Limnol. Oceanogr., 47(1), 2002, 300–305

Ecological interactions of the microparasite *Caullerya mesnili* and its host *Daphnia galeata*

Abstract—It has been repeatedly suggested that parasites play an important role in the ecology and evolution of *Daphnia* populations; however, little is known about *Daphnia*–parasite interactions in lakes with vertebrate predation on *Daphnia.* Between September 1997 and April 1998, an epidemic of the protist parasite *Caullerya mesnili* in *Daphnia galeata* and *Daphnia hyalina* occurred in Lake Constance (Germany), infecting up to 50% of all individuals. Using laboratory experiments, we investigated the epidemiological interactions between this parasite and its host *D. galeata* at the individual and the population level. *C. mesnili* was found to be transmitted directly and horizontally through waterborne infection stages. Transmission of the parasite was dependent on the host density, and all life stages of female *D. galeata* were susceptible to infection. In a life table experiment at low and high food levels, the life expectancy and fecundity of infected *D. galeata* were dramatically reduced at both food levels as compared to the uninfected controls. Additionally, we found a significant interaction between infection and food level, indicating a stronger parasitic effect in well-fed hosts. To test the effects of the parasite at the population level, we compared the size of *D. galeata* populations infected with *C. mesnili* with the size of parasite-free microcosm populations. The population size of infected *D. galeata* was significantly lower than that of uninfected populations after 4 weeks. In all four infected replicate populations, the parasite drove the host population to extinction.

Food limitation and predation are usually regarded as the most important factors regulating the population dynamics of zooplankton in freshwater ecosystems (e.g., Sommer 1989; Gliwicz and Pijanowska 1989). However, a number of reports have stressed the possible role of parasites in influencing and even regulating plankton densities in natural freshwater systems (phytoplankton, Canter and Lund 1951; Bruning et al. 1992; zooplankton, Green 1974; Allen et al. 1993). Parasites have a number of properties that make them very suitable as regulators of their host populations: they reduce fecundity and/or survival of their hosts, their transmission rates increase with host density, and they are often very specific to their host species (Anderson and May 1978, 1979).

Since the seminal review on parasites in Cladocera by Green (1974), a number of studies have reported on the prevalence of endoparasites and epibionts in natural *Daphnia* populations (Brambilla 1983; Threlkeld et al. 1993; Vidtman 1993; Ebert et al. 1997; Stirnadel and Ebert 1997; Bengtsson and Ebert 1998). Several laboratory studies have shown that infection by microparasites reduces fecundity and increases mortality of *Daphnia* hosts (Green 1974; Brambilla 1983; Ebert 1995; Mangin et al. 1995). Additionally, microparasites of *Daphnia* are believed to influence population dynamics, competition, and life history of their hosts (Threlkeld et al. 1993; Ebert et al. 1997). Several workers have predicted that parasite-induced reduction in individual host fecundity and survival may reduce host population density and increase the chance of local extinction (Anderson and May 1981; McCallum and Dobson 1995; Ebert et al. in press). Additionally, the regulating potential of a parasite is predicted to increase if transmission increases as a function of host density and if parasites have relatively short latent periods and produce large numbers of infective stages (Anderson and May 1981). Results of experimental studies with *Daphnia magna* are consistent with these predictions (Ebert and Mangin 1995; Ebert et al. in press).

Few studies have investigated the distribution and abundance of endoparasites within natural zooplankton populations and the subsequent ecological consequences of parasitism in *Daphnia* (Green 1974; Brambilla 1983; Stirnadel and Ebert 1997; Bengtsson and Ebert 1998). The overall picture suggests that parasitism in *Daphnia* populations is common. Most of these studies, however, have concentrated on small bodies of water without vertebrate predation on the *Daphnia* host. Ebert et al. (1997) suggested that levels of parasitism in *Daphnia* populations with vertebrate predation might be lower than in populations with less predation. The reason for this is a higher filtration rate by large daphnids of ponds (and thus uptake rate of infection stages) and an accumulation with age (Ebert et al. 1997). Furthermore, parasite infections have been observed to make *Daphnia* more conspicuous and thus make them a preferred prey for visually hunting predators (Lee 1994).

But during a field survey in Lake Constance, a large prealpine lake in southwest Germany, we discovered (among other parasites) a frequent occurrence of the protozoan endoparasite *Caullerya mesnili* (Chatton 1907) in *Daphnia* spp. in autumn and winter 1997/1998. On average 26.9% of adult *Daphnia galeata* and 13.5% of adult *Daphnia hyalina* were infected with this gut parasite, with maximum values of 50% and 32% respectively (K. Bittner unpubl. data).

Here we present a study aimed at understanding the life cycle and epidemiological interactions of *C. mesnili* with one of its hosts, *D. galeata,* at the individual and population level.

Host–parasite system—Monoclonal offspring of a single female *D. galeata* (Cladocera, Crustacea) isolated in July 1997 from Lake Constance were used for experiments. *C. mesnili* is a sporozoan that infests the gut epithelium of *Daphnia. C. mesnili* is classified as a Haplosporidium (Chatton 1907; Green 1974), but this classification is in discussion (R. Larsson pers. comm.). Although it has been regularly reported in field studies (Green 1974; Stirnadel and Ebert 1997; Bengtsson and Ebert 1998; Little and Ebert 1999), experimental investigations on its epidemiology and the impact on its host are lacking. In February 1998, we isolated the parasite from *Daphnia* from Lake Constance. *C. mesnili* was easily identified by its large spore clusters (up to 100

μm in diameter) consisting of 8–20 oval-shaped spores 10 $\times$ 8 μm in diameter. Parasite infestation was quantified by counting these clusters in the dissected gut under a stereomicroscope (>100$\times$ magnification). We established and maintained mass cultures of monoclonal *D. galeata* parasitized with *C. mesnili* under standard culture conditions in 1.5-liter beakers (20 $\pm$ 1°C, 24 h dim light, daily food ration of 1 mg C L^{-1} *Scenedesmus obliquus*) for several host generations. As medium we used 0.45-μm filtered water from Lake Constance. The parasite cultures were maintained by adding uninfected newborn *D. galeata* from uninfected stock cultures at approximately 3-week intervals. Experimental standard conditions were the same as culture conditions unless stated otherwise.

Transmission mode—For the epidemiology of an infectious disease, it is important to understand whether the parasite is transmitted horizontally (through waterborne infection stages) or vertically (transmission from mother to offspring). We conducted an experiment to test for horizontal transmission. The following possibilities were addressed: (1) infection from live infected hosts (donors) to uninfected animals (recipients), (2) infection from dead infected donors to uninfected recipients, and (3) infection from water in which infected animals had been kept to uninfected recipients. All treatments and controls consisted of groups of 20 neonate *D. galeata* recipients. We incubated 20 newborn recipients for 2 d with either six live infected non-egg–bearing donor females or the remains of six donors that were killed and homogenized or in 250-μm filtered medium from infected stock cultures. Treatments were done in triplicate; the appropriate controls, with uninfected animals from stock cultures instead of infected donors, were done in duplicate. Beginning with day 3, the *D. galeata* recipients from all treatments and controls were checked every other day for the occurrence of spore clusters in their gut cells. In all treatment cultures spore clusters became visible by day 11 at the latest. Thus, living infected hosts, decaying host remains, and contaminated water all were infectious. We observed no spores in any animals in control cultures.

We tested for vertical transmission with 12 infected juvenile females kept singly until they produced the first clutch of eggs. When the eggs had reached the eye-spot stage, we isolated two embryos from each brood pouch and kept them singly in beakers containing 100 ml medium. After 12 d, all 24 offspring were dissected and checked for infection. In no case did we observe the transfer of *C. mesnili* from infected mothers to their offspring, thus making vertical transmission unlikely.

We conclude that *C. mesnili* is horizontally transmitted, which is most likely caused by waterborne infection stages that leave the donor animal with the feces. Infection follows the ingestion of transmission stages by the filter-feeding host.

Density-dependent transmission—To test for host density effects on transmission, single 2-d old juveniles were placed together with single infected (uninfected in the controls) non-egg–bearing adult females in beakers containing 3, 10, 30, 100, 300, or 1000 ml medium. Ten replicates were used per beaker size and there were two uninfected controls per beaker size. After 2 d, during which no food was given, the recipient animals were isolated by placing them separately in 100 ml medium. Daily, we changed medium and added standard food ration. Animals were dissected and examined for the presence of *C. mesnili* after 12 d. The transmission probability declined significantly when the recipient and the donor were kept in larger volumes of water (Fig. 1). Interestingly, the probability that an infected host infects an uninfected newborn decreased linearly with the logarithm of the volume of water (logistic regression of being infected or not compared to volume of water (Proc Catmod; SAS Institute 1998) $\chi^2 = 7.45$, $p = 0.0063$, $n = 60$).

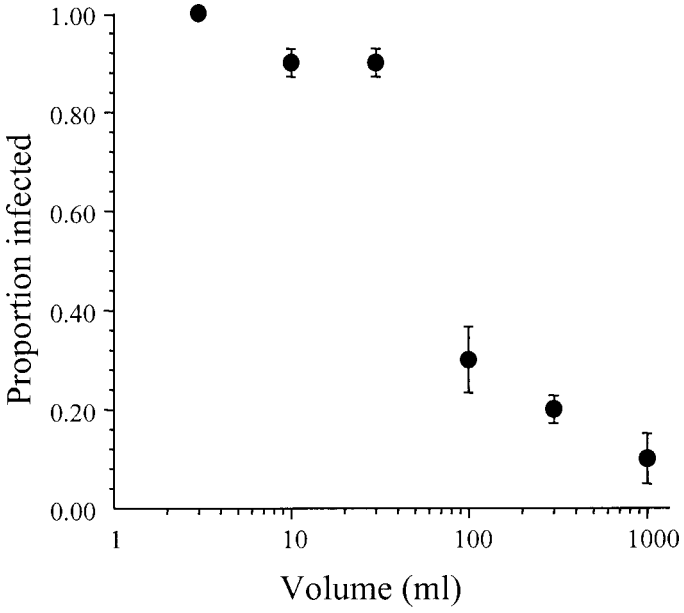

Fig. 1. Decline of transmission of *Caullerya mesnili* with increasing water volume in which the uninfected and infected *Daphnia galeata* were kept for 2 d. Means ($\pm$ binomial SE) are shown.

Susceptibility of different host age classes—Five recipient females per age stage (newborn, <12 h; juveniles, 2 d; preadults, 5 d; adults, >15 d) were kept together with one infected non-egg–bearing adult for 18 h in 100 ml medium without adding food. Eighteen replicates were used for the neonate and juvenile group and 12 replicates each for the preadult and the adult groups. After the infection period, the recipient animals were isolated from the donor animals by placing them in fresh medium. After 12 d during which medium was changed and standard food ration was added daily, all animals were dissected to quantify parasite infection. The proportion of infected animals per beaker was determined and arcsine-transformed data were used for statistical analysis. In 38.9% of the neonate treatments, 50% of the juvenile treatments, 41.7% of the preadult treatments, and 33.3% of the adult treatments at least one of the five recipient animals became infected. There was no significant difference between the proportion of infected animals per beaker among the age treatments (ANOVA: $df = 56$, $F = 1.042$, $p = 0.38$), indicating that all host age classes are approximately equally susceptible to *C. mesnili.*

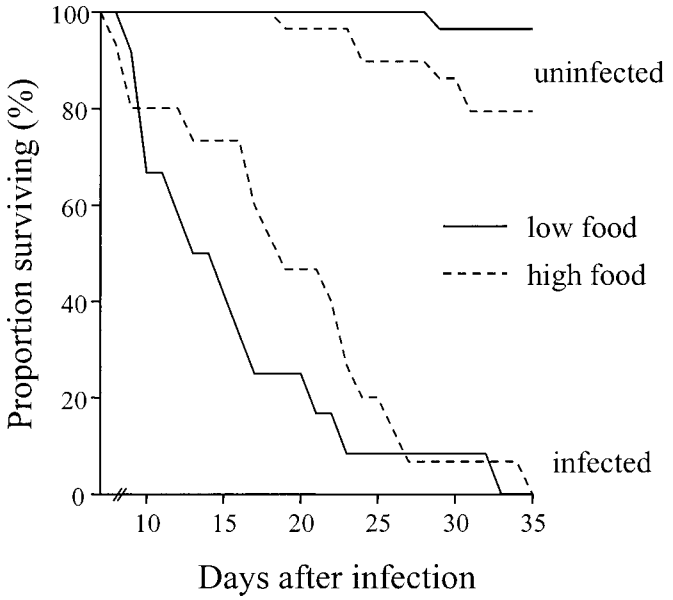

Fig. 2. Survival curves of infected and uninfected *Daphnia galeata* from the life table experiment.

Growth of the parasite within the host—To quantify the production of *C. mesnili* spores within the host and to determine when infection first becomes visible, we infected a cohort of 120 newborns (<24 h) by placing them together with 20 infected non-egg–bearing females for 3 d. Every second day, five animals were removed randomly and the total number of spore clusters in the dissected gut were counted. Until day 6 after exposure to the parasite, we did not detect any *C. mesnili* spores in dissected guts of experimental hosts. Thereafter, spores became visible and the spore load increased, reaching an average of approximately 70 spore clusters per host after about 18 d. As the number of spore clusters per host appeared not to increase further after day 15, we speculate that either the resources of the host were depleted, mature spores were released into the water, or hosts with higher infection intensities died. The latter hypothesis is supported by the observation that the mortality of infected females increased drastically after 15 d. Therefore the experiment was ended on day 20.

Infection and host life history at varying levels of nutrient condition—To estimate the impact *C. mesnili* has on its host we conducted a life table experiment with infected and uninfected individual females fed at two different food levels. A split brood design with one newborn per brood in each of the four treatments was used. Infection was achieved by placing a large infected non-egg–bearing female (uninfected in the controls) with each newborn. Each recipient–donor pair was kept in 100 ml medium. Half of the experimental animals per treatment were fed with the standard food ration (high food level), the second half with the equivalent of 0.2 mg C L^{-1} medium (low food level). After 2 d, each animal was placed in a 100-ml beaker filled with fresh medium. Medium was changed daily to guarantee a continuous food level. Offspring were removed and quantified daily, and hosts were checked for survival. Data for fecundity were log-transformed prior to statistical analysis (two-way ANOVA). Fifteen recipients did not become infected and were therefore removed from the analysis. During the experiment, six experimental animals and six control animals were lost due to handling error. For the final analysis, therefore, 15 infected and 22 uninfected high food level females, and 12 infected and 20 uninfected low food level females were used. The results of this experiment indicate that *C. mesnili* is able to significantly reduce the survival of hosts at both food levels (Fig. 2). After 35 d, all infected animals had died, while more than 80% of the uninfected controls survived (Fischer exact test: $p < 0.001$). The life span of *D. galeata* differed significantly between infected and uninfected treatment (survival analysis, log rank test: high food level, $\chi^2 = 33.9815$, $df = 1$, $p = <0.001$; low food level, $\chi^2 = 40.6486$, $df = 1$, $p = <0.001$) but there was no statistically significant difference between the food levels (survival analysis, log rank test: infected treatment, $\chi^2 = 1.7855$, $df = 1$, $p = 0.1815$). Infected animals kept at high and low food levels died at an average of 19.3 d (standard deviation [SD] = 7.5) and 15.7 d (SD = 7.0), respectively.

During the 35-d experimental period infected *Daphnia* produced on average fewer offspring per female than uninfected *Daphnia* (infected, 2.8 ± 2.9 SD [high food] and 6.2 ± 4.3 SD [low food]; uninfected, 76.3 ± 24.1 SD [high food] and 48.2 ±12.7 SD [low food]).

At high food level in the first clutch and at both food levels in the second clutch infected animals produced significantly

Table 1. Results of two-way ANOVA testing for the effect of the parasite with different treatments of the host (infected and uninfected, and high and low food level) on fecundity (mean offspring per female produced in first and second clutch).

Trait	Source	*df*	MS	*F*	*p*
First clutch	Infection	1	3.78	14.89	<0.001
	Food level	1	0.07	0.27	0.60
	Infection × food level	1	3.05	12.04	<0.001
	Error	64	0.25		
Second clutch	Infection	1	0.31	167.68	<0.0001
	Food level	1	34.73	1.48	0.23
	Infection × food level	1	0.89	4.30	<0.05
	Error	57	0.21		

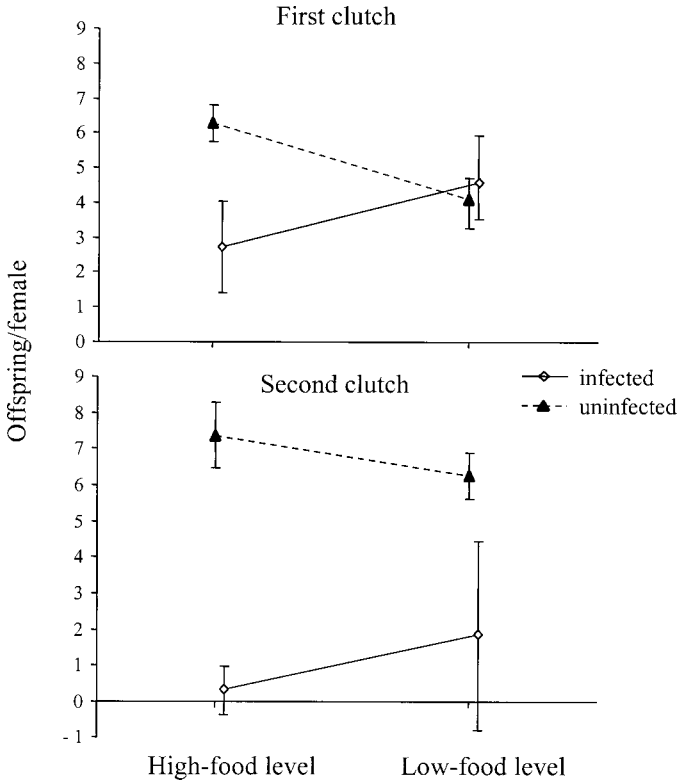

Fig. 3. Reproductive success of *Caullerya mesnili* infected and uninfected *Daphnia galeata* per female at high and low food levels in the first and second clutch. Means (±95% confidence limit) are given.

fewer offspring than uninfected controls (Table 1). We found no significant difference between the animals kept at the two food levels. Most infected animals did not produce a second clutch (Fig. 3). However, at both first and second clutch there was a significant interaction effect, indicating that the difference between the number of offspring in infected and uninfected females was higher in the high food level treatment. These results suggest that *C. mesnili* harms well-fed *D. galeata* relatively more than poorly fed *D. galeata.*

To further investigate the influence of nutritional condition of the host on parasite production we infected 50 newborn *D. galeata* recipients by placing them together with five infected non-egg–bearing females ($n = 6$). After 1 d without adding food, the animals were divided into two groups. Twenty-five animals were fed with the standard food level and the other group was fed at the low food level (0.2 mg C L^{-1}). The medium was changed daily, and offspring were removed. After 12 d, all live experimental animals were dissected, and the parasite spore clusters in the infected *D. galeata* were quantified. *C. mesnili* produced significantly more spore clusters in well-fed than in poorly fed *D. galeata* (Wilcoxon matched pairs test: $N = 6$, $R = 0$, $p < 0.05$). This indicates that the better the nutrient condition of the host, the higher the within-host growth of the parasite.

Parasite impact on host population dynamics—To test the influence of *C. mesnili* on *D. galeata* population dynamics, four infected and four uninfected microcosm populations were propagated for several host generations. Each of the initial populations consisted of five newborn (<24 h). We infected these populations by keeping them together with four infected (uninfected for the controls) non-egg–bearing donor animals in 100 ml medium. After 2 d, the donor animals were removed and the juveniles were placed in 1.5-liter beakers containing 1 liter medium to start the experiment. The standard food ration was added daily. Once a week for 12 weeks, the whole population in each beaker was counted and transferred to new medium. At the end of the experiment, all infected populations were extinct. The comparison between uninfected and *C. mesnili*-infected *D. galeata* populations in microcosms indicates that the parasite has a strong effect on host population dynamics (Fig. 4). In comparison with the controls, *C. mesnili* reduced the growth and density of the infected host population greatly.

From the fourth week onward, the infected populations had significantly lower population densities than the controls (Mann-Whitney *U*-test, $U = 0$, $Z = 2.31$, $p < 0.05$). *C. mesnili* drove all four replicates of infected host populations to extinction.

Discussion—We observed only horizontal transmission of *C. mesnili* to *D. galeata*. This is a common route of dispersal of several parasites of *Daphnia* (Ebert et al. 1997). Even though in the experiments with *C. mesnili* and *D. galeata* transmission from dead animals and contaminated water occurred, we postulate that in natural populations the main source of transmission stages is living donors. Recipient animals were never able to be infected with water contaminated with infection spores older than 2 d (K. Bittner pers. comm.); therefore, the survival time of the transmission stages in water is probably short.

It has been suggested that in natural cladoceran populations density-dependent transmission and impaired transmission at low temperature are the most likely explanations for the seasonal occurrence of microparasites (Brambilla 1983; Yan and Larsson 1988; Ebert et al. 1997). However, the occurrence of *C. mesnili* in Lake Constance was highest in autumn and winter (K. Bittner unpubl. data), when *Daphnia* densities and temperature were low (Gaedke and Straile 1998). The epidemic lasted for 7 months, so it is unlikely that the infection started earlier and infected animals survived longer because of low temperature and reduced predation. It is most likely that even this low host density and temperature is enough to allow the parasite to spread and other factors are limiting for the spread and persistence of the parasite at other times. For most planktonic parasites, a high parasite load at high host densities has been reported (Ruttner-Kolisko 1977; Brambilla 1983; Yan and Larsson 1988). However, a minimum host density for parasite persistence is likely (Ebert et al. 1997). Ebert (1995) found no significant effect of temperature on transmission of micro-

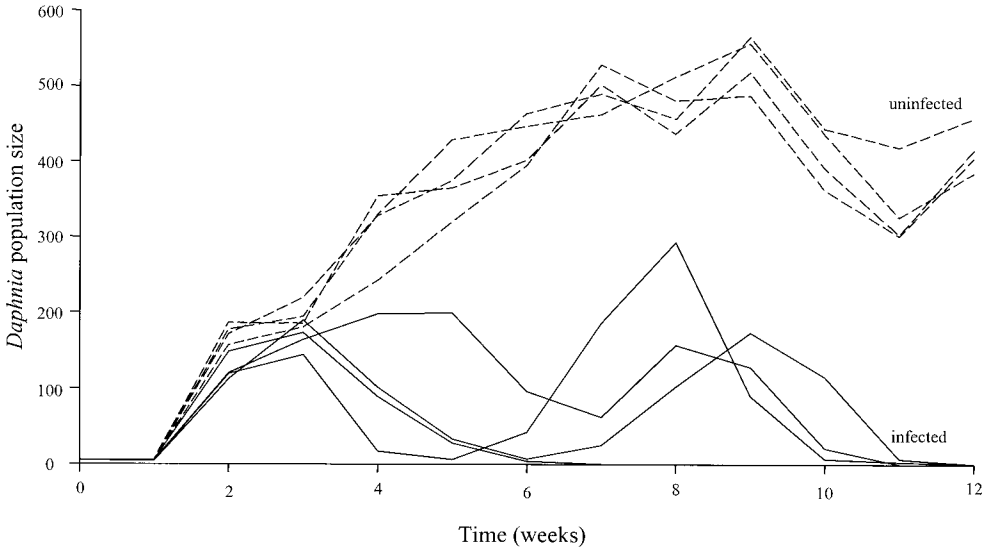

Fig. 4. Population size of *Daphnia galeata* in infected and uninfected microcosm populations. The lines indicate the individual replicates.

parasites above 12°C. Thus, we assume that water temperature differences between our laboratory experiments (20°C) and the field (mean water temperature of the upper 20 m in September 1997, when the *C. mesnili* epidemic started, was ca. 15°C, Bittner unpubl. data) had at most a minimal effect.

The virulence of different parasites to *Daphnia* hosts differs considerably. Some microsporidia, such as *Glugoides intestinalis* (formerly *Pleistophora intestinalis*) and *Ordospora colligata,* reduce the fecundity only slightly (Ebert 1995; Larsson et al. 1998), whereas white bacterial disease and the bacterial endoparasite *Pasteuria ramosa* nearly castrate the host completely (Green 1974; Ebert et al. 1996). Our data showed that *C. mesnili* is on the more virulent end of the range. During field surveys in Lake Constance, we also found that *Daphnia* infected with *C. mesnili* carry significantly fewer eggs in their brood pouch than uninfected animals (K. Bittner unpubl. data).

It has been claimed that *Daphnia* cultures kept under poor conditions are more susceptible to infection (Brambilla 1983; Seymour et al. 1984; Stazi et al. 1994). In contrast, Ebert (1995) found that the food level had no effect on host susceptibility to a microsporidian parasite. We found a significant interaction between infection and food level, indicating a stronger parasitic effect in well-fed *D. galeata* than in poorly fed *D. galeata.* This might be related to our findings that parasite growth inside the host is higher in well-fed hosts than in poorly fed hosts. The same finding was reported for *D. magna* infected with *P. ramosa* and kept at two food levels (Ebert et al. 2000).

There is some evidence that *Daphnia* populations may be regulated by parasites or epibionts (Green 1974; Allen et al. 1993; Ebert 1995). The criterion for a microparasite to be able to regulate its host population is essentially that the net death rate of infected hosts exceeds the net birth rate (Anderson and May 1981). This can happen when the parasite either increases the death rate and/or reduces the birth rate of infected hosts (Anderson and May 1981). In microcosm experiments with different horizontally transmitted parasites in *D. magna* populations, Ebert et al. (in press) found that the mean density of a host population declines as host fecundity reduction increases. This observation is consistent with our results and with epidemiological models (Anderson 1979; McCaullum 1994; McCaullum and Dobson 1995).

A stochastic simulation model by Ebert et al. (in press) indicated that parasites with a strong effect on host survival and fecundity are likely to drive small populations to extinction. This is partially a consequence of the low population densities associated with parasitic castration, but also a consequence of greater density oscillations in these populations (Ebert et al. in press). In our experimental microcosm populations we found evidence for both: infected populations had much lower mean densities and a seeming greater population fluctuation. Consequently, the chance of host extinction increases.

Conclusions—A regulatory function of parasites in natural populations has been debated. Brambilla (1983) suggested that it is unlikely that the microsporidium *Thelohania* sp. is able to regulate *D. pulex* density, although the parasite influences the population growth rate of its host. Other authors have argued that regulation of zooplankton populations by parasites is likely (e.g., Green 1974; Allen et al. 1993). We observed that *C. mesnili* was able to affect survival and fecundity of its host on an individual level and to bring host microcosm populations to extinction. At some times a high level of *C. mesnili*-infected *D. galeata* occurs in Lake Constance. Although natural conditions differ considerably from laboratory conditions (e.g., temperature, light, food, *Daphnia* densities), we suggest that in addition to predation and resource limitation, parasites also may influence *Daphnia* populations in large lakes with high level of vertebrate predators.

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Received: 3 October 2000
Amended: 14 September 2001
Accepted: 25 September 2001

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Acknowledgments

We would like to thank Karen A. Brune and Michael T. Monaghan for their linguistic improvement of this manuscript. We also thank Piet Spaak, Edward McCauley, and two anonymous reviewers for comments that helped improve this manuscript.