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

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).

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).

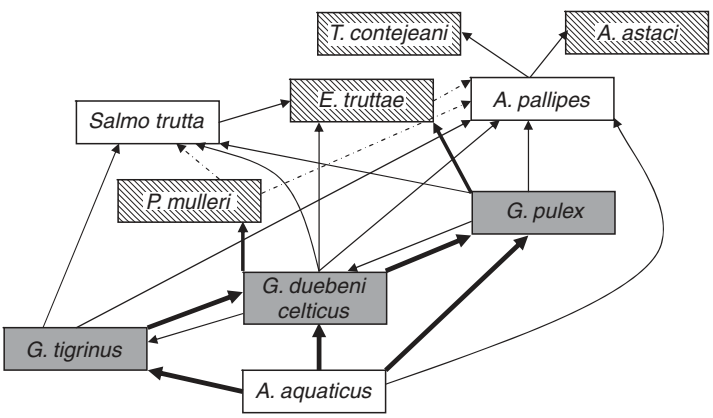


Fig. 1.1. Food web for the native and invasive *Gammarus* system studied in our laboratories. The direction of energy flow via consumption is shown by the arrows linking species (thickness indicates relative strength of interaction; dot-dashed lines depict predation of parasites by non-hosts when infected 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.

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*

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.

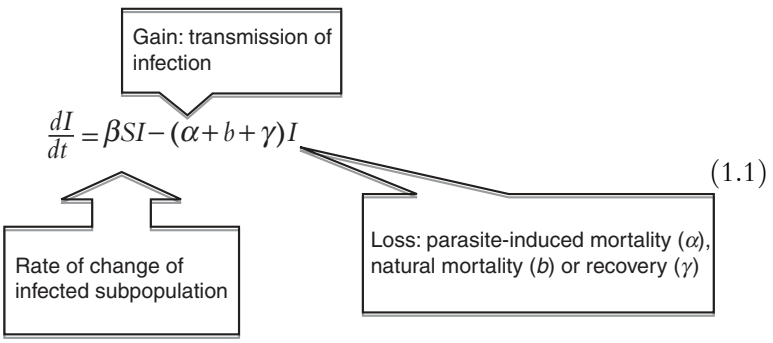
However, parasites and other interactors such as competitors and predators may feed back on host population densities, and that is where the fun begins! How do parasites interact with other species, altering host population dynamics, and what are the consequences for coexistence of all the players, and 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.

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:

Box 1.2 (*continued*)



(1.1)

For the infection to spread when rare, we require $dI/dt > 0$; hence

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

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:

$$R_0 = \frac{\beta N}{\alpha + b + \gamma}. \quad (1.3)$$

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 $dI/dt = 0$ (or $R_0 = 1$), we find:

$$N_T = \frac{\alpha + b + \gamma}{\beta}. \quad (1.4)$$

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

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

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

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