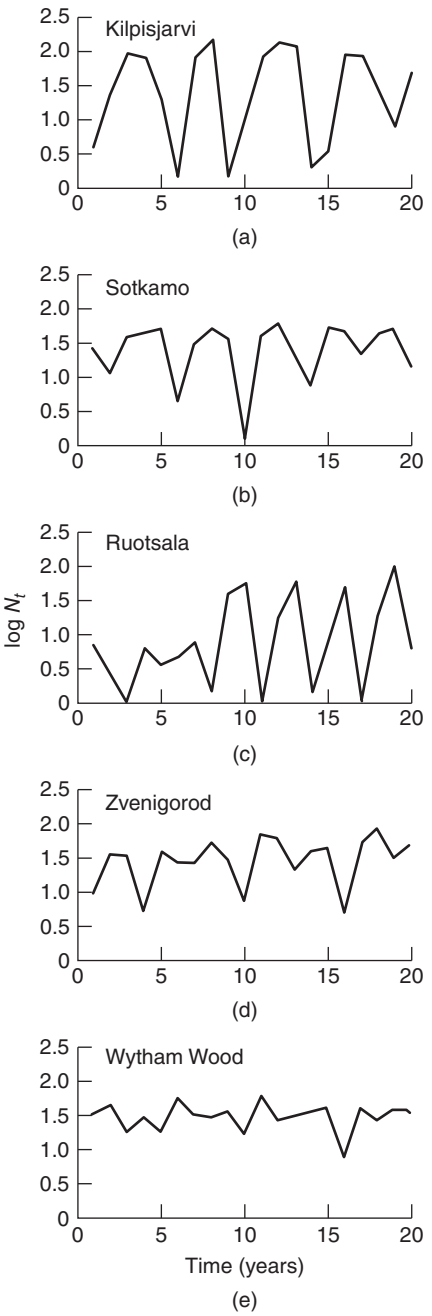

Fig. 9.14 The latitudinal gradient in vole dynamics across northern Europe, with the most northerly sites at the top of the figure. (After Turchin and Hanski 1997.)

influenced by the sunspot cycle, as evidenced by ice cores taken from glaciers. Disentangling the effect of the sunspot cycle from the endogenous rhythm of the tri-trophic consumer–resource interaction presents a sizeable challenge.

King and Schaffer (2001) also used the tri-trophic model to explain the outcome of a series of large-scale field experiments conducted in Kluane National Park, Canada,

during the 1980s and 90s (Krebs *et al*. 1995, 2001b). The Kluane study involved experimental manipulations of food availability, predation risk, and both factors combined to tease apart bottom-up versus top-down trophic mechanisms. The Kluane team found that each of the manipulations had a considerable effect on hare densities and hare demographic rates. Food addition doubled hare densities and predator exclusion trebled them. Both had an 11-fold effect relative to controls. The clear implication is that both bottom-up and top-down processes are important to the natural regulation of snowshoe hares. Despite these results, however, none of the treatments dismantled the hare cycle. This may be because of the use of semi-permeable fencing in the experimental treatments, allowing hare populations within the exclosures to be driven by dynamics generated outside, via immigration.

The best interpretation of the existing information is that the snowshoe hare–lynx cycle is a complex tri-trophic interaction synchronized to some degree by the exogenous environmental rhythm of the sunspot cycle. These results suggest that coupled consumer–resource models can be a vital step in understanding the complex patterns of population dynamics that occur in natural ecosystems.

9.8 Summary

A resource is something an animal needs and whose consumption diminishes its availability to other consumers. Consumers and their resources often form a system in which the rate of increase of the resources is determined by the density of the animals eating them, and the rate of increase of the animals is determined by the density of the resources. Such a complex system can be studied only by breaking it down into its dynamic components, of which three dominate. First, there is the functional response of the animal, the rate of resource intake by a single consumer as a function of resource abundance. Second, there is the numerical response of the consumer, the rate at which its population increases as a function of the resource abundance. Finally, we require supplementary information on the growth rate of resources in relation to resource abundance. On the basis of these functional relationships, the full dynamic behavior of the system can be described. We illustrate this approach with two well-studied consumer–resource systems: kangaroos and their plants in Australia, and wolves, moose, and woody plants in North America. Interactive systems with these components can be deterministically stable (such as the Australian plant–kangaroo system) or unstable (such as the wolf–moose–woody plant system). Deterministic instability is evident in the repetitive population fluctuations (stable limit cycles) and in the nonrepetitive ones (deterministic chaos). Even stable food-chain models can show pronounced long-term fluctuations in response to stochastic environmental variability (centripetal systems). Two well-documented cyclical populations (voles in northern Europe and snowshoe hares in North America) have dynamics consistent with predictions of coupled consumer–resource models.