

# Spatial variation in susceptibility to infection in a snail–trematode interaction

A. C. KRIST<sup>1</sup>\*†, C. M. LIVELY<sup>1</sup>, E. P. LEVRI<sup>1</sup> and J. JOKELA<sup>2</sup>

<sup>1</sup>Department of Biology, Jordan Hall 142, Indiana University, Bloomington, IN 47405-3700, USA

<sup>2</sup>ETH Zürich, Experimental Ecology, ETH-Zentrum NW, CH-8092, Zürich, Switzerland

(Received 27 November 1999; revised 1 April 2000; accepted 10 April 2000)

## SUMMARY

Parasites should be better at infecting hosts from sympatric populations than allopatric populations most of the time (parasite local adaptation). In a previous study of a population of snail parasites (*Microphallus* sp.) from Lake Alexandrina, New Zealand, we found that *Microphallus* was more infective to snails (*Potamopyrgus antipodarum*) in shallow water but not in deep water. Here, we repeated the original study and also monitored the development of the parasite. We found that parasites from shallow water were more infective to hosts from shallow water and developed more rapidly in these hosts. In contrast, parasites from deep water were not more infective to hosts from deep water and did not develop more rapidly in them. These results suggest clinal variation in the susceptibility of these snails, with shallow-water snails more susceptible than deep-water snails. We offer 2 possible explanations for these results. First, gene flow in the *Microphallus* population is primarily from shallow to deep water, leading to an asymmetric pattern of local adaptation. Alternatively, snails from shallow water may be more susceptible for reasons independent of gene flow, perhaps due to differences in host condition between habitats.

Key words: susceptibility, spatial variation, gene flow, host–parasite coevolution, local adaptation.

## INTRODUCTION

Under theory, parasites should become adapted to infecting local populations of their hosts in the same way that organisms become adapted to the local conditions of their physical environments. However, for parasites, the environment (the host population) can also evolve, potentially leading to oscillatory dynamics in both host and parasite allele frequencies (see Hamilton, Axelrod & Tanese, 1990; Peters & Lively, 1999). Nonetheless, parasites should become better at infecting individuals drawn from their local host population than individuals randomly drawn from an allopatric host population most of the time, assuming some degree of host population structure (Ladle, Johnstone & Judson, 1993; Ebert, 1994; Judson, 1995; Gandon *et al.* 1996; Lively, 1999); a number of recent studies are consistent with this prediction (Parker, 1985; Lively, 1989; Ballabeni & Ward, 1993; Ebert, 1994; Ahmed, Mundt & Coakley, 1995; Burdon & Thompson, 1995; Webster & Woolhouse, 1998; see Kaltz *et al.* 1999 for a counter example). The degree of local adaptation, which is usually measured as the infectivity by parasites to local host populations compared to

remote host populations, is expected to be affected by both gene flow between populations, and the effect of infection on host fitness (Lively, 1999).

In previous studies, we have found that the trematode *Microphallus* sp. is locally adapted to populations of its snail host, *Potamopyrgus antipodarum* (Lively, 1989; Lively & Dybdahl, 2000). Such a result suggests that selection resulting from local coevolution results in parasite population structure at loci involved in infectivity. A different result, however, was observed when we conducted reciprocal cross-infection experiments with parasites taken from different depth-stratified habitats within the same lake (Lively & Jokela, 1996). In this case, parasites collected from snails in the shallow water were better at infecting shallow-water snails than deep-water snails, but the converse was not true; parasites collected from deep water were not better at infecting deep-water snails than shallow-water snails. This result is logical in the context of the foraging behaviour of ducks, the definitive host for the trematode. These dabbling ducks forage in shallow water, and therefore would seem much more likely to pick up infections in shallow water. The sexually produced eggs resulting from these infections would then be dispatched over both shallow and deep habitats, but recycling of the infection, and hence coevolution, should be restricted to the shallow water.

Because coevolutionary interactions can lead to periods in which locally adapted parasites appear to

\* Corresponding author: Department of Biology, University of Wisconsin-Eau Claire, Eau Claire, WI 54701, USA. Tel: +715 836 5386. Fax: +715 836 5089. E-mail: krist@hawaii.edu

† Current address: Department of Biology, Gerstacker Hall, Hiram College, Hiram, OH 44234, USA.

be better at infecting remote populations (Morand, Manning & Woolhouse, 1996; Kaltz & Shykoff, 1998; Lively, 1999), studies indicating that parasite populations are not more infective to local populations should be repeated. In the present study, we repeated the cross-infection experiment 1 year after the original experiment. We were especially interested to determine whether the asymmetry between the shallow and deep sources of parasites was maintained. In the original study, we also found evidence suggesting that parasites may have different developmental schedules depending on the source of the host (Lively & Jokela, 1996). Unfortunately, the experiment was not replicated within each sampling time, and hence the result could have been due to random variation among containers having different host–parasite combinations. In the present study, we used more replication within sampling periods to evaluate the hypothesis that development rate is affected by the particular host–parasite combination. We also determined whether parasites cause different levels of mortality in hosts from different sources.

#### MATERIALS AND METHODS

##### *Natural history of the interaction*

The parasite is an undescribed species of *Microphallus* (Digenea: Microphallidae; Lively, 1987) that has 1 intermediate host, the prosobranch snail *Potamopyrgus antipodarum* (Prosobranchia: Hydrobiidae; Gray, 1843), and the definitive hosts are water fowl. Snails become infected by ingesting *Microphallus* eggs. The eggs then hatch, and, if not killed by the snail, the resulting larva develops vegetatively into hundreds of cysts (metacercariae) that lodge in the snail's viscera causing complete sterilization. The cysts 'hatch' following ingestion (along with the snail) by waterfowl, and become hermaphroditic adults. These adults cross-fertilize (see Dybdahl & Lively, 1996 for a genetic analysis) and produce eggs, thus completing the life-cycle. The snails occur as diploid sexuals and triploid asexuals (Dybdahl & Lively, 1995). In Lake Alexandrina, there is a cline in ploidy level such that diploid sexuals are more common in shallow water whereas triploid asexuals are more common in deep water (Jokela & Lively, 1995a; Fox *et al.* 1996).

##### *General methods*

In January 1996, we collected several thousand snails from 2 habitats of Lake Alexandrina on the South Island of New Zealand at a site called West Bay (see Fig. 1 in Jokela & Lively, 1995b). We collected snails in the shallow water from willow roots by dragging a kick net through the roots. We used nets and snorkeling equipment to collect snails from the deep water (4–6 m deep) which live on

*Elodea canadensis*. We conducted the experiment at the Edward Percival Field Station in Kaikoura, New Zealand.

We fed metacercariae of 75 *Microphallus*-infected snails collected from the shallow habitat to 3 mice (each mouse received the metacercariae of 25 infected snails). We used mice as the definitive hosts because *Microphallus* produces viable eggs in mice (Lively & McKenzie, 1991). Similarly, we fed metacercariae of 65 *Microphallus*-infected snails collected from the deep habitat to 3 different mice (each mouse received the metacercariae of 19–26 infected snails). We kept 3 additional mice as controls, which were not fed metacercariae. We collected fecal pellets (containing *Microphallus* eggs in the 6 experimental mice) 2–5 days post-infection (Lively & McKenzie, 1991). For each of the 3 parasite sources (shallow, deep, control), we randomly distributed the faeces among 8 different 2 l containers (8 shallow, 8 deep, 8 control, for a total of 24). The water in each 2 l container was changed frequently to prevent fouling.

We exposed these faeces to 9600 snails (400 adult snails in each of 24 containers). Large numbers of snails were exposed so that we could subsample from each container at 5 different times and thereby follow the development rate of the parasite. We exposed the snails by adding the contents of each of the 2 l containers holding the faeces, to a different 44 l container holding 400 adult snails. For each parasite source there were 8 44 l containers, 4 containing 400 snails from the shallow-water habitat and 4 containing 400 snails from the deep-water habitat. This design allowed snails from both habitats to be exposed to parasites from their own habitat as well as from the foreign habitat. The control snails were necessary because we collected the snails from the lake and consequently some were expected to have been already infected. Also, the controls allow us to determine whether the experimental infection was successful and to determine the proportion of snails that were already infected in each parasite source. Every second day we changed the water in the 44 l containers to prevent fouling. Thirteen days post-exposure, we transported the snails to Indiana University for further study. At Indiana University, we maintained the snails in 14·2 l containers at 17 °C.

##### *Does total parasite prevalence differ between hosts from different sources?*

We sampled the experiment 5 different times (13, 19, 24, 33 and 57 weeks post-exposure). At each time, we measured the length of the shell of 50 individuals per container (total of 1200 snails at each time), determined whether the snails were infected with *Microphallus* or any other digenean, and determined the stage of development for *Microphallus* infections. The procedure for determining the stage of development is described below. We analysed preva-

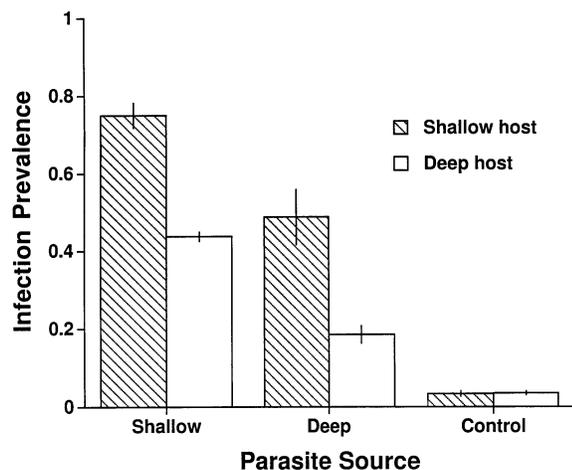


Fig. 1. Prevalence of infected snails by treatment averaged over all 5 time-periods. The prevalence of infection over all times was pooled because the repeated measures analysis revealed no significant effect of time nor any indication that total infection varies with time (the parasite by time interaction was not significant). The overall means for each treatment were calculated using the mean of each replicate mean ( $N = 4$ ). Therefore, the standard error (shown in the error bars) was calculated as  $S.E. = S.D./\text{square root of } N$ , where  $N$  is 4. The overall means for each treatment were calculated with 975–1000 snails.

lence in the experiment using repeated measures ANOVA with each container as the repeated unit. The dependent variable was prevalence of infection. The between-subject effects were host and parasite and the within subject effect was time. Control snails were not included in the analysis because prevalence in the controls was the same in both parasite groups ( $T = 0.07$ , D.F. = 6,  $P = 0.95$ , Fig. 1). We made planned comparisons to examine specific hypotheses about host effects. For this analysis we examined only 3 of the 5 dates, beginning (13 weeks), middle (24 weeks), and end (57 weeks), to avoid singularity in the covariance matrix. The 3 dates were chosen to include the entire duration of the experiment.

#### *Does host mortality differ between parasites from different sources?*

At Indiana University, we counted the number of dead snails in each container every week for a period of 28 weeks. We analysed mortality with a 2-way ANOVA with mortality per box as the dependent variable and host and parasite source as the independent variables.

#### *Does parasite developmental stage differ between hosts from different sources?*

At each time that we sampled the experiment we examined the parasites in each infected snail and

scored each infection as 1 of 5 categories according to the developmental stage of the parasite: (1) germinal; (2) predominantly blastocercariae; (3) mixed blastocercariae and metacercariae; (4) predominantly metacercariae and (5) all metacercariae (as given by Dybdahl & Lively, 1998).

Similar to the analysis of prevalence, we compared the development stage of the parasite in different host–parasite combinations using repeated measures ANOVA with each container as the repeated unit. The dependent variable was developmental stage of the infection. The between-subject effects were host and parasite and the within-subject effect was time. All uninfected snails were excluded from the analysis as were all snails in the control group. After finding a significant host by parasite interaction, we conducted independent contrasts to determine the host–parasite combinations in which development rates differed. For this analysis, we examined the first 3 sampling periods (sampling periods 1–3; 13, 19, and 24 weeks) to avoid singularity in the covariance matrix. The first 3 sampling periods encompass the period before development was complete (stage 5) in most host–parasite combinations (see Fig. 2).

## RESULTS

#### *Does total parasite prevalence differ between hosts from different sources?*

The results were consistent with a previous study that showed clinal variation in infection (Fig. 1). Parasites from shallow water were significantly more infective to snails from shallow-water than deep-water habitats ( $T = 8.2$ , D.F. = 5.9,  $P < 0.001$ , Fig. 1), but deep-water parasites were not better at infecting deep-water than shallow-water snails (Fig. 1). In fact, parasites from the deep water were significantly better at infecting shallow-water snails ( $T = 3.3$ , D.F. = 4.0,  $P = 0.030$ ). Significance levels for the planned comparisons were not adjusted because the contrasts were orthogonal (Sokal & Rohlf, 1995).

We found no effect of time among samples, and no interactions between time and host or parasite or between host, parasite and time (Table 1). Thus, parasite prevalence did not change during the experiment.

#### *Does host mortality differ between parasites from different sources?*

There was no indication of differential mortality according to treatment (Table 2). On average, 21.5 snails died per container, but the deaths were not associated with host source, parasite source, or the host-by-parasite interaction (Table 2). Thus, it is unlikely that the results that follow could have been

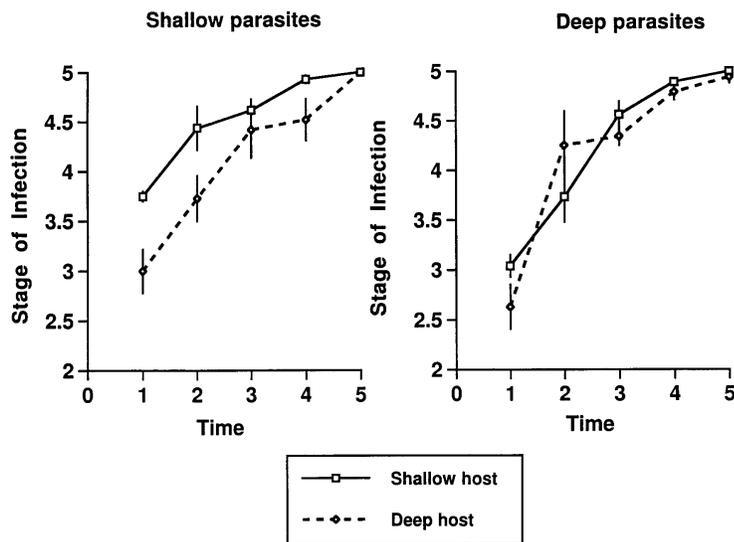


Fig. 2. Relationship between mean stage of infection (parasite development rate) and time in all host–parasite combinations. The vertical bars show 1 standard error of the mean.

Table 1. Repeated measures ANOVA analysis of prevalence of infection over time

(The dependent variable is prevalence of infection. Analysis was conducted on 3 samples only, time 1, time 3, and time 5 (beginning, middle, and end of sampling period).)

Source of variation	D.F.	MS	<i>F</i>	<i>P</i>
Within-subjects effects				
Time	2	109.19	1.80	0.187
Host*Time	2	38.57	0.64	0.538
Parasite*Time	2	33.94	0.56	0.578
Host*Parasite*Time	2	124.39	2.05	0.150
Error	24	60.58		
Between-subjects effects				
Host	1	9555.77	41.95	< 0.0005
Parasite	1	7897.07	34.66	< 0.0005
Host*Parasite	1	68.87	0.30	0.593
Error	12	227.81		

Table 2. Two-way ANOVA summary results for mortality

(The dependent variable is the number of dead snails.)

Source of variation	D.F.	MS	<i>F</i>	<i>P</i>
Host	1	18.37	0.34	0.564
Parasite	2	72.79	1.37	0.281
Host*Parasite	2	21.13	0.40	0.679
Error	18	53.32		

affected by differential host mortality among parasite sources.

#### *Does parasite developmental stage differ between hosts from different sources?*

Analysis of parasite development time revealed significant host and parasite effects and a significant host by parasite interaction (Table 3). The host by

parasite interaction reveals that parasite development rate differed between the two hosts. Independent contrasts revealed that shallow parasites develop more rapidly in shallow hosts than in deep hosts ( $T = 3.0$ , D.F. = 4.7,  $P = 0.034$ , Fig. 2) but deep parasites develop at the same rate in the 2 hosts ( $T = 0.44$ , D.F. = 5.1,  $P = 0.675$ , Fig. 2). Significance levels for the independent contrasts were not adjusted because the contrasts were orthogonal (Sokal & Rohlf, 1995).

Table 3. Repeated measures ANOVA analysis of development rate of parasites in shallow and deep hosts

(The dependent variable is mean development stage of infection. Analysis was conducted on 3 samples only, times 1–3, before development was complete (stage 5) in all host-parasite combinations (see Fig. 2).)

Source of variation	D.F.	MS	F	P
Within-subjects effects				
Time	2	7.95	42.63	< 0.0005
Host*Time	2	0.26	1.40	0.266
Parasite*Time	2	0.28	1.51	0.242
Host*Parasite*Time	2	0.42	2.23	0.129
Error	24	0.19		
Between-subjects effects				
Host	1	1.06	8.23	0.014
Parasite	1	0.66	5.13	0.043
Host*Parasite	1	0.80	6.18	0.029
Error	12	0.13		

## DISCUSSION

Using a reciprocal cross-infection experiment, we examined the infectivity of trematode parasites (*Microphallus* sp.) taken from shallow- and deep-water habitats on host snails (*Potamopyrgus antipodarum*) that were collected in the same habitats. We found that parasites from shallow water were significantly more infective to hosts from shallow water, which is consistent with a similar study conducted in the previous year (Lively & Jokela, 1996). Similarly, we found that parasites from deep water were also significantly more infective to host snails collected from shallow water, which is consistent with the non-significant trend observed in the previous study (Lively & Jokela, 1996). Hence the results are repeatable, and are consistent with other evidence (Lively & Jokela, 1996) for a cline in susceptibility such that susceptibility is highest in snails from shallow water and lowest in snails from deep water.

There are 2 possible explanations for this pattern of results. First, the results are consistent with the idea that gene flow in *Microphallus* occurs from the shore outward to deeper water. The mechanism for this kind of asymmetric gene flow could be based on the foraging patterns of the definitive host of *Microphallus* (Grey Ducks and Mallards). These dabbling ducks forage primarily in the shallow margins of the lake, but move to deep water to rest or escape danger. Hence, these ducks should be depositing parasite eggs over the deeper water even though the infections were gained in shallow water (see Fig. 2 of Lively & Jokela, 1996). If this idea is correct, then only parasites from shallow water should be locally adapted to their hosts, because it is only in the shallow water that the parasites are being recycled back into the same host population. This prediction assumes that snails remain in the habitat

where they were infected. This assumption is supported by previous studies of snails in this lake, which show that snails are genetically structured by habitat (Fox *et al.* 1996). Gene flow, and therefore movement, must be very limited for snails in shallow water to be genetically distinct from snails in deep water. These ideas suggest that one explanation for the results is coevolutionary dynamics between host and parasites that only occur in shallow water.

The second explanation for why parasites from both shallow and deep water are more infective to hosts from shallow water is that these hosts are generally more susceptible to infection by *Microphallus*, independent of coevolutionary effects. For example, host physiological condition may vary from shallow to deep water in such a way that shallow-water snails are simply more easily infected. Additionally, ploidy varies from shallow to deep, such that diploid snails are more common in shallow water and triploid snails more common in deep water (Fox *et al.* 1996). If diploid snails are inherently more susceptible, then hosts from shallow water may be, on average, more susceptible to parasite infection. However, in a previous study in this lake, we found no difference in the prevalence of infections between diploid and triploid snails in the mid-water and deep-water habitats (Jokela *et al.* 1997). Hence, if snails from shallow water are more susceptible to infection, it is probably not caused by differences in inherent susceptibility between diploid and triploid snails, but the hypothesis merits a direct experimental test.

On average, snails from deep water are larger than snails from shallow water. Because snail size may affect susceptibility, if size is related to age, differences in susceptibility in snails from different depths may be related to size. Several studies have shown that age alters susceptibility in snails; some show that older, larger snails are more susceptible to

trematode infection (reviewed by Lim & Heyneman, 1972; Sousa, 1983; Lauckner, 1986), whereas in others, younger, smaller snails are more susceptible to infection (reviewed by Lim & Heyneman, 1972; Anderson *et al.* 1982; Sousa, 1983; Anderson & Crombie, 1984; Lauckner, 1986, Niemann & Lewis, 1990). However, in most of these studies transmission to the snail occurs via an actively swimming miracidium. Thus differences in susceptibility at different ages are related to variation in the attractiveness of hosts to miracidia. Consequently, susceptibility is unlikely to be affected by age in *P. antipodarum* because *Microphallus* is transmitted by passive ingestion of an egg, rather than by miracidia.

We found no evidence that mortality of the hosts differed according to parasite source. There were no interactions between source of the snails and source of the parasite. Hence, under the conditions of this experiment, parasites from both shallow and deep water had equivalent effects on mortality in hosts from shallow and deep water. In contrast, we found that parasite developmental rates differed both between hosts and between parasites from different sources. On the first 2 times that the experiment was sampled, parasites from shallow water developed more rapidly in hosts from shallow water, yet parasites from deep water developed at the same rate in both hosts from shallow and deep water. These results are consistent with those of Lively & Jokela (1996) who found that parasites from different habitats developed at different rates.

Similar to the results for infectivity, the development rate results could be explained by local adaptation or factors independent of coevolutionary dynamics. Consistent with the results for infectivity, the results suggest that local adaptation is present only in parasites from shallow water; parasites from shallow water are 'matched' to their hosts from shallow water and parasites from deep water are not 'matched' to their hosts from deep water. Again, these results are also consistent with the idea that gene flow occurs from the shallow to the deep water of this lake. If gene flow is one-way, then strong selection for slower development must occur in the parasites that survive through a single generation in hosts from deep water. Alternatively, development rate of parasites may depend on the number of parasites that successfully infect the host. If many infect the snail host, then parasites should develop rapidly in order to compete most successfully for limited host resources. If few parasites infect a snail, then development does not have to occur rapidly for parasites to utilize many host resources. This idea assumes that the parasites can detect how many other parasites are present and respond to this cue. If this mechanism is correct, then rapid development of shallow parasites on shallow hosts occurs because the prevalence of infection, and therefore within host competition, is highest in this parasite–host com-

petition. A similar strategy has been shown to occur in the rodent malaria *Plasmodium chabaudii*; more transmission stages are produced when within host competition is high (by infections with different parasite genotypes) than when within host competition is lower (by infections from a single genotype; Taylor, Walliker & Read, (1997)).

In summary, parasites from shallow water were more infective to hosts from shallow water and developed more rapidly in these hosts. The converse was not true, parasites from deep water were not more infective to hosts from deep water and did not develop more rapidly in their local host. This implies that either coevolutionary interactions are more likely in the shallow water, which may explain the higher frequency of sexual individuals in shallow water, or that shallow snails are simply more susceptible to infection independent of coevolutionary interactions.

We thank K. Michelson for maintaining the snail cultures. We also thank M. Winterbourn, J. van Berkel, and J. McKenzie from the Zoology Department of the University of Canterbury for their continued support. This study was supported by The U.S. National Science Foundation (DEB 93177924).

#### REFERENCES

- AHMED, H. U., MUNDT, C. C. & COAKLEY, S. M. (1995). Host-pathogen relationship of geographically diverse isolates of *Septoria tritici* and wheat cultivars. *Plant Pathology* **44**, 838–847.
- ANDERSON, R. M. & CROMBIE, J. (1984). Experimental studies of age-prevalence curves for *Schistosoma mansoni* infections in populations of *Biomphalaria glabrata*. *Parasitology* **89**, 79–104.
- ANDERSON, R. M., MERCER, J. G., WILSON, R. A. & CARTER, N. P. (1982). Transmission of *Schistosoma mansoni* from man to snail: experimental studies of miracidial survival and infectivity in relation to larval age, water temperature, host size and host age. *Parasitology* **85**, 339–360.
- BALLABENI, P. & WARD, P. I. (1993). Local adaptation of the trematode *Diplostomum phoxini* to the European minnow *Phoxinus phoxinus*, its second intermediate host. *Functional Ecology* **7**, 84–90.
- BURDON, J. J. & THOMPSON, J. N. (1995). Changed patterns of resistance in a population of *Linum marginale* attacked by the rust pathogen *Melampsora lini*. *Journal of Ecology* **83**, 199–206.
- DYBDAHL, M. F. & LIVELY, C. M. (1995). Diverse, endemic and polyphyletic clones in mixed populations of the freshwater snail *Potamopyrgus antipodarum*. *Journal of Evolutionary Biology* **8**, 385–398.
- DYBDAHL, M. F. & LIVELY, C. M. (1996). The geography of coevolution: comparative population structures for a snail and its trematode parasite. *Evolution* **50**, 2264–2275.
- DYBDAHL, M. F. & LIVELY, C. M. (1998). Host-parasite coevolution: evidence for rare advantage and time-lagged selection in a natural population. *Evolution* **52**, 1057–1066.

- EBERT, D. (1994). Virulence and local adaptation of a horizontally transmitted parasite. *Science* **265**, 1084–1086.
- FOX, J. A., DYBDAHL, M. F., JOKELA, J. & LIVELY, C. M. (1996). Genetic structure of coexisting sexual and clonal subpopulations in a freshwater snail (*Potamopyrgus antipodarum*). *Evolution* **50**, 1541–1548.
- GANDON, S., CAPOWIEZ, Y., DUBOIS, Y., MICHALAKIS, Y. & OLIVIERI, I. (1996). Local adaptation and gene-for-gene coevolution in a metapopulation model. *Proceedings of the Royal Society of London, B* **263**, 1003–1009.
- HAMILTON, W. D., AXELROD, R. & TANESE, R. (1990). Sexual reproduction as an adaptation to resist parasites (A review). *Proceedings of the National Academy of Sciences, USA* **87**, 3566–3573.
- JOKELA, J. & LIVELY, C. M. (1995a). Parasites, sex and early reproduction in a mixed population of freshwater snails. *Evolution* **49**, 1268–1271.
- JOKELA, J. & LIVELY, C. M. (1995b). Spatial variation for infection by digenetic trematodes in a population of freshwater snails (*Potamopyrgus antipodarum*). *Oecologia* **103**, 509–517.
- JOKELA, J., LIVELY, C. M., FOX, J. A. & DYBDAHL, M. F. (1997). Flat reaction norms and “frozen” variation in clonal snails (*Potamopyrgus antipodarum*). *Evolution* **51**, 1120–1129.
- JUDSON, O. P. (1995). Preserving genes: a model of the maintenance of genetic variation in a metapopulation under frequency-dependent selection. *Genetical Research* **65**, 175–191.
- KALTZ, O., GANDON, S., MICHALAKIS, Y. & SHYKOFF, J. A. (1999). Local maladaptation in the anther-smut fungus *Microbotryum violaceum* to its host plant *Silene latifolia*: evidence from a cross-inoculation experiment. *Evolution* **53**, 395–407.
- KALTZ, O. & SHYKOFF, J. A. (1998). Local adaptation in host–parasite systems. *Heredity* **81**, 361–370.
- LADLE, R. J., JOHNSTONE, R. A. & JUDSON, O. P. (1993). Coevolutionary dynamics of sex in a metapopulation: escaping the Red Queen. *Proceedings of the Royal Society of London, B* **253**, 155–160.
- LAUCKNER, G. (1986). Ecological effects of larval trematode infestation on littoral marine invertebrate populations. *International Journal for Parasitology* **17**, 391–398.
- LIM, H.-K. & HEYNEMAN, D. (1972). Intramolluscan inter-trematode antagonism: a review of factors influencing the host-parasite system and its possible role in biological control. *Advances in Parasitology* **10**, 191–268.
- LIVELY, C. M. (1987). Evidence from a New Zealand snail for the maintenance of sex by parasitism. *Nature, London* **328**, 519–521.
- LIVELY, C. M. (1989). Adaptation by a parasitic trematode to local populations of its snail host. *Evolution* **43**, 1663–1671.
- LIVELY, C. M. (1999). Migration, virulence, and the geographic mosaic of adaptation by parasites. *American Naturalist* **153**, S34–S47.
- LIVELY, C. M. & DYBDAHL, M. F. (2000). Parasite adaptation to locally common host genotypes. *Nature, London* **405**, 679–681.
- LIVELY, C. M. & JOKELA, J. (1996). Clinal variation for local adaptation in a host-parasite interaction. *Proceedings of the Royal Society of London, B* **263**, 891–897.
- LIVELY, C. M. & MCKENZIE, J. (1991). Experimental infection of a freshwater snail, *Potamopyrgus antipodarum*, with a digenetic trematode *Microphallus* sp. *New Zealand Natural Sciences* **18**, 59–62.
- MORAND, S., MANNING, S. D. & WOOLHOUSE, M. E. J. (1996). Parasite-host coevolution and geographic patterns of parasite infectivity and host susceptibility. *Proceedings of the Royal Society of London, B* **263**, 119–128.
- NIEMANN, G. M. & LEWIS, F. A. (1990). *Schistosoma mansoni*: influence of *Biomphalaria glabrata* size on susceptibility to infection and resultant cercarial production. *Experimental Parasitology* **70**, 286–292.
- PARKER, M. A. (1985). Local population differentiation for compatibility in an annual legume and its host-specific pathogen. *Evolution* **39**, 713–723.
- PETERS, A. D. & LIVELY, C. M. (1999). The Red Queen and fluctuating epistasis: a population genetic analysis of antagonistic coevolution. *American Naturalist* **154**, 393–405.
- SOKAL, R. R. & ROHLF, F. J. (1995). *Biometry*, 3rd Edn. W. H. Freeman and Company, New York.
- SOUSA, W. P. (1983). Host life history and the effect of parasitic castration on growth: a field study of *Cerithidea californica* Haldeman (Gastropoda: Prosobranchia) and its trematode parasites. *Journal of Experimental Marine Biology and Ecology* **73**, 273–296.
- WEBSTER, J. P. & WOOLHOUSE, M. E. J. (1998). Selection and strain specificity of compatibility between snail intermediate hosts and their parasitic schistosomes. *Evolution* **52**, 1627–1634.
- TAYLOR, L. H., WALLIKER, D. & READ, A. F. (1997). Mixed-genotype infections of the rodent malaria *Plasmodium chabaudi* are more infectious to mosquitoes than single-genotype infections. *Parasitology* **115**, 121–132.