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# Epidemiology of a *Daphnia* brood parasite and its implications on host life-history traits

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**Abstract** Parasites influence host life-history traits and therefore might crucially shape host populations in natural systems. In a series of laboratory experiments, we studied the impact of an oomycete brood parasite on its *Daphnia* (waterflea) host. We asked whether *Daphnia* dump the infected brood and subsequently are able to reproduce again as was occasionally observed in a preliminary study. No viable offspring developed from infected clutches, but 78% of the infected females produced healthy offspring after releasing the infected brood while molting. Neither those offsprings' development success nor their mothers' reproductive potential was affected by the brood parasite. However, infected *Daphnia* had a reduced life-span and suffered an increased susceptibility to another parasite, an unidentified bacterium. Additionally, we studied the prevalence of this brood parasite and the unidentified bacterium in a natural *Daphnia* assemblage in a pre-alpine lake, across changing demographic and environmental conditions. The brood parasite epidemic seemed to be host-density dependent. Our results show that the brood parasite's impact on the host population is enhanced when combined with the unidentified bacterium.

**Keywords** *Daphnia* · Increased susceptibility · Infection · Density dependence

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## Introduction

The relevance of host-parasite interactions in natural populations has become the center of attention in many recent studies. In agreement with theoretical models (Anderson 1978), parasites have been shown to regulate the density and, in extreme cases, even lead to extinction of natural host populations (Ebert et al. 2000a; Hudson et al. 1998). Parasites in ecosystems play a crucial role in food-web stability and energy-flow modification. Hence, parasites are thought to sustain the structure and biodiversity in ecosystems (Hudson et al. 2006; Marcogliese 2002).

However, as much as parasites have a strong effect on an ecosystem, they are affected by the ecosystem themselves, which results in diseases being spatially restricted. Physical habitat conditions have been shown to influence parasite epidemics: the outbreak of a parasitic yeast in *Daphnia dentifera* populations was most pronounced in steep-sided basins (Caceres et al. 2006). Nutrition status of aquatic habitats also influences host-parasite interactions: limb deformations in amphibians, caused by a parasitic flatworm, were more frequent in highly eutrophic habitats (Johnson and Chase 2004). Other factors that have dramatic effects on diseases are environmental stressors such as pollutants or habitat alteration (Lafferty and Kuris 1999). In addition to spatial variation in epidemic outbreaks, many diseases show strong seasonality and are absent during a considerable portion of the year. Hence,

fluctuations in prevalence seem to be coupled to seasonal fluctuations in environmental conditions (Altizer et al. 2006; Lass and Ebert 2006). Various environmental drivers including temperature (e.g., Ebert 1995; Mouritsen and Jensen 1997), host density (e.g., Bittner et al. 2002; Morand and Poulin 1998), host food quantity (e.g., Bittner et al. 2002; Ebert et al. 2000b) and selective predation on infected hosts (Pulkkinen and Ebert 2006; Willey and Willey 1993) were shown to cause and shape epidemics, in both laboratory experiments and field censuses. Therefore, to understand the emergence of diseases in natural systems, and thus to investigate the impact of parasites on infected hosts, it is important to know what conditions trigger or prevent an epidemic. Consequently, the combination of epidemiological studies with fitness assays can reveal the ecological significance of a disease.

Due to their clonal reproduction and short generation time, crustacean zooplankton of the genus *Daphnia* provide a useful model system to explore host-parasite interactions in ecological and evolutionary studies (Ebert 2005). Moreover, in natural assemblages *Daphnia* are commonly infected with a broad variety of parasites, with microparasites (Anderson and May 1979) of the bacteria, fungi, and protozoa being especially common (Green 1974; Ebert 2005). Since *Daphnia* play a central role in lake food webs (Lampert and Sommer 1999), parasites directly influencing the *Daphnia* assemblage might thereby indirectly shape energy flux in lake ecosystems.

The goals of this work were to investigate the impact of a common *Daphnia* pathogen (brood parasite) on its infected host as well as to examine possible environmental and demographic conditions causing outbreaks of brood-parasite epidemics in the *Daphnia* assemblage in Greifensee, Switzerland. We additionally surveyed the occurrence and impact of another parasite (unidentified bacterium) for possible interactive effects. Finally, based on the results, we discuss the ecological significance of the two parasites for natural *Daphnia* assemblages.

## Material and methods

### Host–parasite system

The study was performed with *Daphnia* collected from Greifensee, a medium-sized (8.5 $km^2$), highly eutrophic pre-alpine lake in Switzerland. Greifensee is inhabited by *Daphnia galeata*, *D. galeata* × *hyalina* hybrids, and their respective backcrosses (Keller and Spaak 2004). We studied the epidemiology of two parasites commonly infecting this *Daphnia* assemblage. The first parasite, visible as a brown spot in the brood pouch of its host, infects the *Daphnia* clutch. It belongs to the water molds (Saprolegniaceae) (Little and Ebert 1999; Stazi et al. 1994), which are members of the protists Oomycetes (Kamoun 2003). As this is still under discussion, we will refer to it as "brood parasite." The other parasite, a pathogenic bacterium (hereafter referred to as "unidentified bacterium"), infects the hemocoel of *Daphnia* and causes the host to appear opaque (Bittner 2001; Wolinska et al. 2004).

### Field survey

The course of the epidemics of both parasites was studied biweekly (spring, summer) or monthly (autumn, winter) from February 2003 till June 2005. Zooplankton collection, discrimination of different *Daphnia* age classes, screening for parasites, and analysis of the genetic composition of the *Daphnia* assemblage by allozyme electrophoresis were done as described in Wolinska et al. (2004). Prevalence was defined as the proportion of infected females in the random sample of 80–100 *Daphnia*. Temperature profiles were obtained from the municipal Agency for Waste, Water, Energy and Air Management (AWEL, http://www.labor.zh.ch/internet/bd/awel/gs/gq/de/see.html), Zurich, Switzerland.

### Experiments

Infected and uninfected adult *Daphnia* females used in the experiments were selected in each case from a single zooplankton sample collected during the brood-parasite spring epidemic in April/May 2005. Animals were selected from a single zooplankton sample to minimize the variability in parasite strains and to reduce host genetic variance. In all experiments, we used water from Greifensee as culture medium, which was filtered through a 0.45-µm sieve unless otherwise stated. After the first water exchange, on day 2 for the life-history experiment and on day 3 for the infection and susceptibility experiments, we replaced the medium every other day. Infected *Daphnia* donors (i.e., females with visible infection) were removed during the first water exchange. The water was stirred daily to maximize the encounter rate of spores in the infection and susceptibility experiments. During each water exchange, we retained a third of the "old" medium to maintain parasite spores in the system. *Daphnia* were fed every other day with chemostat-grown *Scenedesmus obliquus* at a concentration of 1 mg C $l^{-1}$. All experiments were performed at 12°C, the average temperature in the lake during the spring epidemic, and under a 16:8 h light:dark regime. We removed dead *Daphnia* and free-swimming neonates daily. In the infection and susceptibility experiments, we additionally removed animals that became infected to preserve a constant probability of infection for the remaining individuals.

*Infection experiment*

The goal of this experiment was to learn about the transmission mode of the brood parasite and the unidentified bacterium. Juveniles born within 48 h from uninfected females, sampled from the lake, were randomly assigned to the four different treatments. In each experimental unit, five juveniles were kept together in a 150-ml jar either with (1) five brood parasite-infected donor females, (2) five unidentified bacterium-infected donor females, (3) without donor females (to test for presence of spores in the lake water), and (4) without donor females in autoclaved lake water (control). Every treatment was replicated three times. Water was filtered prior to use only through a 95-μm mesh sieve to keep parasite spores in the medium. We measured the final prevalence of infected animals per experimental unit. The whole experiment was terminated on day 15, when embryos were observed in the brood pouches of uninfected *Daphnia*.

*Life-history experiment*

The influence of the brood parasite on host fitness was examined by comparing life-history traits of infected and uninfected *Daphnia*. As we were unable to establish a brood-parasite infection in the laboratory, 50 infected and 50 uninfected *Daphnia* females with eggs or embryos in the brood pouch were selected from the lake. *Daphnia* were measured under a dissecting microscope from the base of the spine to the top of the eye and randomly assigned to 100-ml jars. In addition to the daily examination for infected or dead *Daphnia*, we removed and counted neonates, and determined the body size of three randomly selected ones. As soon as a mother deposited a new clutch in her brood pouch, we measured her body size and determined the clutch size. Time between egg deposition and release was also recorded. When females produced a third laboratory clutch, they were removed from the experiment. The study was terminated on day 23. To determine the taxa and multilocus genotypes (MLG) of experimental *Daphnia*, females were frozen at −80°C for later allozyme electrophoresis. This was used to test that differences between brood parasite-infected and uninfected *Daphnia* were not confounded by genetic background.

*Susceptibility experiment*

The goal of this experiment was to compare the susceptibility of uninfected and brood parasite-infected females to the unidentified bacterium. All *Daphnia* were selected from a lake sample. Five *Daphnia*, all either infected with the brood parasite or uninfected but gravid, were put together with five unidentified bacterium-infected donor females in 200-ml jars. There were three replicates for each of the two treatments. The experiment was terminated on day 15, and the prevalence of the unidentified bacterium was measured in each experimental unit.

Data analysis

Density data were log-transformed, whereas prevalence data were arcsine-square root-transformed (Sokal and Rohlf 1995). All (M)ANOVAs, the Cox-regression analysis, and the correlation were performed with SPSS 14.0.

*Field survey*

Correlations between *Daphnia* density and brood-parasite prevalence in field samples were measured by Pearson's correlation coefficient.

*Infection experiment*

Data were analyzed with a one-way ANOVA to test for differences in prevalence of the unidentified bacterium among the three infection treatments.

*Life-history experiment*

A chi-square test was used to ascertain whether the number of *Daphnia* producing a first clutch differed significantly between infected and uninfected females. We tested the effect of the factors "infection status" and "initial body size" with a full-factorial, repeated-measures MANOVA where "increase in body size" and "time between two successive clutches" were set as variables for the mother's reproductive potential. We defined two levels corresponding to the two lab-released clutches ("clutching event"). Neonate development success was analyzed similarly, where the effect of "initial body size," "infection status," and "neonate-release event" was tested on the "number of released neonates," "clutching success" (the number of effectively released neonates divided by the number of initially deposited eggs), "average neonate size," and "development time" (days between egg deposition and release). In both MANOVAs, initial body size of the infected mother was included as covariate. Body sizes of adult females in the infected and uninfected treatment were compared with a repeated measures ANOVA where we tested the influence of four "clutch" levels and "infection." Survival was tested with a Cox regression using "infection status" and "initial body size" as covariates, inserted in the model with the forward stepwise (Wald) method. Differences in taxa composition between infected and uninfected *Daphnia* were analyzed with an $R \times C$ test (Sokal and Rohlf 1995), whereas differences in MLG composition were tested with the pairwise test of differentiation (Goudet 2002).

*Susceptibility experiment*

We used an exact one-tailed Mann–Whitney *U*-test to evaluate whether brood parasite-infected females were more prone to the unidentified bacterium than initially uninfected ones.

## Results

Field survey

The *Daphnia* density from February 2003 to June 2005 and the chronological sequence of the brood-parasite epidemic and the unidentified-bacterium epidemic are shown in Fig. 1. The brood parasite exhibited an annual infection pattern with a prevalence peak (~30%) in spring and another peak during autumn/winter (5% in 2003, 15% in 2004). The brood-parasite epidemic mainly appears in periods when the lake is thermally stratified and surface temperatures are roughly above 10°C. *Daphnia* density and brood-parasite prevalence peaked synchronously ($n = 59$, $r = 0.44$, $P < 0.01$). The unidentified bacterium was present in spring and summer and reached a maximum prevalence of ~10%.

Experiments

*Infection experiment*

The brood-parasite infection appeared in none of the treatments, the unidentified bacterium emerged in all infection treatments between day 3 and 7, and no infections appeared in the control (Fig. 2). There were no differences in the prevalence of unidentified bacterium among the treatments ($F_{3,8} = 2.34$; $P = 0.15$).

*Life-history experiment*

Due to different physical conditions of the experimental animals, not all could be used in each analysis, which resulted in different degrees of freedom in the reported statistical tests. All brood parasite-infected *Daphnia* were capable of dumping the infected brood but were less successful than originally uninfected ones in producing at least one viable clutch thereafter ($\chi^2 = 9.69$, $df = 1$, $P < 0.01$), with 78% of the individuals producing a clutch in the infected group, compared to 100% in the uninfected group. After dumping the infected clutch, none of the brood parasite-infected females became infected with this parasite again. Twelve brood parasite-infected *Daphnia* and only three initially uninfected animals became infected with the unidentified bacterium; the unidentified-bacterium infection occurred between day 3 and 15 ($\chi^2 = 5.35$, $df = 1$, $P < 0.05$). These animals with secondary infections were not included in subsequent analyses. There was neither an effect of the brood-parasite infection on the mother's reproductive potential ($F_{2,35} = 1.67$, $P = 0.20$) nor on the development success of neonates ($F_{4,45} = 0.83$, $P = 0.52$). Infected and uninfected females did not differ in body size ($F_{3,35} = 2.59$, $P = 0.07$) at any time. The Cox-regression analysis showed that "infection status" and "initial size" are plausible factors, influencing *Daphnia* survival (Table 1; Fig. 3). There were neither significant differences in taxa composition ($G = 5.83$, $df = 5$, NS) nor in MLG composition ($n_{inf} = 29$, $n_{un} = 38$, NS) between the infected and uninfected groups.

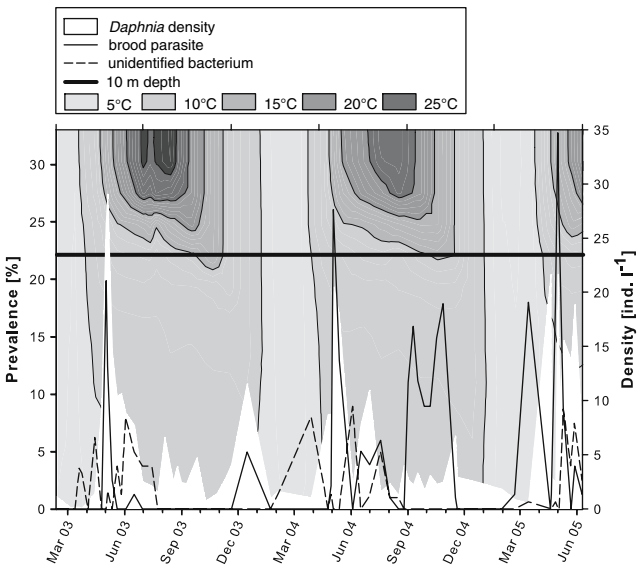


**Fig. 1** *Daphnia* density in Greifensee from February 2003 to June 2005 (*white area*), prevalence of the brood parasite (*solid line*) and the unidentified bacterium (*dotted line*), and the temperature profile (*background plane*). The *black horizontal line* indicates the 10 m depth

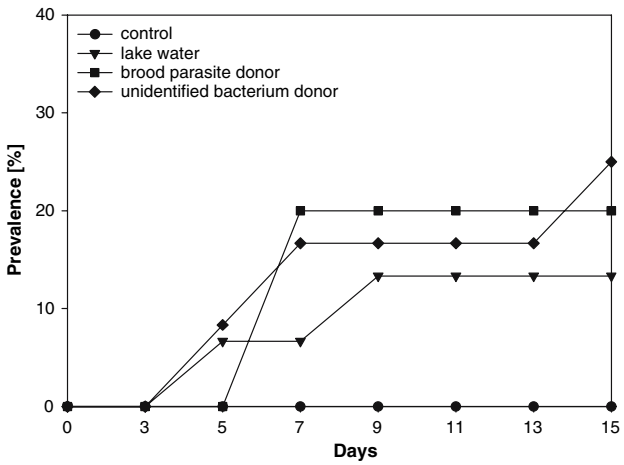


**Fig. 2** Mean prevalence of the unidentified bacterium in each of the four different treatments (infection experiment)

**Table 1** Results of a Cox regression, testing the influence of initial body size and brood-parasite infection on the survival of *Daphnia* (life-history experiment)

| | | B | SE | Wald | *df* | *P* | Exp (B) |
|---|---|---|---|---|---|---|---|
| Step 1 | Initial body size | 0.377 | 0.175 | 4.672 | 1 | 0.031 | 1.458 |
| Step 2 | Infection status | −0.722 | 0.355 | 4.138 | 1 | 0.042 | 0.486 |
| | Initial body size | 0.477 | 0.183 | 6.744 | 1 | 0.009 | 1.610 |

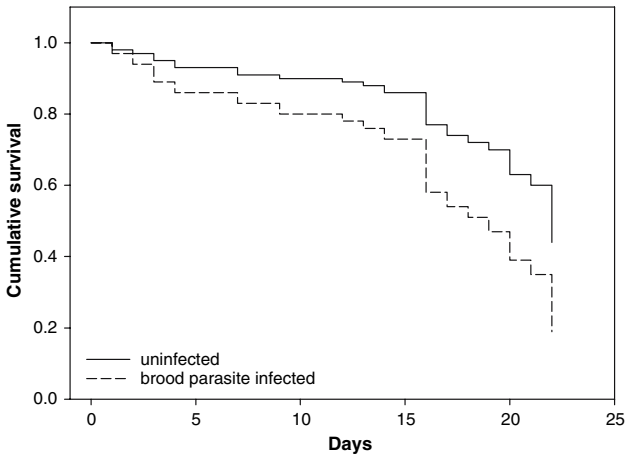


**Fig. 3** Cumulative survival of brood parasite-infected and uninfected *Daphnia* over time. Each step indicates the probability that an individual survives until the next step (life-history experiment)

*Susceptibility experiment*

Females initially infected with the brood parasite were more prone to the unidentified bacterium infection (57%) than initially uninfected (13%) females ($U = 0$, $P = 0.05$).

## Discussion

We observed that the brood-parasite infection was restricted to *Daphnia* eggs and embryos, and that all infected females were capable of dumping infected clutches while molting. Previously infected females that got rid of the parasite were seemingly immune under laboratory conditions. Comparable results were obtained in a study of a copepod brood parasite, where infected females shed the infected clutches and produced viable ones thereafter (Burns 1985). Infected copepods had a higher mortality as did infected *Daphnia* in our experiment. This observed increased mortality (Fig. 3) may reflect the costs of shedding the parasitized brood (Sheldon and Verhulst 1996) or might be due to toxins produced by the parasite. In both studies, experimental animals were collected from the field, and since it takes time to develop visible disease symptoms, one could argue that the higher mortality of infected females was due to an age effect rather than a parasite effect. In our study, however, we counterbalanced potential age differences between brood parasite-infected and uninfected daphnids by selecting only gravid females and by showing that the two groups did not differ in body size. Genetic background should have no confounding effect, because there were neither taxa nor MLG differences between both groups.

Compared to other *Daphnia* pathogens that completely castrate their hosts and potentially even eradicate entire populations (e.g., white bacterial disease, Ebert et al. 2000a), the brood parasite is less harmful for its host: 78% of the initially infected females were able to recover and reproduce again. However, there might be indirect costs of a brood-parasite infection. The parasitized clutch appears as a brownish spot in the brood pouch, making otherwise transparent *Daphnia* easier to detect by predators that hunt visually. Selective predation by fish on infected hosts was previously shown for other *Daphnia*-parasite systems (Duffy et al. 2005; Willey and Willey 1993). Moreover, we observed that fitness costs due to a brood-parasite infection were drastically increased, because *Daphnia* infected with the brood parasite were more prone to an infection by the unidentified bacterium, as was shown in the life-history and susceptibility experiments. Whether the increased susceptibility was a direct effect of the brood-parasite infection or an indirect effect through a general loss of vigor of infected females is not evident from our data. The close association between the two parasites is also apparent from the field survey; the unidentified-bacterium epidemic and the brood-parasite epidemic coincide in spring in Greifensee (Fig. 1). Moreover, in a recent survey of *Daphnia* parasites across Swiss and northern Italian lakes, in 11 of 15 lakes with the brood parasite, the unidentified bacterium was also present (Wolinska et al. 2007). The frequent coexistence and synergistic effect of the two parasites might have important consequences for natural *Daphnia* assemblages. The horizontally transmitted unidentified bacterium seems to be highly virulent: infected *Daphnia* did not reproduce again and died on average 1 day after becoming visibly infected in the susceptibility experiment (data not shown). Moreover, the phenomenon of synergistic effects imposed by different parasites might be of general importance because co-occurrence of different parasites is quite common in natural *Daphnia* systems (Bittner 2001; Stirnadel and Ebert 1997; Wolinska et al. 2007).

The brood parasite seems to require suitable host conditions and appropriate environmental prerequisites to successfully initiate an epidemic. From the correlation it is apparent that the *Daphnia* population needs to be dense to initiate an epidemic. Several epidemiological models predict a positive relationship between host population density

and the abundance of directly transmitted parasites (Anderson 1978; Anderson and May 1978). When host density is low, the contact rates between infected and uninfected individuals are limited and the disease is expected to have a lower impact. In many natural parasite communities, it has been shown that host population density and average parasite abundance are strongly positively correlated (e.g., Altizer et al. 2003; Arneberg et al. 1998; Morand et al. 2000). Indeed, it has been shown that a brood-parasite epidemic is more likely to break out in eutrophic than oligotrophic lakes (Wolinska et al. 2007), which corroborates our findings, because lake trophy is a good predictor of *Daphnia* density (e.g., Keller et al. 2002).

Hall et al. (2006) proposed that if selective predation pressure on infected individuals is strong enough, predators can prevent or halt an epidemic. The brood-parasite epidemic falls exactly in the time period when fish predation is expected to be highest in the lake. When there are low oxygen levels at deeper depths, which coincides with the thermal stratification of the lake (Lampert and Sommer 1999), neither fish nor *Daphnia* can migrate down, and they both aggregate in the upper parts of the water column (Lampert 1993; Lampert and Sommer 1999). Hence, fish predation pressure might work as a controlling factor of the brood-parasite epidemic, which therefore persists only for a short time. Another reason for the abrupt halt of the brood-parasite epidemic might be a crash of the *Daphnia* assemblage in eutrophic lakes, caused by a sudden food shortage commonly occurring in spring (Lampert and Sommer 1999). Prior to a crash, there are high food conditions in the lake leading to an enormous increase in *Daphnia* density (Fig. 1). Hence, high eutrophic conditions might be two-edged and might explain seasonal fluctuations in brood-parasite prevalence: on one hand, they allow the outbreak of an epidemic due to an increase in the host density, but on the other hand, they could also be responsible for an abrupt ending due to a subsequent crash in the host density (Fig. 1).

In conclusion, our study showed that conditions influencing host-parasite systems may comprise complex interactions between different parasites and environmental factors, effects that are not evident when considered in isolation. Specifically, the impact of one parasite might be considerably magnified by a weakened resistance towards other parasites present in the system. Although the brood parasite is not very virulent alone, the level of fitness reduction due to both infections, brood parasite and unidentified bacterium, might be severe enough to considerably influence the natural *Daphnia* assemblage. Furthermore, this effect may be reinforced by stochastic processes during the crash in *Daphnia* density and other components of the lake food web, such as predator and food abundance. Hence, even in the absence of genotype-specific infections, the brood-parasite epidemic has an impact on the entire *Daphnia* assemblage and consequently on the entire lake community.

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