1 · *Introduction*

Ring a ring of roses,
A pocket full of posies,
Atishoo, atishoo,
We all fall down.

Sporadic reports appear of a mysterious disease afflicting people in a far-off country. Within weeks the disease has spread to towns and cities and is reaching epidemic proportions in that country, and within months it has circulated around the world. The origins of this new disease are initially unclear but, mysteriously, large-scale die-offs of wildlife and domestic animals presage the outbreaks in several countries. Many people and animals die; furthermore, we start to see changes throughout natural communities, involving the resources and consumers of afflicted species. Eventually the disease dies out in humans and domestic stock, and the infection, if it persists, goes largely unnoticed in a handful of wildlife species. What was going on? Could we prevent it happening again, and will there be long-term consequences for natural communities? This is the plot behind many B-movies, but also something that happens in reality all the time. For example, the recent sporadic outbreaks of highly pathogenic avian flu involve transmission though a suite of wildfowl and domestic bird species, with occasional spillover into man. At the turn of this new century, as West Nile virus (WNV) spread throughout the United States, its arrival in a new county was heralded by reports of dead and dying birds, also host to the virus. The spread of chestnut blight through the deciduous forests of northern America at the turn of the previous century changed the landscape forever and affected many species associated directly or indirectly with these magnificent trees; similar effects were observed with the emergence of Dutch elm disease in northern Europe in the middle of the last century. Currently, the extinction of many hundreds of amphibian species seems a real possibility with the

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ongoing spread of a new fungal disease, chytridiomycosis. Similar events have happened throughout history; the English nursery rhyme 'Ring a ring of roses' is thought by many to refer to outbreaks of plague (perhaps bubonic plague in Europe in the 1340s, or the Great Plague of London in 1665), the onset of which was signified by symptoms of a rash, followed by sneezing and rapid death. As the story goes, people tried in vain to protect themselves from infection by carrying various nostrums or posies of scented flowers.

Parasites are involved in many other processes within host populations and communities that can ultimately feed through to influence species coexistence and ecosystem function. For instance, parasites play a key role in honeybee colony loss, which is an emerging threat to biodiversity and agricultural production in Europe and America. Studies of parasites in Californian salt marshes reveal that parasites, which account for a substantial proportion of the biomass in these ecosystems (equivalent to a small herd of elephants per hectare), alter food web structure, dramatically enhancing the density of trophic links in the web. This can have implications for ecosystem health, as densely linked food webs are more robust to perturbation. Biological invasions are a major driver of biodiversity loss and, through their effects on the interactions of their hosts with other species, parasites can influence the outcome of biological invasions in a diversity of species ranging from plants to crustaceans to mammals. For instance, by reducing growth and survival of perennial bunchgrasses, barley yellow dwarf virus and its variants facilitate the replacement of native bunchgrasses by annual grasses in the prairies of the United States. By reducing the predation rates and survival of infected hosts, fungal and microsporidian parasites modify the interaction between native and invasive crayfish, facilitating the extirpation of the native white-clawed crayfish (*Austropotamobius pallipes*) in the freshwaters of England. By causing high mortality in red squirrels, squirrel pox virus alters competition between red and grey squirrels, facilitating the invasion of grey squirrels and the replacement of red squirrels in the United Kingdom.

Work in our laboratories on the amphipod *Gammarus* and its parasites over the last two decades reveals the range of effects parasites can have, from the level of parasite effects on individual host fitness; through parasite mediation of host–host interactions, including competition and predation which may determine which species can coexist; to altering the functional role of species within ecosystems and influencing the success of biological invasions (Box 1.1).

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Box 1.1 Parasitism in freshwater amphipod ecosystems

Amphipod crustacea are keystone species in freshwater ecosystems. Through processing nutrients and providing prey for larger invertebrates and vertebrates, they provide important ecosystem services. They process the primary basal energy resource (leafy detritus) through shredding, with strong impacts on community structure. They also predate smaller species in the food web, influencing macroinvertebrate diversity and species richness. Furthermore, they are key prey for commercial and recreational fish stocks and for wildfowl. Therefore, the impact of parasitism on amphipod population dynamics, and on their competitive and predatory interactions, could have profound ramifications for the diversity and structure of aquatic communities, as well as having economic costs.

In rivers and lakes in Northern Ireland, a suite of gammarid amphipods occur. The native *Gammarus duebeni celticus* is subject to invasions by at least three species of invader; *G. pulex*, *G. tigrinus* and *Crangonyx pseudogracilis*. The native and invasive species interact through competition for prey, as well as through intraguild predation (predation between species of the same guild), and these interactions are mediated by parasites (Fig. 1.1).

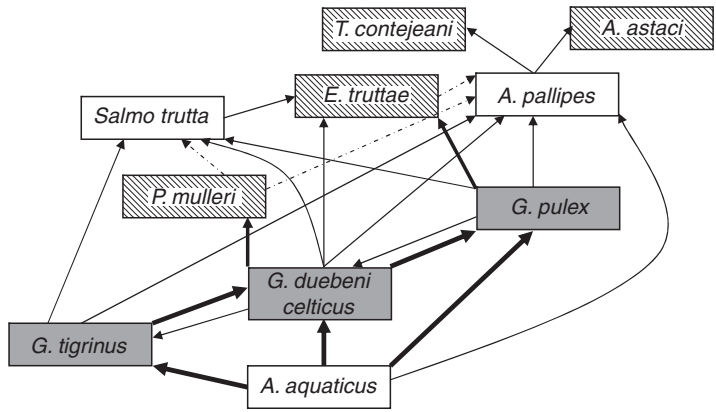


Fig. 1.1. Food web for the native and invasive *Gammarus* system studied in our laboratories. The direction of energy flow via consumption is shown by the arrows linking species (thickness indicates relative strength of interaction; dot-dashed lines depict predation of parasites by non-hosts when infected prey are eaten). Shaded boxes mark the three *Gammarus* species arranged in an intraguild predation hierarchy; stippled boxes are parasites. Brown trout (*Salmo trutta*) are definitive hosts for *Echinorhynchus truttae* and are therefore placed below this parasite in the web; other interactions as described in the text. The web is highly simplified; not all (host or parasite) species or interactions are shown.

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Box 1.1 (*continued*)

These gammarids prey upon smaller macroinvertebrates, and a key prey is the isopod *Asellus aquaticus*. Infection of *G. duebeni celticus* by the microsporidian *Pleistophora mulleri* reduces the predatory impact on the isopod, thus also modifying competition with other amphipods. In contrast, the acanthocephalan parasite *Echinorhynchus truttae* increases the predatory strength of *G. pulex*, likely facilitating the exclusion of the native *G. duebeni celticus* by the invader. *G. pulex* invasions have also been found to reduce macroinvertebrate diversity and richness, hence parasite modification of predatory behaviour may have ramifications throughout the community.

Intraguild predation (IGP) is also modified by parasites. *G. duebeni celticus* is a stronger IG predator than the invader *G. tigrinus*, yet coexistence occurs in several areas and may be facilitated by the microsporidian parasite *P. mulleri* (Dunn 2009). *P. mulleri* is specific to *G. duebeni celticus* and has no discernible effect on survival. However, the infection weakens IGP by *G. duebeni celticus* on *G. tigrinus*, enhancing coexistence in field manipulations. Similarly, the acanthocephalan parasite *Echinorhynchus truttae* weakens IGP by *G. pulex* on the less predatory *G. duebeni celticus*.

Moving up through the trophic levels, gammarids are preyed upon by fish and wildfowl, and their predation risk is influenced by parasites. Whilst parasites such as the microsporidian *P. mulleri* may increase vulnerability to predation (a by-product of the infection), the trophically transmitted acanthocephalan parasites *E. truttae* and *Polymorphus minutus* enhance transmission to their definitive host (fish and wildfowl, respectively) by manipulating the antipredator behaviour of their amphipod host. *E. truttae* is likely to have a greater impact on *G. pulex* as parasite prevalence is two-fold greater than in *G. duebeni celticus*. The outcome for the predator is mixed; whilst prey might be more available (trout productivity is higher in areas of *G. pulex* invasion), the chances of infection will also increase.

Gammarids are also predated by the white-clawed crayfish (*Austropotamobius pallipes*) and the impact of this predator is mediated by parasitism. Outbreaks of crayfish plague (*Aphanomyces astaci*) can cause crayfish mortality, whilst the microsporidian *Thelohania*

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Box 1.1 (*continued*)

contejeani reduces the predatory impact of the crayfish on its amphipod prey.

Hence parasites influence a variety of interspecific interactions, and may have potential effects throughout the community.

These complicated systems require a new approach that combines aspects of community ecology and parasitology, which we term *ecosystem parasitology*. Until recently, community ecology has historically ignored parasites, and parasitology has, in turn, largely ignored the community context in which infections spread. Attempts to meld these fields began in the 1980s with reviews and mathematical treaties by ecologists and epidemiologists Andrew Dobson, Pete Hudson, Peter Price, Robert Poulin, Roy Anderson and Robert May. Their papers provided an exciting route forward, but one that is only now gaining momentum. In order to integrate these disciplines, we need to combine some key concepts from community ecology and parasitology.

1.1 Concepts from epidemiology

Underlying much of modern epidemiology is the concept of R_0, the parasite's basic reproductive number, the number of secondary cases arising from each primary infection (Box 1.2). This measure predicts whether a parasite or pathogen can spread initially in a population of susceptible hosts; simply, if R_0 is greater than 1, the parasite can spread initially, if R_0 is less than 1, it cannot persist in the population. Another key concept arising from simple models of parasite spread within host populations is N_T, the threshold host population size for parasite establishment. Many (but not all) models of disease spread predict a threshold population size below which parasites and pathogens cannot become established.

However, parasites and other interactors such as competitors and predators may feed back on host population densities, and that is where the fun begins! How do parasites interact with other species, altering host population dynamics, and what are the consequences for coexistence of all the players, and for the structure and stability of communities as a whole? In order to examine these questions, we need to utilise developments from community ecology that allow epidemiological (host–parasite) models to be placed in a community context.

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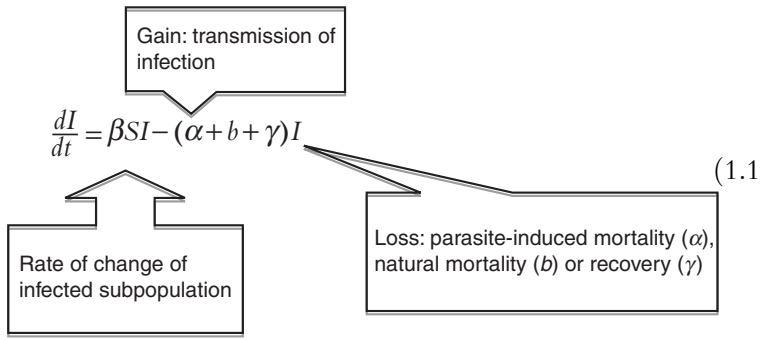
Box 1.2 R_0 and N_T

Basic reproductive number R_0. Whether a parasite spreads in a population depends on whether a single infection results in more than one infection in the following infection cycle. This is the concept behind the basic reproductive number R_0 (pronounced 'R nought'), defined as the average number of secondary cases produced by each primary case of infection in a completely susceptible population. For microparasites like human influenza viruses, this corresponds to the number of people infected by each infectious person. For macroparasites such as tapeworms, R_0 is the average number of tapeworms successfully reaching reproductive age produced by a single adult tapeworm.

Deriving R_0 depends on characteristics of both the parasite and host. The very simplest models of parasite–host dynamics start with the assumption that the host population is held to a constant density (which we shall call N) by factors other than disease. This is probably a reasonable assumption for many diseases in humans, for which epidemiological models were first developed. For a microparasite (such as flu or measles), we distinguish two host classes: those infected (I: for infected or infectious), and those yet to be infected (S: for susceptible). For a directly transmitted disease (i.e. one acquired directly from an infectious individual), susceptible individuals become infected at a rate dependent on contacts with infected individuals (assumed here to be proportional to population density I; this is known as density-dependent transmission; see Box 1.4 for an explanation), multiplied by the per-contact transmission efficiency of the disease (β). Once they become infected, individuals recover at a rate γ, entering the susceptible class again. Infected individuals die from the infection at rate α; infected and susceptible individuals also die from other causes ('natural' mortality) at rate b. This is one of the simplest epidemiological models we can have: all hosts are either in the S or I class and the parasite's only direct effect is to cause additional mortality to the infected class (for some of the more frequently met complications, see Box 1.4). From these definitions, we can write down the equation for the rate of change in density of the I class, in terms of the losses from and gains to this class:

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Box 1.2 (*continued*)



For the infection to spread when rare, we require $\mathrm{d}I/\mathrm{d}t > 0$; hence

$$\beta SI > (\alpha + b + \gamma)I \Rightarrow \frac{\beta S}{\alpha + b + \gamma} > 1. \tag{1.2}$$

This latter expression is closely related to R_0 (Anderson & May, 1981; 1991). When the disease is rare, almost all of the population are susceptible, so $S \cong N$; substituting $S = N$ into the above, we obtain:

$$R_0 = \frac{\beta N}{\alpha + b + \gamma}. \tag{1.3}$$

This makes intuitive sense: each infected individual produces new infections in a susceptible population at a rate βN, and each infection lasts for $1/(\alpha + b + \gamma)$ on average (because duration in a class is the reciprocal of the rate of loss from that class); hence R_0 represents the average number of new infections produced by each primary infection.

Threshold population size for parasite establishment N_T. In this simple model, R_0 depends positively on host population density. Since we require $R_0 > 1$ for the parasite to spread, there is a threshold population size N_T below which the parasite cannot spread. Solving $\mathrm{d}I/\mathrm{d}t = 0$ (or $R_0 = 1$), we find:

$$N_T = \frac{\alpha + b + \gamma}{\beta}. \tag{1.4}$$

This threshold means that parasites cannot invade populations smaller than N_T, but they can be maintained in larger populations.

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Box 1.2 (*continued*)

It also implies that a host population cannot be driven extinct purely as a result of disease; the infection will die out once the population is reduced below N_T. Extending this concept to multi-host communities, populations of some host species may meet their species-specific N_T and others may not; the former may then act as 'reservoirs' for infection of the latter, which act as 'sinks'. Interestingly, not all epidemiological models have a threshold for parasite establishment. In one common variant (that of frequency-dependent transmission; Box 1.4), the spread of infection is independent of host population size, so parasites are predicted to spread in and potentially threaten the existence of small populations.

Force of infection is a concept related to R_0 which is sometimes easier to estimate in real populations. The force of infection (often denoted λ) is the per capita rate at which susceptibles become infected; in other words, it is a measure of the risk of becoming infected. In our simple model,

$$\lambda = \beta I. \tag{1.5}$$

Force of infection is thus dependent on the frequency of infectious individuals in the population (and hence, all other things being equal, the chances of engaging in contact that might lead to infection). In large populations with stable age structures and constant infant death rates where the parasite has reached equilibrium, the force of infection can be estimated as the reciprocal of the average age at which hosts become infected. This approach has been used for human diseases such as measles. Age at infection measures how long an individual has avoided infection (duration in the S class); its reciprocal therefore measures rate of transition into the I class.

R_0 in community ecology. R_0 is equivalent to the net reproductive rate r of organisms. Few organisms actually reproduce successfully at average rate r because other factors (density dependence in birth or death rates, for instance) intervene. The same applies to R_0 for parasites: in the initial stages of an epidemic, secondary cases are produced at a rate R_0, but as the epidemic progresses, susceptible individuals become more scarce and control measures may be taken, reducing the average number of secondary cases each infection generates. Under these changing conditions, the *effective reproductive number* for the parasite (referred to as R, or R_{int} for R

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Box 1.2 (*continued*)

under intervention) also changes. If the parasite reaches equilibrium, each infection must generate exactly one new case on average ($R = 1$), otherwise the frequency of infection would change. R_0 is nevertheless a useful concept, like r. Parasites with a higher R_0 will increase more rapidly, all other things being equal. Parasites with an R_0 less than 1 will not be able to spread at all. Just as organisms are likely to have a different r in different habitats, parasites will probably have a different R_0 when infecting different host species. Hence, when we are dealing with systems involving multiple host species, parasite spread depends on a composite R_0 reflecting community composition and contacts.

1.2 Concepts from community ecology

Key to understanding how species interact within communities is a robust understanding of the types of interactions in which species engage, and how these affect the population densities of the interacting species (Box 1.3). Two species may interact directly (for example, via predation of one on the other) or indirectly via a third species (for instance, two prey species can interact indirectly via their shared natural enemy). Indirect interactions may be either density- or trait-mediated; that is, effects on the population of a focal species may result from a change in the population density of the species with which it interacts (a density-mediated effect), or from changes in the behaviour or morphology of that species (a trait-mediated effect). Parasites are prime candidates for causing trait-mediated indirect effects, because they often debilitate rather than immediately kill their hosts. However, parasites are also very good at generating density-mediated effects through their effects on host mortality, which feed through into population density.

This book is largely about the indirect effects of parasitism. Direct effects are covered in the extensive epidemiological treatments on one-host–one-parasite systems such as those by Anderson, May and Hassell. However, in order to place parasites in a community context, we need an understanding of the indirect effects of parasitism on other species. Analysis of community modules provides one approach to this. Community modules (Holt, 1997) are sets of three or more strongly interacting species. They provide a link between the artificial simplicity of

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Box 1.3 Ecological interactions

Direct and indirect interactions: interactions such as interference competition and predation are considered as direct interactions because individuals of one species interact directly with the other and have direct effects on each other; these may be reciprocally negative (competition:−−), positive (mutualism ++) or beneficial to one partner and detrimental to the other (predation, parasitism: +−). Arguably of equal importance are indirect effects, which occur when the impact of one species on another is mediated by the action of a third. Pure resource (exploitation) competition (a −− interaction between the consumers mediated by the resource species) is an example; another is apparent competition (see below).

Trait- and density-mediated interactions: indirect effects (the effect of species A on species B via the actions of species C) can be density- or trait-mediated. Interactions are density-mediated when species C causes changes in A's population density, which affect its interaction with B. Trait-mediated interactions occur when C causes a change in behaviour, physiology, morphology or life history in species A, which affects its interaction with species B. This concept of trait-mediated indirect effects (TMIEs) originates in the distinction between density versus behaviourally propagated effects (Abrams, 1992), and short- versus long-term indirect effects (Holt & Kotler, 1987; see below). Most examples of TMIEs come from the behavioural ecology of predator–prey relationships; for instance, the presence of predator C can increase refuge-seeking behaviour in species A, which reduces its foraging rate, so influencing competition with species B (see Werner & Peacor, 2003 for a review). As parasites rarely kill their hosts immediately, but frequently alter host behaviour and physiology, parasitism modules are potentially a rich source of TMIEs. For instance, in our native–invasive *Gammarus* system (Box 1.1), parasites can increase or decrease the predation activity of their hosts, and increase or decrease predator avoidance by their hosts.

Apparent competition occurs when two species that do not compete for resources have reciprocal negative effects (−−) on each other via the action of a shared natural enemy. Apparent competition was first described in terms of the density-mediated effects of predators. For instance, population growth of prey A provides resource for predator C, enabling an increase in the population density of C; the consequent increase in predation has a negative impact on prey B's

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Box 1.3 (*continued*)

population density (Holt, 1977). However, it can also be trait-mediated (Holt & Kotler, 1987), and it can also be mediated by parasites (Chapter 2). The knock-on effects of parasite-mediated apparent competition for ecological communities can be extensive: it underpins species coexistence/exclusion rules (Section 2.3); it can lead to unwanted side-effects in biological control (Section 2.3); and it can influence biological invasions (Section 6.5) and the dynamics of emerging infectious diseases (EIDs) (Section 8.2).

Short- and long-term effects: density- and trait-mediated indirect effects are likely to act on different timescales. Because density-mediated effects result from changes in the density of one species affecting the demographic responses of others, they should take longer to propagate to other species than the effects of direct interactions between species (Abrams, 1992). Behavioural and other trait-mediated effects occur on the same, or shorter, timescales as direct effects (Holt & Kotler, 1987). Because they occur rapidly, these short-term effects can, in theory, be very powerful in structuring communities, potentially overriding the effects of density-mediated processes (Bolker *et al.*, 2003). Short-term processes are also of interest because they produce a greater variety of interactions than those arising from density alone. For instance, in the apparent competition module (consisting of two prey and one shared enemy), all the combinations (+−,−+, ++, −−) of indirect interactions are theoretically possible (Holt & Kotler, 1987; Abrams & Matsuda, 1996). This great variety of interactions can have knock-on effects at other trophic levels, generating (often counter-intuitive) relationships between species (Abrams, 1992). Thus, understanding the potential of parasites to induce short-term trait-mediated effects could change how we view their role in community ecology (Hatcher *et al.*, 2006; Tompkins *et al.*, 2010).

single-population or two-species dynamics and the intractable complexity of entire communities. Modules provide a basis for analysing indirect interactions mediated by a third species, and how interactions such as predation and parasitism themselves interact.

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1.3 Parasites

Parasites are organisms that live on or in another host organism for all or part of their life cycle, deriving resources (usually nutrients) from the host. In contrast with predators, parasites do not immediately kill their host, although infection may increase the probability of host mortality, and some parasites eventually kill their host to facilitate their transmission to the next host. Hosts may also recover from parasites. Although ecologists have tended to ignore parasites, parasitism is in fact a major consumer strategy (Dobson *et al.*, 2008; Lafferty *et al.*, 2008a). For example, it has been estimated that 40% of known species are parasitic (Dobson *et al.*, 2008), and recent studies indicate that parasites are involved in a significant proportion of food web linkages (Lafferty *et al.*, 2006a; 2006b; Amundsen *et al.*, 2009).

Parasites can be divided into micro- and macroparasites, a distinction that not only reflects their size, but also the different modes of replication and transmission, which require different theoretical approaches (Box 1.4). Microparasites replicate directly within the host (often intracellularly), leading to increasing parasite burden within the host. Transmission occurs by direct contact between host individuals or via a vector (e.g. a mosquito). In addition to these horizontal transmission routes, some microparasites may be vertically transmitted from generation to generation of hosts. Microparasites may trigger an immune response in vertebrate hosts, leading to recovery and lifetime immunity to future infection.

In contrast, macroparasites grow within the host, but reproduction is followed by transmission to the new host, often through release of eggs and subsequent ingestion of egg or (free-living) larval parasites by the new host. Macroparasites may have direct life cycles or indirect life cycles involving two or more host species. As macroparasites have high antigenic diversity, host immunity may be delayed or may not occur.

Parasitoids are animals (usually insects) that are free-living as adults and that live on or in their host (usually an insect) during development, eventually killing the host. Hosts may recover by encapsulating and killing a parasitoid, but this does not result in future immunity from infection. Theoretical models sometimes treat parasitoids as predators; however, this fails to take into account the time delay before host death, during which time the host continues to interact with other community members as a competitor/predator/prey.

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Box 1.4 Modelling parasite transmission strategies

Deciding how to represent the details of parasite transmission is not a trivial problem. Firstly, the dynamics of transmission are different for micro- and macroparasites, and parasitoids. For parasites with direct transmission, successful transmission depends on the pattern of contact between susceptible and infectious hosts. Several different models for this have been proposed, the most common of which are density- and frequency-dependent transmission. Not all parasites have direct transmission; some have free-living stages, others utilise intermediate hosts, with or without transmission via free-living stages to one or more of the hosts. Vertical (inherited) transmission requires a different modelling approach to horizontal (contagious) transmission. Finally, host characteristics need to be considered; most importantly, aspects of immunity vary widely. Hosts may (or may not) recover from infection; if so, they may (or may not) become immune to reinfection; this acquired immunity may (or may not) be life-long. Capturing these variants requires different model structures, and the population dynamics and outcomes can be strongly dependent on these; below are some of the more frequently met distinctions.

Density-dependent transmission. Here, the number of new infections per unit time is proportional to the density of infectious hosts multiplied by that of susceptible hosts. Most models of directly transmitted microparasites, like the one in Box 1.2, assume transmission is density dependent, resulting in βSI new infections per unit time (where β is the transmission efficiency, denoting the proportion of S–I contacts that lead to successful transmission). Transmission of this type is also called 'mass action', because it assumes homogeneous mixing of the S and I classes as if they were molecules contained in a closed system. Although simplistic, it appears to be a reasonable approximation for the transmission of many pathogens (Anderson & May, 1981; McCallum *et al.*, 2001).

Frequency-dependent transmission. In this model, the number of new infections per unit time is proportional to the density of susceptible hosts multiplied by the frequency (proportion) that are infected, resulting in $\beta SI/N$ new infections per unit time. This type of transmission model is more appropriate when per capita contact rates are fairly constant, irrespective of population density, in which case the per capita chance of becoming infected is proportional to

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Box 1.4 (*continued*)

the prevalence of infection (I/N) in the population. This is the standard transmission model for STDs, where the number of sexual partners an individual has depends on social and mating systems rather than population density. Vector-transmitted diseases like malaria are also modelled this way, because infection risk for susceptible hosts depends on the proportion of vectors that have fed from an infected host. Basic theory for frequency-dependent transmission shows that, in contrast to density-dependent transmission (Box 1.2), there is no threshold host population size for parasite establishment (N_T). Consequently, such parasites can be maintained in small populations and could theoretically contribute to the extinction of that population. These different transmission modes have different consequences for community structure and stability (Chapter 7).

Recovery and immunity. These are not the same concept in epidemiology. Infected individuals may recover, re-entering the susceptible class, or in some cases they may become immune, in which case they are neither susceptible nor infected, and enter a new class (often called the 'removed' (R) class, because they are removed from the population available for infection). A further complication is that many diseases have a latent period after initial infection, in which individuals have contracted the infection but cannot transmit it to others. This requires a further distinction between individuals that have been exposed to infection (population class E) and those that have become infectious (I class). Compartment models are used to represent these structures; each class is represented by a compartment, and gain and loss equations like those in Box 1.2 represent the rates of transition between compartments. These models are often referred to with acronyms reflecting the ordering of compartments used (Fig. 1.2).

Macroparasites require a different approach to modelling than the SI compartment approach used for microparasites. Firstly, it is not generally appropriate to consider hosts (certainly, large vertebrate hosts) as either infected or uninfected; most hosts in a population may harbour a particular macroparasite (e.g. a helminth species), but they vary greatly in burden. Infections tend to be persistent; they are usually associated with increased morbidity rather than mortality, and the extent of deleterious effects are usually correlated with parasite burden. Macroparasites frequently

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Box 1.4 *(continued)*

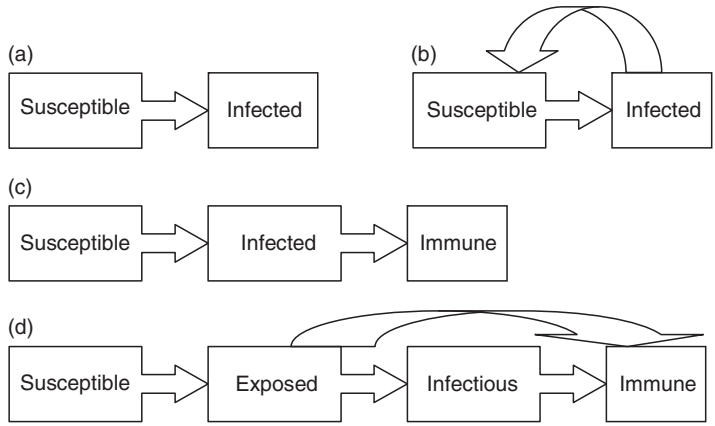


Fig. 1.2. Compartment models for microparasite infections. Each box represents a distinct stage (compartment); transitions between stages are shown by block arrows. Other loss and gain terms (birth, death, isolation or quarantine; not shown) change the fluxes into and out of compartments. Models shown are: (a) SI – there is no recovery; once infected, individuals remain so or die; (b) SIS – infected individuals can recover, re-entering the susceptible class; (c) SIR – infected individuals recover, becoming immune ('removed'); (d) SEIR – a latent (exposed) phase occurs after infection before hosts become infectious; exposed and infectious individuals can recover and acquire immunity. There are many further variations around these themes.

have an aggregated distribution among hosts: a few hosts are heavily infected (and suffer most of the fitness consequences), whereas most hosts have relatively few parasites. Macroparasite models take account of parasite distribution between hosts, and include fitness effects such as influence on fecundity, as well as mortality. Models track changes in host and parasite population densities, with parasite burden in the host described by a statistical distribution (the negative binomial distribution is often used; a clumping parameter can be set to describe the degree of parasite aggregation among hosts). Despite these differences, there are some broad similarities between the predicted effects of macroparasites and microparasites on communities (for instance, both can cause apparent or parasite-mediated competition (Chapter 2); both can cause trophic cascades through effects on consumer species (Chapter 7)).

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Box 1.4 (*continued*)

Complex life cycles. Many macroparasites have complex life cycles with obligate use of more than one host species. Transmission between host species may involve trophic transmission, where the parasite is transmitted to the definitive host (or another intermediate) via its consumption of an intermediate host; reproduction in the definitive host leads to the production of free-living stages. Models of such systems require a separate variable for each host species, and variables for each of the free-living parasite stages. Trophic transmission requires that suitable predation functions be incorporated into the model, including the possibility that parasitic infection influences the likelihood of being eaten. Aggregated parasite distribution and deleterious effects linked to burden need to be considered for most host species. Given these complications, it is perhaps not surprising that few models of parasites with complex life histories have been used to inform general questions about the roles of parasites in communities, as models tend to be tailored to specific systems.

Free-living stages. Many macro- and microparasites have free-living infective stages that survive outside the host. Some of these (baculovirus resting bodies or fungal spores, for instance) can remain viable in the environment for long periods. Free-living stages need to be represented as another class (compartment), and produce qualitatively different dynamical behaviour to the simple models of direct transmission. The time lags inherent in transmission from free-living stages can introduce cyclical dynamics. Also, for systems with multiple host species, the existence of a common pool of infective stages breaks down any community structure in transmission (the distinction between interspecific versus intraspecific transmission becomes redundant); model structure therefore bears more similarity to that for parasitoids.

Parasitoids are in some ways more like predators than microparasites, because they tend to kill their host; ovipositing females search actively for hosts and can potentially become satiated (their ova depleted), leading to similar expectations of a functional response between 'prey' density and predation/parasitism. However, they are also different from predators: they do not kill their prey instantly; they may share prey with other parasitoids or parasites; and they may be consumed during development in the host by predators, parasites or other parasitoids. Adults are effectively free-living stages (but not

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Box 1.4 (*continued*)

long-lived, so not intrinsically responsible for time lags); this common pool of infective stages tends to break down transmission structures as discussed above. One consequence of this is that models of the indirect effects on hosts of shared parasitoids behave more like those of predators than parasites (Chapter 2).

Finally, a note on *variables and parameters*. The distinctions we have drawn above mostly refer to variables: that is, the different classes of individuals (S, I, E, R) which each need to be represented separately as they vary over the course of the calculations. Increasing the number of variables can make models more realistic, but impedes their analysis. There can be no general methodologies for finding solutions (identifying and characterising equilibria, for instance) for systems with more than four variables. Parameters, on the other hand, are constants, which take values depending on the differences one wishes to express about the classes under consideration. For instance, infected individuals may experience a different predation rate to uninfected hosts; this would be reflected in different parameters for predation in the equations for each variable. Well-chosen parameters reflect quantities that one might be able to measure empirically. Increasing the number of parameters can also make models more realistic, and does not make them much harder to solve analytically, but it can make them more difficult to comprehend and interpret.

1.4 Aims of this book

In this book we examine the effects of parasitism in communities, starting with the structurally simpler modules that are the basic building blocks of a community. Throughout the book, our approach is to introduce a subject area and its theoretical underpinnings, in combination with empirical examples and applications. We do not go into the detailed theory of more complicated examples, but do present basic models to show the reader how mathematics has been used to develop predictions and structure ideas. We believe that researchers and practitioners in community ecology cannot be effective without at least a basic appreciation of the theory behind their science, and it is at this level that we have aimed the mathematical presentation. This means

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that in the earlier chapters, where the basic principles are developed, there are a few more equations than in the latter half of the book, where these principles are applied. We hope the models discussed in the earlier chapters will allow the interested reader to interpret the assumptions and predictions of more complicated models that are beyond the scope of this book. We begin in Chapter 2 with an examination of parasitism in competition modules, first examining the interaction between parasitism and intraspecific competition, allowing a brief reprise of the one-host–one-parasite epidemiological models on which much of the subsequent theory is based. We then move onto the case of interspecific competition and parasitism, in which we see that parasites and competitors can interact in a great variety of ways. From a community perspective, indirect interactions in these modules can be regarded as horizontal effects of parasitism, as they ramify between species at the same trophic level. In Chapter 3 we examine some vertical effects, looking at the interaction between parasitism and predation. Again, there are many structurally distinct ways in which parasitism can interact with predation, and the module approach helps to clarify these differences. In Chapter 4 we consider a more complicated module – that of parasitism combined with intraguild predation (IGP, the predation of potential competitors). IGP is increasingly recognised as an important component of community structure, and doubting readers will find ample evidence in the preceding chapters as we show how many parasitism–competition and parasitism–predation interactions may also be considered under the remit of IGP.

From an analysis of these basic building blocks, we move on to examine the community-wide ramifications of parasitism, where the conservation and biodiversity implications of parasitism become clearer. In Chapter 5 we examine the community impacts of parasites and pathogens of plants. This offers the reader a reprise of how the concepts developed in the preceding chapters apply to plant systems, and how effects at the module level propagate from these basal resources up throughout the community. In Chapter 6 we examine how parasites influence biological invasions via their effects on invading or native species. These examples illustrate how the effect of parasites at the level of competition and predation propagate through the community to determine invasion outcomes. We then go on, in Chapter 7, to examine how parasites influence general ecosystem processes and properties. This rapidly developing field (which we call ecosystem parasitology) is revealing a number of keystone and structural roles for parasitism,

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including effects on biodiversity, the structural stability of food webs and ecosystem health. Finally, Chapter 8 investigates emerging infectious diseases from a community ecology perspective; this is a very current problem with implications for human health and wildlife conservation, and best of all exemplifies how greater collaboration and integration between parasitologists and community ecologists is needed as we enter this third millennium.

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