7 The Epidemiology of Parasitic Diseases in *Daphnia*

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7.1 Introduction

Parasites and pathogens may be directly or indirectly involved in the ecology and evolution of a broad range of phenomena: population dynamics and extinctions, maintenance of genetic diversity and sexual selection, to name just a few. Certainly parasites – here broadly defined to include viruses, bacteria, protozoa and helminths – possess features which make them very attractive as explanatory factors in the evolution and ecology of their host. These features include their typically narrow host range, the adverse effects parasites have on host fecundity and survival, and the density dependence of transmission (Hassell and May 1973; Anderson and May 1979, 1991; May and Anderson 1979). However, the bulk of available information stems from theoretical and laboratory studies, while studies in natural populations are scarce. For example, experimental approaches give clear support for density dependence of transmission (Blower and Roughgarden 1989; Ebert 1995; D'Amico et al. 1996; Knell et al. 1996), but there exists very little data showing density-dependent transmission in natural populations (Dobson and Hudson 1986; Scott and Dobson 1989). Similarly, although laboratory studies have demonstrated a clear effect of parasites on host density (Sait et al. 1994; Mangin et al. 1995), reports of parasite-mediated reduction of host density in the field are rare (Dobson and Hudson 1986; Scott and Dobson 1989). It is essential for our understanding of host–parasite interactions to compliment the results of laboratory work with data from natural populations.

7.1.1 Parasites in Zooplankton Populations

Traditionally, zooplankton ecology has focused on the effects of intra- and interspecific competition and on predation, and plankton dynamics have been claimed to be internally generated (McCauley and Murdoch 1987; McCauley 1993). Feedback loops between short-term changes in zooplankton density and predators have been dismissed as unlikely because such feedback is contingent upon the rate of change in the density of a particular predator species being a function of the prey’s current density (McCauley and Murdoch 1987; McCauley et al. 1988). Microparasites, however, do have exactly those features which can lead to feedback loops. They are often host specific and have a very
short generation time, which enables them to respond very quickly to changes in host density. Here, we investigate the role that microparasites play in the ecology of their Daphnia hosts. First, we summarize the results of field surveys investigating the abundance of parasites in natural Daphnia populations. We then outline some of the biological features of these microparasites, and finally we present a model of their epidemiology.

7.2 The Abundance of Daphnia Microparasites in Natural Populations

The first step in understanding the relevance of parasites to their natural host populations is to assess their abundance. To quantify microparasite prevalences (here defined as the proportion of adult hosts that are infected with micro-endoparasites) of Daphnia in natural populations field samples were collected from sites in England and Sweden. First, three English ponds were studied over a period of 1 year (about 10–12 Daphnia generations, 65 samples in total) and host density and fecundity were assessed, together with parasite prevalence, richness, diversity and host specificity (Stirnadel and Ebert 1997). Parasite prevalences were high throughout the year, averaging 84% in adult D. magna, 53% in D. pulex and 36% in D. longispina. In all three host species clutch size in parasitized females was significantly lower than in uninfected females (>20% reduction in D. magna, >25% reduction in D. pulex and >7% reduction in D. longispina). Whether reduced fecundity was a result of parasitism or whether the infections were a consequence of the hosts being weakened for some other reason is difficult to assess. Nonetheless, laboratory experiments confirmed the adverse effect of various parasites on host fecundity (e.g. Ebert 1994a,b, 1995; Mangin et al. 1995; Ebert and Mangin, in prep.). Only two of the 11 common micro-endoparasites found in these three ponds (17 species in total) showed no specificity within the three Daphnia host species; the other nine common parasites infected either only one or two of the three sympatric host species, or differed in their host specificity across the three ponds. The latter observation might indicate specialization of parasites to the currently, or formerly, predominant host community in a pond.

To assess the possibility of extrapolating our findings from these three strongly infected ponds to other populations, single samples were analysed from 43 Daphnia populations in southern England. Of all populations 91% suffered from endoparasite infections (mainly Microsporidia), with mean prevalences of 43% in D. magna, 69% in D. pulex and 43% in D. longispina (Brunner 1996; Brunner and Ebert, in prep.). A further, similar survey was conducted in rockpools along the Swedish east coast near Uppsala (Bengtsson and Ebert, in prep.). In 24 of 50 (48%) D. pulex populations and 9 of 25 (36%) D. longispina populations investigated micro-endoparasite infections were found. Across all ponds average microparasite prevalences were 15.5% for D.
pulex and 9.1% for D. longispina. Although average prevalences were lower in the Swedish rockpools than in English ponds, Swedish infections were primarily due to a single, extremely virulent microsporidium species. This undescribed microsporidium infected ovaries and fat cells of its hosts and reduced clutch size by 98%.

Given that many microparasite infections are only detectable after visible signs of infection have developed, our observations are probably conservative. The results of these field surveys clearly indicate that Daphnia parasites are abundant and are likely to play an important role in both the ecology and evolution of their hosts.

It should be mentioned here that vertebrate predators (fishes and newts) were absent in most of the populations surveyed and therefore we must be careful in extrapolating our results to habitats with high levels of vertebrate predation on Daphnia. There are two factors suggesting that levels of parasitism in Daphnia populations with strong vertebrate predation might be lower than in populations with less predation. Firstly, the likelihood of being infected increases with body size (Vidtmann 1993; Stirnadel 1994), which is probably a result of both higher filtration rates (and thus parasite spore uptake rates) and an accumulation effect with age. In ponds with high adult mortality – as is typical for ponds with fish and newt predators – parasite prevalence might therefore be reduced. Secondly, choice test in the laboratory show that some diseases make their hosts more conspicuous through a reduction in transparency and increase the likelihood of predation strongly (Lee 1994; Lee and Ebert, in prep.). Similarly, increased susceptibility to predation was reported for hosts carrying large loads of epibionts (Willey et al. 1990; Allen et al. 1993; Chiavelli et al. 1993; Threlkeld and Willey 1993).

Thus, parasite mortality in Daphnia populations with fish predators is high because of the lower life expectancy of their hosts, and additionally due to the preferences of visually hunting predators for conspicuous Daphnia. Therefore, Daphnia parasites might not be able to persist in the presence of strong fish predation (see also our mathematical model in Sect. 7.5.2). Consistent with this hypothesis is the observation that large Daphnia species are typically found in fishless ponds, and that most described Daphnia parasites are reported from large Daphnia species such as D. magna and D. pulex. Very few parasites have been reported from small Daphnia species, which co-occur with fish, such as D. cucullata or D. galeata (Green 1974).

### 7.3 The Biology of Transmission in Aquatic Systems

#### 7.3.1 Waterborne Transmission

The most important aspect of epidemiology is the mode of transmission. To our knowledge, the first description of a plankton parasite life cycle, including
a test of the mode of transmission, was Chatton’s (1925) description of the amoeba *Pansporella perplexa* in *D. pulex*. This parasite is transmitted between hosts via waterborne infective stages, which are ingested by the filter-feeding hosts. It was later shown for many other plankton parasites that waterborne transmission is a common route of dispersal. In laboratory transmission experiments we have confirmed the existence of waterborne spores for several *Daphnia* microparasites, including the bacteria White Bacterial Disease and *Pasteuria ramosa*, the amoeba *Pansporella perplexa*, the yeast *Metschnikowiella bicuspidata*, and the microsporidium *Glugoides intestinalis* (formerly *Pleistophora intestinalis*). Waterborne transmission is also common for many described epibionts of the Cladocera (Green 1974; Threlkeld et al. 1993). There are, however, also vertically transmitted (often transovarian) parasites of *Daphnia* (Mangin et al. 1995) as well as some which need a secondary host to complete development (Green 1974). Here, we concentrate on plankton parasites with direct, waterborne and horizontal transmission.

### 7.3.2 Survival of Transmission Stages Outside the Host

Planktonic populations typically undergo tremendous fluctuations in density, often over several orders of magnitude. Some plankton organisms might even temporarily disappear from their habitat and survive in the form of resting stages. Since these bottlenecks in host density pose a problem for horizontally transmitted parasites, Green (1974) suggested that plankton parasites should have persisting transmission stages which can endure phases of low host density. He suggested that pond sediments form spore banks for these infective stages, similar to the way that they harbour resting stages of many plankton organisms.

To test this hypothesis mud samples were collected from different ponds harbouring parasitized populations of *D. magna*. Subsamples of these sediments were placed in beakers and uninfected *D. magna* added. When the hosts were dissected after 24 days, infections with three different microparasites were found: the bacterium *P. ramosa* and the yeast *M. bicuspidata* were found in the haemolymph and the microsporidium *G. intestinalis* was found in the host gut (Ebert 1995). Using a mud sample that had been kept at 4 °C for 4 years, it was still possible to infect the *Daphnia* with *P. ramosa*. Similarly, mud samples containing spores of *G. intestinalis* remained infectious after storage at 4 °C for 3 months and in a different study for 5 months at 12 °C (D. Ebert unpubl.). These estimates of spore durability should be interpreted with caution until a systematic investigation to estimate spore durability appropriately (e.g. their half-lifetime) is completed. Nonetheless, the results clearly confirm Green’s (1974) hypothesis that pond sediments can serve as “parasite spore banks” and that parasites can survive periods of low host density in a “sit-and-wait” stage.
The uptake of spores from the sediments is a consequence of poor feeding conditions for the hosts. Some cladocerans change their behaviour when feeding conditions deteriorate and switch from normal filter feeding in the free water to a browsing behaviour on bottom sediments. This behaviour serves to stir up particles from the sediments, which are then ingested by filter feeding (Horton et al. 1979; Freyer 1991). It is important to note here that spore uptake from the pond sediments is a density-independent form of transmission.

In considering the survival of transmission stages, it would appear that parasites in aquatic systems face less problems than their terrestrial counterparts, as many common sources of spore mortality present in terrestrial parasites do not exist for waterborne transmission stages. For example, desiccation, one of the main threats to the survival of air- or soilborne spores, is irrelevant in the aquatic environment. In addition, water not only provides protection from UV radiation, but its high heat capacity also buffers the effect of rapid changes in temperature and prevents overheating. Since the protection of spores from adverse environmental effects can be considered to be costly, one might speculate that aquatic parasites should be able to shift the trade-off between offspring quantity and quality towards production of more offspring.

7.4 The Spread of Microparasites

After a parasite appears for the first time in a new host population it can only persist if on average each infection causes at least one secondary infection — that is, the basic reproductive rate of the parasite, $R_o$, must be larger than 1. There has been much discussion concerning which factors are responsible for the spread of a parasite in a plankton population.

7.4.1 Parasite Transmission Is Density Dependent

The standard assumption of epidemiological theory that parasite transmission is density-dependent has often been discussed with regard to plankton parasites (e.g. Canter and Lund 1951, 1953; Miracle 1977; Brambilla 1983). Certainly the most convincing data are that presented by Canter and Lund (1953), who observed strong fluctuations of the diatom *Fragilaria crotonensis* in an English lake. Whenever the density of this algae reached more than about 100 cells/ml, a fungal parasite (*Rhizophidium fragilariae*) spread rapidly and host density dropped by two orders of magnitude. For *Daphnia* no such example exists, although the reported data do not contradict density dependence (see Sect. 7.6). We have used an experimental approach to test for density dependence.

The microsporidian gut parasite *G. intestinalis* in *D. magna* proved to be an ideal system for experimental epidemiology. The life cycle of *G. intestinalis* is
Density-dependent transmission was also tested for *M. biscuspidata* and *P. ramosa* (D. Ebert, in prep.). These parasites are transmitted only after the infected host has died and the spores are released from the cadaver. The appropriate way to test for density dependence is therefore to produce spore suspensions and to test the infectivity of different spore concentrations. In both species of parasite, transmission probability was reduced when spore density was low, but transmissibility reached 100% when the spore density was high.

From these experiments we conclude that density dependence is indeed a real phenomenon in the spread of parasitic infections in *Daphnia* populations. However, the finding of a density-dependent mechanism of transmission does not reveal the practical importance of it for the epidemiology of natural *Daphnia*-parasite populations.
7.4.2 Parasite Transmission Can Be Limited by Low Temperatures

It has often been observed that plankton epidemics are predominantly found during the warm summer months. Ruttner-Kolisko (1977) proposed that transmission of a microsporidian parasite in rotifers is impaired at low temperatures. We tested this hypothesis with *G. intestinalis* in *D. magna*. It was found that parasite transmission was strongly impaired below 12°C. This is consistent with the observation that *G. intestinalis* decreased in late autumn in *D. magna* populations in south England (Stirnadel 1994). Poor transmissibility at temperatures below 25°C were reported for *P. ramosa*, parasitizing the cladoceran *Moina rectangularis* (Sayre et al. 1979). In contrast, *P. ramosa* in *D. magna* can be transmitted at 15, 20 and 25°C in the laboratory (Ebert et al. 1996). Thus it appears that the temperature criterion is species- and strain-dependent.

7.4.3 Host Stress Might Facilitate Parasite Spread

It has been claimed that *Daphnia* cultures kept under poor conditions are more susceptible to infections (Seymour et al. 1984; Stazi et al. 1994), and France and Graham (1985) have observed higher rates of microsporidiosis in crayfish in acidified lakes. We could not find any experimental evidence to support the stress hypothesis. Transmission of *G. intestinalis* appeared to be largely independent of the feeding conditions of *D. magna* and did not differ among age groups or host sexes (Ebert 1995). Other forms of stress and other parasites have not yet been evaluated.

7.5 Epidemiology of *Daphnia* Microparasites

The results discussed thus far indicate that the invasion, spread and persistence of a microparasite in *Daphnia* populations cannot be attributed to one single factor. Rather, the relevant factors might vary temporally, or act synergistically. In the following sections we develop a framework to encompass the different facets of microparasite epidemiology.

7.5.1 A Mechanism for Invasion, Spread and Decline of Parasites in Cladocerans

For parasites in the temperate zone the epidemiology of most microparasites follows a similar pattern (Green 1974; Ruttner-Kolisko 1977; Redfield and Vincent 1979; Brambilla 1983; Yan and Larsson 1988; Vidtmann 1993). Prevalences are typically low in winter and in early spring. After host plankton
densities peak in spring parasite prevalence increases. Prevalence fluctuates throughout the summer, decreases in the autumn, and in many cases parasites disappear in winter completely. Green (1974) suggested that the epidemics of some microparasites (e.g. the bacterium Spirobacillus cienkowski) start when a benthic feeding host acquires a parasite from the mud. Once the cycle is started other cladocerans that are partially benthic and partially free water foragers become infected and transmit the parasite to those cladocerans which live in the free water. The parasites disappear from the pond when the hosts go into diapause at the end of the season.

Ebert (1995) proposed a single species version of this model. Following diapause Daphnia hatch from their ephippia and re-colonize a pond. Under good feeding conditions in spring the population increases rapidly, until a severe food shortage leads to a population crash. Starving Daphnia change their behaviour and switch from filter feeding in the free water to a browsing behaviour on the bottom sediments, which serves to stir up food particles from the sediments (Horton et al. 1979; Freyer 1991). During this browsing, the animals pick up the long-lasting parasite spores from the bottom of the pond. Once the first hosts are infected, the disease can spread further through density-dependent horizontal transmission. The epidemic ends when either environmental conditions deteriorate (e.g. low temperature prevents transmission) or host density falls under the critical value necessary for parasite persistence.

7.5.2 A Mathematical Model for the Epidemiology of Plankton Parasites

We now develop an epidemiological model of an aquatic host-parasite system, which takes into account the features of Daphnia microparasites. In particular, we look into the combined effects of density-dependent host-to-host transmission (waterborne transmission), density-independent transmission from the spore bank in the pond (sediment-borne transmission) and the long durability of spores in the sediments. We model the case of the planktonic crustacean Daphnia and a horizontally transmitted microparasite, developed from a mathematical model of this system by Weisser et al. (in prep.). Our model is not intended for the analysis of long-term dynamics of host-parasite interactions. This allows us to make one further simplification. We assume that the number of spores in the spore bank effectively remains constant over a single season, not being significantly affected by spore uptake or by the sinking of spores into the sediment.

The dynamics of the microparasite infection of a single Daphnia consumer population is described by following the temporal variation in numbers of algae cells, A, uninfected, X, and infected, Y, Daphnia individuals, and of free-floating spores, Z. The model takes the following form:
\[ \frac{dA}{dt} = rA \left( 1 - \frac{A}{K} \right) - f(A)(X + Y) \]
\[ \frac{dX}{dt} = [\psi_x(A) - \mu]X + \psi_y(A)Y - \beta XZ - H(A)X \]
\[ \frac{dY}{dt} = \beta XZ + H(A)X - \mu Y \]
\[ \frac{dZ}{dt} = sY - mZ \]

(1-4)

The rationale underlying this formulation can be described as follows: Algae, \( A \), on their own are self-sustaining and their growth is described by a simple logistic equation with growth rate \( r \) and carrying capacity \( K \). The function \( f(A) \) determines how the \textit{Daphnia} feeding rate depends on the algae density \( A \) (Lampert 1987). The functions \( \psi_x(A) \) and \( \psi_y(A) \) describe the algal-dependent birth rates of unparasitized and parasitized individuals respectively. Since we exclude vertical transmission, both infected and uninfected females produce uninfected offspring. The functions \( f(A) \) and \( \psi_y(A) \) are monotonically increasing with saturation at high algal densities (Lampert 1987). The feeding function, \( f(A) \), can be caricatured by the form \( f(A) = dA/a_i \) for \( A < a_i \) and \( f(A) = d \) for \( A > a_i \), where \( a_i \) is the algal density above which consumer feeding is no longer limited by resource density (Fig. 7.2). For \( \psi_x(A) \) we assume that there is a minimum algal density \( a_0 \) below which reproduction is zero (e.g. Lampert 1987, Fig. 7.2). The caricatured form for the reproduction function \( \psi_x(A) \) is given by \( \psi_x(A) = 0 \) for \( 0 < A < a_i \), \( \psi_x(A) = b_i (A - a_i)/(a_i - a_i) \) for \( a_i < A < a_0 \), and \( \psi_x(A) = b_i \) for \( A > a_i \), where \( i = X \) for unparasitized individuals and \( i = Y \) for parasitized \textit{Daphnia} individuals. We assume that the effect of the parasite is to lower the birth rate of its host so that \( b_x > b_i \). Both infected and uninfected \textit{Daphnia} individuals die at rate \( \mu \).

To model the infection process we distinguish between density-dependent uptake of free-floating spores and density-independent transmission via spore uptake from the sediment. The rate at which uninfected consumers are infected by free-floating spores is proportional to the number of encounters between spores, \( Z \), and uninfected hosts, \( X \), with proportionality constant, \( \beta \). If algal density falls below \( a_0 \), \textit{Daphnia} individuals feed on the ground and take up spores at rate \( H(A) \) which increases for smaller \( A \) (Fig. 7.2). Infected \textit{Daphnia} release spores into the water at rate \( s \), and the spores die at a rate \( m \). All parameters are positive.

### 7.5.3 Analysis of the Model

In the absence of density-independent transmission (\( H = 0 \)), one can investigate the ability of a parasite to invade an uninfected \textit{Daphnia} population by
calculating the basic reproductive rate of the parasite, $R_0$. This gives the number of secondary infections a single infected individual will produce during its lifetime, if introduced into a population of uninfected hosts at equilibrium (Anderson and May 1979). In the present context, if an infected individual of the consumer species is introduced into an uninfected population at equilibrium, it will produce spores at rate $s$ until it dies. Since the life-
expectancy of a consumer is $1/\mu$, the lifetime production of spores of an infected individual can be calculated as $s/\mu$. The probability that a single spore infects a host during its lifetime depends on the life expectancy of a spore ($=1/m$), and on the equilibrium density of Daphnia individuals in the uninfected population, $X^{\text{uninfected}}$. Thus, the basic reproductive rate of the parasite is given by:

$$R_o = \frac{s\beta}{\mu m} X^{\text{uninfected}}$$  \hspace{1cm} (5)

An infection will only spread in the host population if $R_o > 1$. Equation (5) shows that a parasite can increase $R_o$ by increasing the transmission efficiency, $\beta$, the rate at which spores are released by infected host individuals, $s$, or by increasing spore longevity, $1/m$. $R_o$ also increases if the Daphnia equilibrium density of an uninfected host population, $X^{\text{uninfected}}$, is increased. Increasing Daphnia birth rate, $b$, increases $X^{\text{uninfected}}$. Also, $X^{\text{uninfected}}$ can be increased by increasing the algal growth rate, $r$, or increasing the carrying capacity of the resource, $K$ (Weisser et al., in prep.). In contrast, increased host mortality $\mu$ can reduce $R_o$. Thus, in a situation in which Daphnia is heavily predated upon, for example by fish, the basic reproductive rate of a parasite is reduced. This implies that some parasites might be able to persist in a Daphnia population only in the absence of predators. The Appendix gives the equilibrium values and stability properties of the model.

Suppose Daphnia feed on the sediment and take up spores from it whenever algal density falls below $a_g$. If $a_g > A^*$ (the algal equilibrium density calculated in the absence of density-independent transmission), then there will always be uptake of spores by Daphnia from the ground. Under these conditions the parasite will persist in the population even though the infection might not be able to spread via waterborne spores alone (i.e. $R_o < 1$). If, however, $a_g < A^*$, then Daphnia will not feed on the sediment once the population densities become close to the equilibrium densities, and initial parasite infection can only persist if $R_o > 1$. Figure 7.3 illustrates the effect of $a_g$.

It has been acknowledged that the combination of a saturating consumer functional response with a high nutrient supply can be very destabilizing in resource-consumer models (Rosenzweig 1971; DeAngelis 1992). This is also true in Daphnia populations (Murdoch and McCauley 1985; McCauley and Murdoch 1987). In our case, if the algal carrying capacity is close to the algal density, which is limiting host feeding and reproduction, $K < a_g$, the system returns to a locally stable equilibrium point. But if $K$ is high relative to $a_g$, then pronounced fluctuations can develop in the system. Unfortunately, the simple expression for $R_o$ given in Eq. (2) applies only if the pre-infection population has a constant size. Weisser et al. (in prep.) describe and provide computer simulations of the expected parasite invasion behaviour when the pre-infection population exhibits stable limit cycles.

Weisser et al. (in prep.) also describe the way in which the presence of the parasite is expected to modulate the nature, and occurrence, of any periodic
cycles, showing that it enhances the oscillatory properties of the system. Briefly, when $R_o$ is very high or the birth rate of infected individuals is very low positive equilibria are more likely to become unstable and bifurcate into stable periodic cycles (Fig. 7.4). Put rather simplistically, this is because both the increasing of virulence and/or the increasing of $R_o$ decrease total Daphnia density at equilibrium up to a point when it is no longer stable. The analytical predictions are summarized in the Appendix, in which we also derive an expression predicting that uptake of spores from the pond sediments will have
a dampening effect, serving to mitigate the influence of the free-floating spore-derived infection. This is illustrated in Fig. 7.5: because spores are regularly taken up from the sediment, infection is a more consistent feature of the system and less dependent upon the spore density in the water. The effect is strongest in that range of parameter values where the transition from local stability to stable periodic cycles occurs.

Buffering of the community fluctuations is not the only possible consequence of spore uptake from the ground. A more interesting, and potentially more important, function of the sediment spores is their possible role in re-initiating infection of Daphnia on a seasonal basis. Assume that the parasite is initially absent from a Daphnia population in early spring. With increasing temperature and sunshine the carrying capacity of algae increases. These increasing levels of algae cause the system to start to oscillate, and thus – because the fluctuations can lead to algal density regularly falling below \( a_s \) – there will be a repeated uptake of spores from the sediment; and the parasite thereby re-infests the host population (Fig. 7.6). This scenario provides a mechanism by which Daphnia populations can be newly infected each spring.

### 7.6 Discussion

Over the last three decades freshwater zooplankton population dynamics were believed to be shaped by the effects of inter- and intraspecific competition and
Fig. 7.5. The dampening influence of spore uptake from the pond sediments on the population dynamics of algae, *Daphnia* and parasite spores. $H(A) = d(a_3 - A)$. Upper graph: Oscillations, $a_3 = 10^6$. Lower graph: Monotonic damping, $a_3 = 5 \times 10^5$. Parameters for both graphs: $K = 3.5 \times 10^6$, $a_1 = 10^3$, $a_1 = 10^3$, $b_2 = 0.45$, $b_2 = 0.255$, $m = 25$, $m = 0.2$, $d = 1.5 \times 10^6$, $b = 10^3$, $s = 1000$, $d = 10^4$. (--) Algae, $A$; (....) uninfected *Daphnia*, $X$; (--.--.) infected *Daphnia*, $Y$; (--) free-floating spores, $Z$

predation. Subsequently, in the 1990s, parasites and epibionts were added to this list (e.g. Threlkeld et al. 1993; Ebert 1995), yet their relative importance in natural populations remains to be investigated. The results of our field surveys confirm that microparasites are indeed very abundant in natural *Daphnia* populations and it seems likely that they play a significant role in population dynamics, competition and life history of their hosts. Others have established the role of epibionts in natural populations (for a review see Threlkeld et al. 1993).
Density-dependent transmission is undoubtedly an important factor for the epidemiology of microparasites, including *Daphnia*. Nevertheless, the existence of additional density-independent transmission creates an interesting twist to the dynamics of parasite invasion. First, if a parasite infects hosts while they browse on the pond substrate it can persist in that host population even when each primary infection produces less than one secondary infection, that is even when $R_0 < 1$. Second, interactions with feeding conditions generate a complex scenario. Under poor feeding conditions, browsing on pond sediments continually causes new infections, but not under good feeding conditions; whereas good feeding conditions not only increase host numbers but also increase host-to-host transmission, and thereby the parasite’s reproductive rate. Stress due to poor feeding conditions seems not to increase host susceptibility, but the consequent behavioural alteration does, by exposing the hosts to the sporebank (Ebert 1995).

This complexity is reflected in the results of various field studies on microparasite epidemiology in zooplankton. Brambilla (1983) observed microsporidian epidemics in *D. pulex* in three successive years, with peak prevalences close to 100% in adult females. Parasites were generally present whenever the host density was above ten animals/l, but in one year the parasite suddenly disappeared in mid-summer despite high host densities. Vidtmann (1993) observed that the microsporidium *Larssonia daphniae* was present only when host density was high, and yet was often absent during periods of high host density. Similar results were reported by Yan and Larsson (1988).
Ruttner-Kolisko (1977) described a significant relationship between host density and prevalence, and even attributed a strong population decline of the rotifer *Conochilus unicornis* to a microsporidian epidemic: "... *Plistophora* finally terminates its host species". Stirnadel (1994) was not able to detect density-dependent interactions between any of three *Daphnia* species and their microparasites. Moreover, there was no detectable food effect on parasite prevalence, but for some parasites a seasonal pattern was detected. Green (1974) found seasonal patterns in parasite prevalence but did not relate these to changes in host density. Despite this paucity of published evidence in support of a critical role for density dependence in *Daphnia* epidemiology, most studies do note that there is a minimum host density for parasite persistence, although the behaviour at high densities has yet to be determined.

The mathematical model we present highlights some of the features peculiar to plankton parasites. In a previous model, Weisser et al. (in prep.) showed that host-specific microparasites are able to promote coexistence among *Daphnia* species which would, in the absence of parasites, competitively exclude one another from a community. It was also shown that the basic reproductive rate of the parasite, $R_0$, as used in classical epidemiology models inspired by terrestrial systems, must be adapted when the uninfected system is cyclic rather than steady. Here we describe why the existence of a density-independent transmission term – caused by uptake of sediment spores under conditions of poor *Daphnia* feeding – significantly modifies the conditions for parasite persistence. The basic reproductive rate [as defined in our model, Eq. (5)] can become redundant altogether as a means of predicting parasite persistence when there is such a large, non-depleting spore bank in the sediment. Instead it is both the feeding behaviour of *Daphnia* and the properties of the resource that determine parasite invasions. We describe the dynamics of a mechanism by which infection could occur on a seasonal basis: rising ambient temperatures or sunlight levels, say, in the spring lead to an increase in algal carrying capacity, and consequently to an increasing likelihood of oscillatory behaviour; if large oscillations develop the algal density can temporarily dip low enough to provoke bottom-feeding by the *Daphnia*, leading to uptake of spores from the pond sediment, and thus to the initiation of a new infective epidemic.

Although, our model was developed for planktonic organisms, our results concerning the role of a density-independent mode of infection could also have relevance to a number of soilborne diseases. Fleming and coworkers (1986) investigated the density-dependent transmission of a virus in different populations of the soil-dwelling pasture pest *Wiscana* sp. (Lepidoptera: Hepialidae). Evidence for density-dependent transmission was found only in young pastures, not in old pastures. The lack of density dependence in old pastures could be a result of transmission occurring mainly from a spore pool, which had been accumulated over several generations. In laboratory populations of a virus-insect system, Sait and coworkers (1994) attributed their failure to detect density dependence to the rapid accumulation and long
persistence of virus transmission stages within the cages. Viral contamination of the soil has been repeatedly claimed to be the source of various viral infections (e.g. granulosis virus and nuclear polyhedrosis virus infecting lepidopterans (Kellen and Hoffmann 1987; Young 1990; Woods et al. 1991). Similarly, the insect pathogen Bacillus thuringiensis is often found to accumulate in the soil (Dai et al. 1996). Thus it appears that durable transmission stages and their accumulation in pond sediments or soil might be a widespread phenomenon in natural host-parasite systems.

Daphnia and its micro-parasites are one of the few systems where both host and parasites have generation times short enough to allow experimental ecological studies to be carried out in the laboratory. The wide range of parasites available (bacteria, fungi, protozoa) allows for the testing and comparison of epidemiological, evolutionary and genetic models of infectious diseases. Our model shows that interactions of only a few ecological factors can produce complex epidemiological patterns. Only laboratory-based experimental tests can lead to the disentanglement of the factors acting simultaneously on interacting species. Nevertheless, field studies remain essential in determining the relative importance of these factors in the real world.

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The Epidemiology of Parasitic Diseases in Daphnia

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Appendix

Equilibria and Stability

The model described by Eqs. (1)–(4) has four possible equilibria:

• $E_{i}$: No species present. This state is always vulnerable to invasion by the algae.

• $E_{a}$: Only algae present ($A^{*} = K$). The Daphnia can invade provided that $y_{A}(K) > \mu + H(K)$. If $H(K) > 0$ then the Daphnia and its parasite invade simultaneously.

• $E_{ii}$: Algae and Daphnia, without parasite. The equilibrium levels of algae and Daphnia are given by the solutions of $y_{A}(A^{*}) = \mu = 0$ and $\mu = 1 - A^{*}/K) - f(A^{*})X^{*} = 0$. This state exists only if $y_{A}(K) > \mu + H(K)$ (i.e. if $Eii$ is unstable), and can go unstable in two ways: by bifurcating into stable oscillations (see below), or by being invaded by the parasite. If $H(A^{*}) = 0$ then the parasite invades when the “basic reproductive rate”, $R_{0} = sB^{*}/m_{u}$, is greater than unity (i.e. the eigenvalue $\lambda = sB^{*}/m_{u} - \mu$ is positive). If $H(A^{*}) > 0$ then the parasite always invades, and knowledge of $R_{0}$ is superfluous.

• $E_{ii}$: Algae, Daphnia and parasite. This equilibrium exists only if state $E_{ii}$ is unstable. It can go unstable by bifurcating into limit cycles, as described below. The equilibrium conditions are
\[
\begin{align*}
ra^*(1 - A^*/K) &= f(A^*)(X^* + Y^*) \\
Y^*(\mu - s\beta/m) &= X^* H(A^*) \\
X^*(\psi_x(A^*) - \mu) &= -Y^*(\psi_y(A^*) - \mu)
\end{align*}
\] (A1)

Limit Cycles

For this analysis we follow Weisser et al. (in prep.) and take the rate of change in the number of free-floating spores to be fast in comparison with the other variables, so that a pseudo-steady state hypothesis may be applied to the spore dynamics such that \(Z = Ys/m\). The general Jacobian for small perturbations about steady state \(E_p\) is

\[
\begin{pmatrix}
Q^* & -f(A^*) & -f(A^*) \\
-2X^*H'(A^*) & -\psi_y(A^*)Y'/X^* & \psi_y(A^*) - X^*s\beta/m\mu \\
X^*H'(A^*) & \mu Y'/X^* & -H(A^*)X'/Y^*
\end{pmatrix}
\] (A2)

where \(Q = r(1 - 2A/K) - f'(A)(X + Y)\), \(P = \psi_y'(A)X + \psi_y'(A)Y\), and dashes indicate differentiation with respect to \(A\). This leads to the cubic eigen-equation

\[
\lambda^3 + g_1\lambda^2 + g_2\lambda + g_3 = 0
\] (A3)

where

\[
\begin{align*}
g_1 &= -Q + HX/Y + \psi_y Y/X \\
g_2 &= -Q[HX/Y + \psi_y Y/X] + (\mu - \psi_y)Ys/m + fP \\
g_3 &= (\mu - \psi_y)\left[-QYs\beta/m - fH'(X + Y)\right] + fP[HX/Y + \psi_y Y/X]
\end{align*}
\]

The Routh-Hurwitz conditions tell us that this state is stable only if \(g_1 > 0, g_2 > 0\) and \(\xi = g_1 - g_2g_3 < 0\). If the first two of these conditions are satisfied then there is the possibility of a Hopf bifurcation occurring at \(\xi = 0\), going unstable as \(\xi\) increases. In the vicinity of such a bifurcation the oscillations will have period \(2\pi/\omega\), where \(\omega = g_1\). In Weisser et al. (in prep.) we prove analytically for a system the same as here, except with \(H(A) = 0\) for all \(A\), that the limit cycles associated with this bifurcation – both in the absence and presence of parasites – must be stable (i.e. the bifurcation is supercritical). Using basic continuity arguments (differentiability w.r.t. parameters: Arnold 1973), it is possible to show that the equivalent limit cycles in the case \(H(A) > 0\) must likewise be stable.

It is important to note that if the uninfected system exhibits stable oscillations then the use of \(R_0\) for prediction of parasite invasion requires it to be modified as an average over the whole cycle (including taking account of any periods when \(A\) drops low enough that \(H > 0\)), and this becomes less reliable the larger the magnitude of the oscillations.

Influence of Parasitism and of Spore Uptake

It is shown in Weisser et al. (in prep.) that the limit cycles are more likely to occur the larger \(K\), the carrying capacity of the algae. Furthermore, the presence of the parasite \((\beta > 0)\) is expected to enhance the magnitude and/or likelihood of oscillations. Here we indicate how the uptake of spores from the pond floor \((\beta > 0\) and \(H > 0\)) is likely to influence the situation. For understanding the effect of \(H(A)\) on the Hopf bifurcation it is useful to rewrite \(\xi\) in the form

\[
\xi = Q(g_1G_1 + fP) + G_3
\] (A4)
The Epidemiology of Parasitic Diseases in *Daphnia*

where

\[
G_1 = H \frac{X}{Y} + \psi_r \frac{Y}{X} \\
G_2 = \left[ (\mu - \psi_r) \left( f \frac{P Y}{X} - f H' (X + Y) - \frac{Y S}{m (H X / Y + \psi_r Y / X)} \right) \right]
\]

Thus the Hopf bifurcation occurs when

\[
Q = Q_0 = -G_1 / (g_s G_1 + f P)
\]  \hspace{1cm} (A5)

going unstable for increasing \(Q\). It is easily shown that in the absence of infection \(Q_0 = 0\). Although \(Q\) occurs on both the right-hand side and left-hand side of Eq. (A5), this form is useful because it allows insight into the role of \(H\): if we assume that changing \(\beta\) and/or \(H\) does not significantly alter the size of \(Q\) (in Weisser et al. (in prep.) it is shown that when using realistic parameter values the magnitude of \(Q\) is predominantly controlled by the relative sizes of \(K\) and \(a_s\), and only insignificantly by the indirect effects of \(\beta\) and \(H\)), then the effect of changing \(H\) can be understood simply by examination of \(Q_0\). Thus we see that increasing \(H\) decreases \(g_s\) but increases \(g_u\), so that \(Q_0\) becomes smaller. What this means is that whatever the intrinsic effect of the parasite upon the Hopf bifurcation, the effect of added benthic spore uptake is to act in opposition. We therefore predict, from this somewhat heuristic analysis, that uptake of spores from the pond bottom will have a dampening effect upon the oscillations.