The parasite *Lernaeocera branchialis* on caged cod: infection pattern is caused by differences in host susceptibility

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SUMMARY

Variation in host susceptibility causes significant differences in infection rates between hosts living in a semi-natural situation. Such knowledge has implications for population dynamics and evolutionary models of host–parasite interactions as well as for estimations of parasite abundance. Infection rates by *Lernaeocera branchialis* (L.) were measured through time and space on caged Atlantic cod (*Gadus morhua* L.). One group of hosts, identified by their infection history, developed significantly higher infection rates than the others. These were fish which had been infected previously, but had lost their infection. Differences between groups were consistent through both time and space. Two types of cod seem to have been present in the caged population; a small group of inherently susceptible fish, which were infected, and reinfected if the parasite was lost, and another group of resistant hosts with a small chance of becoming infected.

Key words: Atlantic cod, *Gadus morhua*, *Lernaeocera branchialis*, susceptibility.

INTRODUCTION

Most macroparasites show a non-random distribution across their host population (Shaw, Grenfell & Dobson, 1998). The level of parasite aggregation has consequences for population regulation (Anderson & May, 1978; May & Anderson, 1978), as well as for epidemiological studies since, at increasing levels of aggregation, a larger number of hosts must be sampled in order to estimate parasite abundance. Furthermore, several evolutionary models hypothesize that parasites can be important selective agents on their hosts (Barbehenn, 1969; Hamilton & Zuk, 1982; Freeland, 1983; Hamilton, Axelrod & Tanese, 1990). A necessary requirement for such parasite-mediated selection is that the trait to be selected must covary with parasite numbers (Goater & Holmes, 1997; Skorping, 1998). For example, in the hypothesis proposed by Hamilton & Zuk (1982) on sexual selection, it is assumed that differences in male heritable susceptibility will be reflected in the distribution of parasites. The importance of parasites as selective agents is likely to increase when parasite distribution becomes less aggregated because more hosts harbour a higher number of parasites.

In order to understand why wild-living parasites aggregate within their host populations we need studies where the effect of heterogeneities in exposure rate to infective stages can be separated from heterogeneities in host susceptibility. As pointed out by Clayton, Pruett-Jones & Lande (1992), a major shortcoming in many studies has been that workers have been using variation in parasite numbers as a measure of variation in susceptibility. Experimental studies in the laboratory, where individuals are exposed to a known number of infective stages, have repeatedly shown that host susceptibility varies between individuals (Chevassus & Dorson, 1990; Bakke *et al*. 1992; Wakelin, 1994). These are not easily extrapolated to natural situations because exposure rates may differ widely from those in the field (Quinnell & Keymer, 1990), and also because host susceptibility may be affected by conditions in the laboratory (Lloyd, 1995). An alternative is to run field experiments where hosts can be exposed to naturally occurring transmission stages under environmental conditions more similar to those experienced in the wild. By using ectoparasites on individually marked hosts, it should be possible to compare infection rates between host individuals, and examine if differences in rates are consistent through time.

This paper describes fluctuations in the population of *Lernaeocera branchialis* (L.) on caged cod (*Gadus morhua* L.) for 598 days. *L. branchialis* uses mainly different species of flatfish as intermediate hosts (Kabata, 1979), but can also develop on other fish species (Lester & Roubal, 1995). After mating, the pregnant female has a short free-living period

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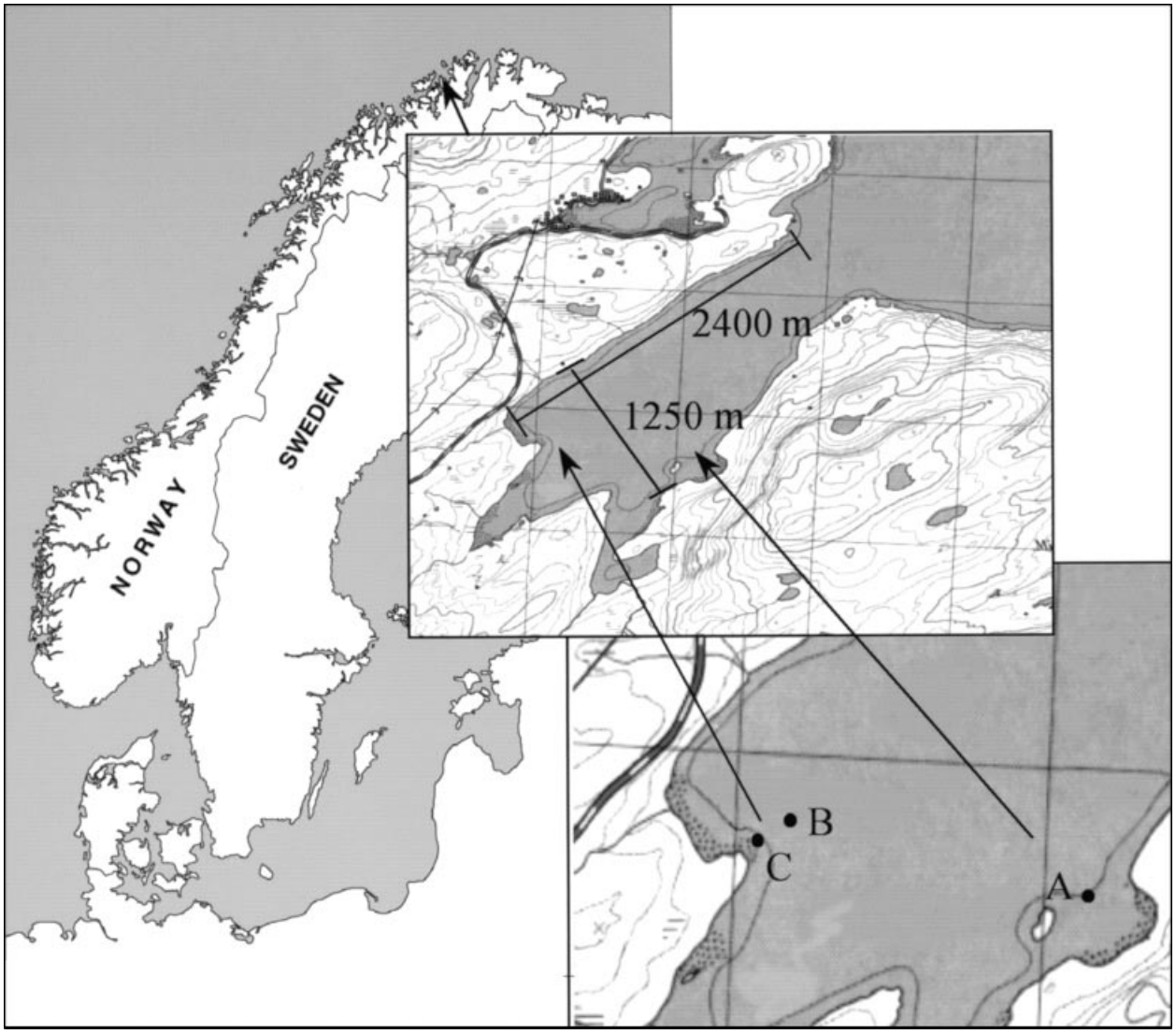


Fig. 1. Chart of Kvalfjorden with cage locations marked by dots. Four net bags were placed in cage A, while cages B and C had 1 net bag each.

searching for the definitive host, a gadoid fish. If successful, she will settle at the base of the gill arches on the ventral side, and undergo a metamorphosis where the head penetrates into the heart region of the fish (Grabda, 1991). The parasite is known to influence both growth and level of liver fat, and may be lethal, especially to young fish (Khan, Lee & Barker, 1990). The parasite may live up to 18 months (Lester & Roubal, 1995) and can not move between hosts.

In the present study a field experimental approach was used to address the following question. Do infection rates vary between hosts due to inherent differences in susceptibility? Exposure can be assumed to be random within each of the host locations used in this experimental set-up. If then the pattern of infection is caused mainly by factors unrelated to host susceptibility, infection rates should show random variations between host individuals.

MATERIALS AND METHODS

Six hundred cod were caught by floating trawl off the coast of Finnmark County, Norway, and caged in Kvalfjorden (70° 42′ N and 23° 48′ E). The fish were allowed to recover from capture and acclimatized for 2 weeks while fed several times a day with artificial food. Thereafter, the fish were caged in 6 net bags (height 4 m, volume 40 m^3 each) at 3 different locations in the fjord. One hundred cod were placed in each of 4 net bags in cage A (see Fig. 1). These were all free from infection by *L. branchialis*, with 3 accidental exceptions. The depth at low tide was 8 m at this cage. This paper is part of a larger study, and fish from another 2 cages, which were placed in the study area for other reasons (labelled B and C in Fig. 1), were also included in the present data. At caging 100 cod were placed at random, with respect to infection, in one net bag in each of cage B and C (Table 1). The cages were placed 200 m and 50 m

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Table 1. Infection levels of *Lernaeocera branchialis* on the caged cod at the start of the experiment are given separately for the 3 cages in the experiment together with the mean and range of both body mass and length of the fish at caging, as well as increase in body mass and length through the study period

(Cage-labelling refers to Fig. 1)

	Cage A	Cage B	Cage C
Infection			
Abundance	0·01	0·85	0·71
Prevalence	1·1 %	38·5 %	40·9 %
Mass (g)			
Mean	1876·4	1855·2	2013·2
Range	1025·0–2905·0	1110·0–2525·0	1110·0–4300·0
Increase, mean	2454·6	2408·6	2248·7
Increase, range	410·0–4170·0	705·0–4490·0	310·0–5070·0
Length (cm)			
Mean	60·6	60·5	61·2
Range	47·5–71·0	48·0–66·0	50·0–79·5
Increase, mean	10·6	10·3	9·5
Increase, range	3·5–21·0	2·0–18·5	1·0–20·5

from the shore respectively. Depths at low tide were 31·5 m at cage B and 18·5 m at cage C. In the fjord the average water level change is 1·8 m (Statens Kartverk, 2001) between high to low tide. Strong tidal currents, which are especially pronounced close to the shores (Fig. 1), continuously replaced the water within the cages.

Before the separation into different cages fish were inspected for *L. branchialis*, and length and mass were recorded (Table 1). All fish were also individually tagged with external anchor tags (T-tags). Before handling, each fish was anaesthetized in 0·15 % chlorobutanol ($C_4H_7Cl_3O$). During the experimental period the fish were anaesthetized, *L. branchialis* counted and length and mass recorded 6 times; on days 0, 74, 327, 431, 522 and 598 after caging. The large gap in collection times between the second and third sample was caused by bad weather conditions which made it impossible to transport the fish between the cages and the location where they were anaesthetized and inspected. On day 598 all surviving fish were killed and sexed. The otoliths were removed for age determination. During the experiment cod were fed cuttings from the codfillet industry and herring meal mixed with commercial fish food ('Salmomix 45 %'). Food was added to the cages twice a week, and what was not eaten, sank out of the cage within a period of less than 30 sec.

Only fish which survived longer than day 327, were included in the analyses. All statistical tests were run using generalized linear models (GLMs) in the Glim4 computer package (Crawley, 1993). Changes in frequency distribution of parasites through time were tested using a contingency table (Poisson errors and log link function). Changes in rates of infection were treated as binary data: group (i) included fish where numbers of parasites increased during a time-period, and group (ii) included fish where numbers of parasites did not increase. The data were used in a logistic regression model with binomial errors in Glim4 (Crawley, 1993). Groups of cod, identified by infection history, were tested for differences in age distributions using contingency tables (GLM with Poisson errors and log link function). The variables were included in the models in a forward stepwise manner. Significance of effects in the models were tested by comparing the change in deviance by the removal of a term from the model with the values of Chi-square tables in accordance with Crawley (1993).

RESULTS

A total of 495 cod survived the experimental period. Of the survivors, 339 fish could be identified throughout the study, while the remaining individuals had lost their tags and were subsequently re-tagged. Of the 339 cod, which were identified throughout the study, 79 % remained uninfected, while the rest of the fish harboured the parasite at one or more sampling points. During the study period there was a decline in numbers of hosts harbouring more than 1 *L. branchialis* (Fig. 2; $\chi^2 = 9{\cdot}29$; $P = 0{\cdot}0023$ at 1 D.F.). The death of 22 fish was caused by predation by the otter, *Lutra lutra*, which in most cases made the fish impossible to inspect for parasites. These killings took place during the dark period each year, between late November and early February. The otter entered the cages through self-made holes in the net. However, among the dead fish which could be inspected, intensity and prevalence were within the levels measured among the survivors

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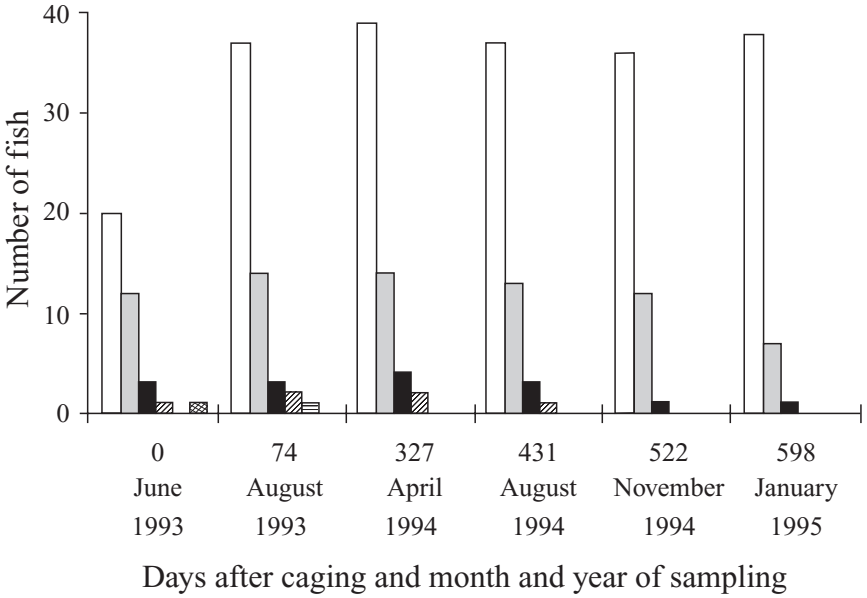


Fig. 2. Numbers of cod harbouring different numbers of *Lernaeocera branchialis* during the period of caging. Only fish which appeared in all samples ($n = 339$) were included. Number of *L. branchialis* per fish □ 1; ▨ 2; ■ 3; ▨ 4; ▤ 5; ▩ 6.

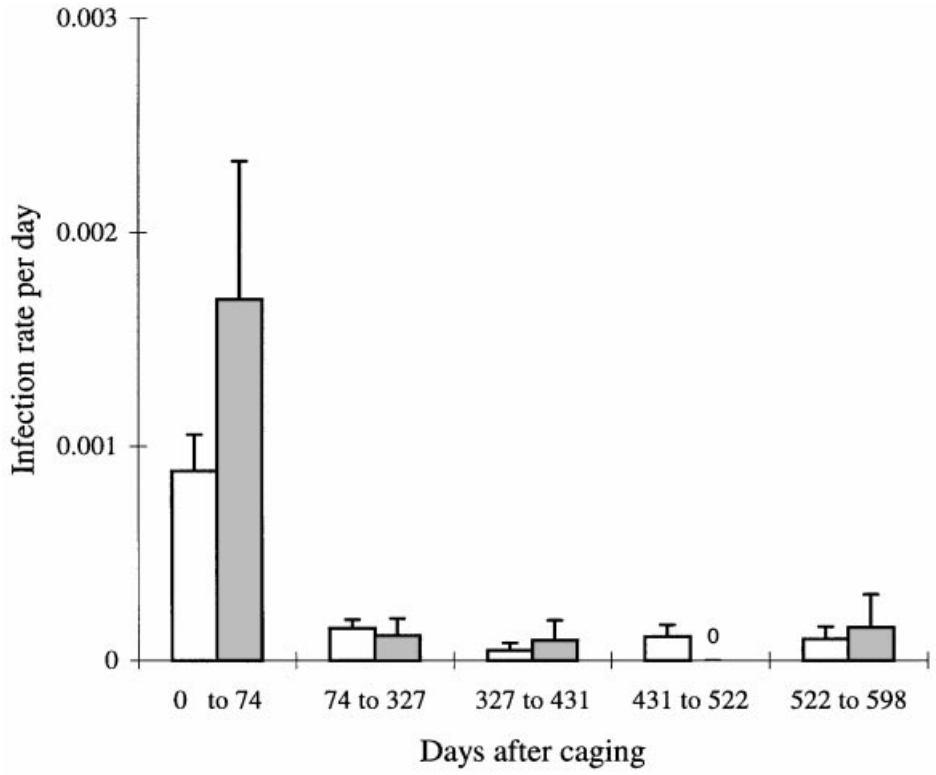


Fig. 3. Infection rates, with standard errors, by *Lernaeocera branchialis* for fish which were free from infection at the start of a time-period (open columns), compared to rates for fish which harboured the parasite (filled columns). '0' = No fish acquired new infections.

(intensity = 1.78, prevalence = 0·36, $n = 22$). Data on the surviving fish therefore seem not to have been biased by parasite-related deaths.

The rate at which *L. branchialis* established within the caged population was high during the first time period (Fig. 3), but then dropped to a lower level ($\chi^2 = 50{\cdot}5$; $P < 0{\cdot}001$ at 1. D.F.). Seasonal fluctuations in the changes in infection rates were not apparent from the analyses. Inclusion of a second order and third order parameter of the 'time' variable did not exert significant influence on infection rates ($\chi^2 = 0{\cdot}30$; $P = 0{\cdot}58$ at 1 D.F, and $\chi^2 = 2{\cdot}56$; $P = 0{\cdot}11$ at 1 D.F., respectively).

Infection rates were compared between groups of

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Table 2. The influence of infection history and location on infection rates of *Lernaeocera branchialis* on cod during successive time-periods

('Infection history' identifies 2 groups among the cod. Fish which were uninfected at the start of the time-period are compared to fish which harboured the parasite (see also Fig. 3). 'Location' refers to the 3 locations in the fjord where the cod were caged. In the analysis 'infection rate' was treated as a binary response variable, and included in models (GLMs) with binomial errors.)

Days after caging	Inf. history (D.F. = 1)	Location (D.F. = 2)	Inf. history × loc. (D.F. = 2)
0–74	$\chi^2 = 1{\cdot}93$ $P = 0{\cdot}16$	$\chi^2 = 4{\cdot}94$ $P = 0{\cdot}09$	$\chi^2 = 0{\cdot}31$ $P = 0{\cdot}86$
74–327	$\chi^2 = 1{\cdot}37$ $P = 0{\cdot}24$	$\chi^2 = 2{\cdot}44$ $P = 0{\cdot}30$	$\chi^2 = 0{\cdot}38$ $P = 0{\cdot}83$
327–431	$\chi^2 = 0{\cdot}28$ $P = 0{\cdot}60$	$\chi^2 = 0{\cdot}92$ $P = 0{\cdot}63$	$\chi^2 = 0{\cdot}002$ $P = 0{\cdot}999$
431–522	$\chi^2 = 1{\cdot}76$ $P = 0{\cdot}19$	$\chi^2 = 1{\cdot}64$ $P = 0{\cdot}44$	$\chi^2 = 0{\cdot}001$ $P = 0{\cdot}999$
522–598	$\chi^2 = 0{\cdot}13$ $P = 0{\cdot}72$	$\chi^2 = 0{\cdot}30$ $P = 0{\cdot}86$	$\chi^2 = 3{\cdot}099$ $P = 0{\cdot}21$

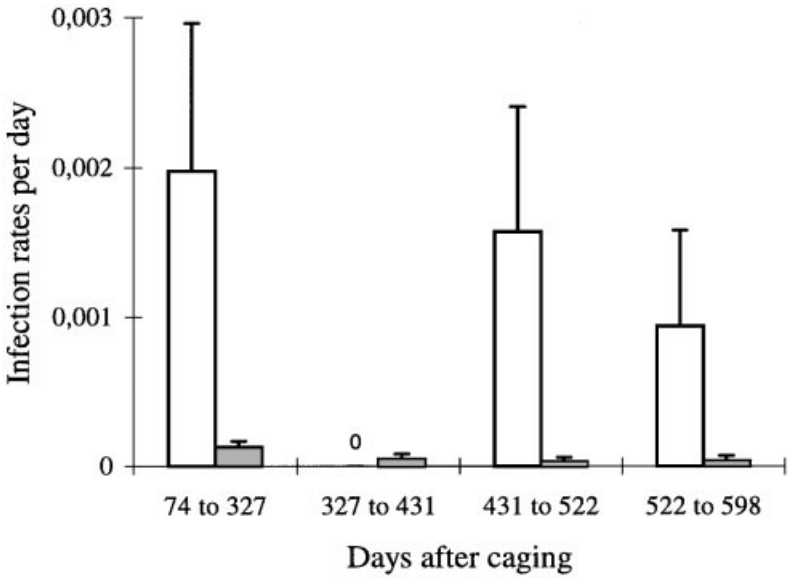


Fig. 4. Infection rates, with standard errors, by *Lernaeocera branchialis* for 2 subgroups of the fish classified as 'uninfected' in Fig. 3. Fish which had lost the infection, and therefore were free from the parasite at the start of the time-period (open columns), are compared to fish which were not recorded as infected prior to the start of the time-period (filled columns). '0' = No fish acquired new infections.

cod identified by their infection history. Rates did not differ between fish which were uninfected at the start of a time-period, compared to infected fish (Fig. 3). This was shown statistically by the lack of difference within all of the 5 time-periods which were compared (the statistics are given in Table 2). However, 1 subgroup among the uninfected fish showed significantly higher rates of infection than the others. These were the fish which had been infected, but had lost their infection prior to the start of the time-period under investigation (Fig. 4). This loss of infection was identified among individuals which had harboured the parasite at one or more of the previous samplings but were free from infection at the start of the time-period in question. Within 3 of the 4 time-periods, these cod were more likely to be infected than the cod which had never harboured the parasite at any of the previous sampling points (the statistics are given in Table 3). In the second interval in Fig. 4, where infection rate is zero in the group of previously infected cod, only 11 of the individuals which were free from infection after 327 days in the cage, had been recorded as infected earlier in the study. None of these were recorded as infected after 431 days in the cage. Furthermore, when compared to the group of cod which harboured the parasite at the start of a time-period, the group of cod which had lost all their parasites, showed significantly higher infection rates in 2 of the 4 time-periods (the statistics are given in Table 4). This

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Table 3. Fish which had been infected by *Lernaeocera branchialis*, but had lost their infection prior to the start of the time-period, are compared to fish which were not recorded as infected (see Fig. 4)

(These are subgroups of the 'uninfected' fish in Fig. 3. In the analyses 'infection rate' was treated as a binary response variable, and included in models (GLMs) with binomial errors.)

Days after caging	Inf. history (D.F. = 1)	Location (D.F. = 2)	Inf. history × location
74–327	$\chi^2 = 7{\cdot}92$ $P = 0{\cdot}005$	$\chi^2 = 2{\cdot}14$ $P = 0{\cdot}34$	$\chi^2 = 3{\cdot}06$ $P = 0{\cdot}08$ (1 D.F.)
327–431	$\chi^2 = 0{\cdot}11$ $P = 0{\cdot}74$	$\chi^2 = 0{\cdot}61$ $P = 0{\cdot}74$	$\chi^2 = 0{\cdot}0002$ $P = 0{\cdot}999$ (2 D.F.)
431–522	$\chi^2 = 13{\cdot}60$ $P = < 0{\cdot}001$	$\chi^2 = 3{\cdot}58$ $P = 0{\cdot}17$	$\chi^2 = 0{\cdot}24$ $P = 0{\cdot}89$ (2 D.F.)
522–598	$\chi^2 = 7{\cdot}03$ $P = 0{\cdot}008$	$\chi^2 = 1{\cdot}66$ $P = 0{\cdot}44$	$\chi^2 = 0{\cdot}118$ $P = 0{\cdot}94$ (2 D.F.)

Table 4. Fish which had been infected by *Lernaeocera branchialis* previous to the time-period in question, but had lost the infection (a subgroup of the 'uninfected' in Fig. 3), are compared to fish which harboured the parasite at the start of the period (the 'infected' fish in Fig. 3)

(In the analyses 'infection rate' was treated as a binary response variable, and included in models (GLMs) with binomial errors.)

Days after caging	Inf. history (D.F. = 1)	Location (D.F. = 2)	Inf. history × location
74–327	$\chi^2 = 7{\cdot}31$ $P = 0{\cdot}007$	$\chi^2 = 4{\cdot}64$ $P = 0{\cdot}10$	$\chi^2 = 0{\cdot}0008$ $P = 0{\cdot}977$ (1 D.F.)
327–431	$\chi^2 = 0{\cdot}21$ $P = 0{\cdot}65$	– –	– –
431–522	$\chi^2 = 10{\cdot}68$ $P = 0{\cdot}001$	$\chi^2 = 3{\cdot}22$ $P = 0{\cdot}20$	$\chi^2 = 0{\cdot}001$ $P = 0{\cdot}999$ (2 D.F.)
522–598	$\chi^2 = 2{\cdot}41$ $P = 0{\cdot}12$	$\chi^2 = 0{\cdot}34$ $P = 0{\cdot}84$	$\chi^2 = 2{\cdot}82$ $P = 0{\cdot}24$ (2 D.F.)

pattern may have been caused by age-related differences in infection rates. If so, age of the fish should differ between groups identified by infective history. This was not found to be the case within any of the time-periods ($\chi^2 < 8{\cdot}57$; $P > 0{\cdot}29$ at 7 D.F.).

Differences in infection rates between individuals did not depend on which location the fish were placed in. This appeared from the fact that 'location' neither affected infection rate (Tables 2, 3 and 4), nor influenced the effect of 'infection history' within any of the time-periods (non-significant effects of the 'infection history × location' interactions; Tables 2, 3 and 4).

DISCUSSION

In this study, infection rates did not vary randomly between individual cod. Fish which had been infected previously with *L. branchialis* and had lost the infection, had a significantly higher rate of infection than cod which previously were recorded as uninfected. This difference was consistent through time and between locations. With respect to infection rates, 2 types of cod therefore appear to have been present in the caged population; one large group with a relatively small chance of becoming infected and another smaller group with a much higher risk of acquiring the parasite. The strong water currents in the caging area, which caused continuously replacement of the water within the cages, should produce random variation in exposure to infective stages among fish within each cage. Furthermore, during the experimental period there were no significant differences in infection rates between the cages. Therefore, the differences in rates between the 2 groups of cod, could not have been caused by differences in the rate of exposure, but must have been related to phenotypic differences.

An alternative explanation would be that the smaller group of hosts may have differed in their behaviour in a way that made them more frequently exposed to transmission stages, for example, by

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occupying the bottom of the net bags. Poulin, Rau & Curtis (1991) showed that variation in behaviour influenced infection rates among laboratory reared brook trout fry (*Salvelinus fontinalis*) infected by the crustacean ectoparasite *Salmincola edwardsii*. However, if host behaviour were an important factor in the present study the frequently exposed hosts should show high levels of infection rates throughout the study period, independent of their previous infection status. This was not observed.

The assumption that the caged cod population initially consisted of a small group of inherently susceptible fish among a larger group of resistant ones, would explain both the decrease in the infection rates within the whole group of cod, and the fluctuating rate of infection among the fish which had carried the parasite earlier, but lost it. The infection rate within the whole group should decline because the number of susceptibles rapidly became infected and moved into the infected class with a low probability of reinfection. Since this parasite may live for 18 months (Lester & Roubal, 1995), few of these fish had lost their infection after 327 days in the cage. This may explain the lack of new infections among the group of susceptibles during the second time-interval.

Are cod with a previous record of carrying the parasite more vulnerable to reinfection due to parasite-induced increase in susceptibility? Such patterns were observed in the laboratory on juvenile sticklebacks (Gasterosteidae) infected by the crustacean ectoparasite *Argulus canadensis* (Wilson, 1916; Poulin & FitzGerald, 1989), and on brook trout fry infected by another crustacean ectoparasite *S. edwardsii* (Poulin *et al*. 1991). However, the present data seem not to fit this explanation. *L. branchialis* did not induce higher infection rates among the fish harbouring the parasite, because the number of fish carrying more than 1 parasite did not increase with time, and fish infected by the parasite at the start of a given time-period did not show higher infection rates than uninfected hosts. A supposed increase in susceptibility due to earlier infection may also take effect only after the fish have lost the infection. However, if parasite-induced facilitation of re-infection were the most important factor causing changes in infection rates, we would expect rates to be low at the start of the study period, and thereafter increase as parasites died and numbers of previously infected fish increased through time. This was not observed.

The argument that the caged cod initially consisted of a small group of inherently susceptible fish among a larger group of resistant ones, depends on the assumption that the observed temporal variations in infection rate are caused by changes in availability of susceptibles. The distribution of the cod population in 2 distinct groups, susceptible or resistant hosts, may, however, not be a realistic model. Susceptibility can appear as a gradient from highly susceptible to resistant hosts. This view does not change the argument that infection rates decreased through time due to declining probability of encountering susceptible hosts because the highly susceptible individuals were infected during the first part of the study and moved into the infected class with a low probability of reinfection.

Other explanations for the observed decrease in infection rate with time are possible, but less likely. A decrease in rates of infection with time could be caused by stress during the caging process, which may increase initial susceptibility to parasites (Lloyd, 1995). However, fish seem to acclimatize to cages over a short time. Pickering (1987) argued that within 3 weeks most individuals of several species do not show signs of immune suppression. Seasonal or annual changes in densities of transmission stages may cause a decrease in rate of infection through time. If densities decreased through the first autumn and winter, the rates of infection should have increased again over the second summer (from April to early November which would be from 327 to 522 days in the cage). This did not happen. Neither is it likely that annual fluctuations in densities of transmission stages caused the observed pattern, because changes in rates of infection with time differed between groups of cod. For example, infection rates increased in the group of infected cod between the second-to-last and the last time-period. At the same time the group of cod which had lost their infection, showed a 40% decrease in infection rates. It is therefore reasonable to conclude that the decrease in infection rates with time was caused by a declining number of susceptible hosts.

Are the present results comparable to the wild situation? It is not likely that factors tied to the caging situation, for example stress, would affect a fraction of the caged cod, i.e. the susceptible fish identified by their infection history, more seriously than the rest of the population. However, the availability of food may be more uniformly distributed, and for part of the year more abundant, between caged individuals than experienced by wild-living fish. As a consequence, differences in host resistance caused by fluctuation in nutritional resource may be more pronounced among wild-living hosts. The frequent availability of high quality food may explain why the numbers of multi-infected hosts decreased throughout the study period.

Since this is a relatively pathogenic parasite (Khan, 1988; Khan *et al*. 1990), cod that are able to avoid it should have a fitness advantage relative to the susceptible group. Why then, does resistance not spread to the whole cod population? We suggest 2 possible mechanisms that may maintain such polymorphism in susceptibility.

Given that parasites show virulence specific to genotypes, Hamilton (1980) hypothesized that be-

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cause of frequency-dependent selection, parasites may generate cyclical processes where the frequencies of host genotypes are changing with time.

Susceptibility to *L. branchialis* may also be maintained if resistance is costly and therefore affects some other factors related to fitness. Life-history theory suggests that hosts may increase their fitness by suppressing responses against parasites, in order to restrict the operation of the immune system for frequently occurring and pathogenic parasites (Behnke, Barnard & Wakelin, 1992).

The present data show that variation between individuals in rates of infection was consistent through time and space when living in environments similar to the natural one. We conclude that this pattern was caused by inherent differences in host susceptibility.

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