

Host manipulation by parasites and risk of non-host predation: is manipulation costly in an eye fluke–fish interaction?

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ABSTRACT

Question: Is host manipulation by a trophically transmitted parasite costly in terms of non-host predation?

Organisms: A trematode eye fluke (*Diplostomum spathaceum*), its rainbow trout (*Oncorhynchus mykiss*) intermediate host and pike (*Esox lucius*) non-host predator.

Background: The parasite has been shown to increase the vulnerability of fish to simulated avian predation by impairing fish vision. However, this may also predispose fish to non-host predators such as piscivorous fish, which would lead to transmission failure and could override the benefits of manipulation.

Methods: In a laboratory experiment, we predisposed pairs consisting of one infected and one uninfected rainbow trout to predation by pike, and recorded their preference between prey types.

Results: Infected and uninfected fish did not differ in their susceptibility to piscine predation.

Conclusions: Together with previous results, our findings suggest that manipulation of a fish host increases the probability of parasite transmission to bird hosts, and thus can be a parasite strategy evolved to enhance transmission.

Keywords: *Diplostomum spathaceum*, parasite–host interactions, transmission, Trematoda.

INTRODUCTION

Wide interest in the evolutionary ecology literature on parasite–host interactions has focused on parasite-induced host manipulation. Since completion of parasite life-cycles through successful transmission between hosts is usually an unlikely event (Dobson *et al.*, 1992), the ability of trophically transmitted parasites to manipulate the phenotype of infected hosts to increase their susceptibility to predation by target hosts (the next host in the

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life-cycle) may be favoured by selection (Rothschild, 1962; Holmes and Bethel, 1972). Parasite-induced changes in host phenotype have been described in several parasite–host interactions associated with trophic transmission (reviewed by Moore, 2002), and in those systems host manipulation has usually been considered as a parasite strategy, evolved to increase transmission efficiency. However, there may also be costs associated with manipulation (e.g. increased energy demand, increased risk of non-host predation), which decrease parasite fitness by counterbalancing or even outweighing the benefits of manipulation. Thus, the potential costs of manipulation need to be evaluated accurately when estimating its effect on parasite fitness. However, costs of manipulation have been considered only in very few parasite–host systems (Levri and Lively, 1996; Levri, 1998; Mouritsen and Poulin, 2003; Tompkins *et al.*, 2004), and thus knowledge of the adaptiveness of host manipulation is still limited.

One potential cost related to host manipulation is that infected individuals may also be predisposed to being caught by predators that are unsuitable hosts for the parasite. In extreme cases, non-host predation may even override the benefits of manipulation and reduce parasite fitness. For example, increased susceptibility to non-host predators has been reported in the *Curtuteria australis* (Trematoda)–cockle interaction. In this system, the parasite reduces the ability of the cockle to burrow into the substrate (Thomas and Poulin, 1998), which predisposes infected individuals to predation not only by bird definitive hosts, but also by whelk and fish non-host predators (Mouritsen and Poulin, 2003; Tompkins *et al.*, 2004). Non-host predation reduces the probability of completion of the parasite life-cycle, and thus the adaptive value of host manipulation in this system remains unclear (Tompkins *et al.*, 2004). Increased risk of non-host predation may also be common in other interactions where host manipulation occurs, since several non-trophically transmitted parasites are known to predispose infected hosts to predation, even though this always leads to death of the parasite (e.g. Hudson *et al.*, 1992; Murray *et al.*, 1997; Vance and Peckarsky, 1997; Steen *et al.*, 2002). Therefore, separating host susceptibility to different types of predators is essential when evaluating the effect of manipulation on parasite transmission probability.

In the present study, we examined intermediate host vulnerability to non-host predation as a cost of manipulation in the *Diplostomum spathaceum* (Trematoda) parasite–fish interaction. The parasite has a three-stage life-cycle with a bird definitive host, and snail and fish intermediate hosts (see Chappell *et al.*, 1994). Trophic transmission is involved in parasite transmission from fish to birds. Our earlier studies have shown that this parasite reduces the escape response (Seppälä *et al.*, 2004) and cypsis of fish (Seppälä *et al.*, 2005a), and increases their susceptibility to simulated avian predation (Seppälä *et al.*, 2004, 2005b). This suggests that host manipulation may be an adaptation of the parasite to enhance its transmission to bird hosts. Since *D. spathaceum* parasites lodge themselves in the lenses of the fish eyes and induce cataract formation (e.g. Rushton, 1937, 1938; Shariff *et al.*, 1980; Karvonen *et al.*, 2004), host manipulation in this system is probably caused mainly by the reduced vision of infected fish (Seppälä *et al.*, 2005b). Therefore, manipulation could also predispose fish to non-host predators such as piscivorous fish, possibly overriding the benefits of manipulation.

The aim of this study was to test whether *D. spathaceum* eye flukes increase the vulnerability of fish to piscine predators. We investigated this experimentally under laboratory conditions by comparing the susceptibility of infected fish and uninfected fish to predation by pike (*Esox lucius*). We demonstrate that the vulnerability of infected fish to predation by pike did not differ from that of controls. This suggests that the parasite may benefit from host manipulation and manipulation can be considered as a potential adaptation to enhance parasite transmission.

MATERIALS AND METHODS

Study animals and parasite exposure

The parasite *D. spathaceum* matures in the intestine of fish-eating birds (definitive host) such as gulls (Lariidae) and terns (Sternidae) (Chappell *et al.*, 1994) where it reproduces sexually. The eggs of the parasite are released in the water with the bird's faeces where they hatch into free-swimming miracidia. The miracidia infect aquatic snails (first intermediate host) where the parasite reproduces asexually to produce free-swimming cercariae. Cercariae infect a range of fish species (second intermediate host) (Valtonen and Gibson, 1997) by penetrating the gills and skin. In fish, parasites migrate to the eye lenses where they develop into metacercariae (Chappell *et al.*, 1994). For successful transmission to the definitive host, the infected fish has to be eaten by a fish-eating bird. In the lenses of fish, parasites induce cataract formation (Rushton, 1937, 1938; Shariff *et al.*, 1980; Karvonen *et al.*, 2004), which increases the susceptibility of fish to predation after metacercariae have completed their development and become infective to birds (Seppälä *et al.*, 2005b).

For the experiment, we used laboratory-infected juvenile rainbow trout (*Oncorhynchus mykiss*). From the variety of fish host species suitable for the parasite, we selected rainbow trout because it is relatively susceptible to infection (Betterton, 1974) as well as being suitable for laboratory experiments. Fish were obtained from a commercial fish farm where they had been reared in indoor tanks supplied with ground water, which ensured that they had no eye flukes or other helminth parasites. We recognize that the behaviour of farmed fish may not be exactly the same as that of free-living fish. However, wild fish are commonly infected with several parasite species, which favours the use of fish farmed in ground water. Eight months before the experiment, we infected randomly chosen fish with *D. spathaceum* cercariae under laboratory conditions at a water temperature of 18.0°C. Since complete development of metacercariae usually takes 1–2 months depending on water temperature (see Sweeting, 1974), this procedure ensured that the parasites were fully developed by the time of the experiment and capable of manipulating fish (Seppälä *et al.*, 2005b). Cercariae were released by 10 naturally infected *Lymnaea peregra* snails. We pooled all cercariae into one suspension, and estimated the cercarial density from twenty 1-ml samples. Fish were exposed to the parasites concurrently in six tanks each containing 300 fish in 250 litres of water. Three randomly chosen tanks received an infection dose of 100 cercariae per fish. The purpose of this exposure procedure was to produce fish with infection intensities [intensity indicates the number of parasites in an infected host (Bush *et al.*, 1997)] high enough to induce possible effects but still corresponding to natural intensities (see Valtonen and Gibson, 1997; Marcogliese *et al.*, 2001). Fish in the remaining three tanks were sham exposed with water and retained as uninfected control fish. During the exposures, water flow through the tanks was turned off, and the tanks were aerated with aquarium pumps. After 30 min of exposure, the water flow was turned on, and the water volume in each tank was increased to 1385 litres. Fish were maintained under these conditions until the experiment and fed daily with commercial fish pellets. During the maintenance, water temperature was decreased to 4–5°C for the winter season. More fish were infected than used in the experiment described below, because fish were also used in studies describing impaired crypsis (Seppälä *et al.*, 2005a) and increased susceptibility to simulated avian predation (Seppälä *et al.*, 2005b).

Pike was used as the predator because of its predation efficiency under laboratory conditions and its common occurrence in Finland. Pike regularly feed on several fish species,

including salmonids, which leads to death of eye flukes in prey fish. Eight pike (body length 35–45 cm) were caught with a lure from the wild 8 months before the experiment. They were maintained in two tanks in the laboratory each containing 920 litres of water, and fed with live rainbow trout, roach (*Rutilus rutilus*), and perch (*Perca fluviatilis*) of similar size as the study fish used in the experiment.

Experimental design

The predation experiment was conducted in four tanks (190×190 cm, water depth 30 cm; Fig. 1). To produce shelter for fish, we stood eight bricks ($26 \times 12.5 \times 5.5$ cm) on their long sides close to the edges of each tank (Fig. 1). The tanks were illuminated from above with fluorescent lamps (True Light 36 W) placed 280 cm above the water surface. Light intensity was set to 200 lux measured from the water surface and the lighting was adjusted so that all parts of the tanks received similar light intensity. A light–dark cycle of 14 h : 10 h was used and all predation trials (see below) were conducted during the lit hours. Water flow through the tanks was set to $10 \text{ litres} \cdot \text{min}^{-1}$. During the experiment, water temperature ranged between 13.2 and 17.1°C, corresponding to the natural temperature fluctuations at the time of the experiment.

The eight pike were randomly divided into two groups of four individuals, which were used in turn in the experiment. At each round, four pike were placed randomly into the experimental tanks, one in each, and allowed to recover from the transfer for 3 days before the experiment. In each trial, one infected and one uninfected rainbow trout were randomly selected and transferred to each tank. Rainbow trout were placed into a round cage (diameter 65 cm, height 50 cm) in the middle of the tank (Fig. 1) and allowed to recover from the transfer for 12 h before the experiment. Pike were allowed to select their location freely. In the experiment, rainbow trout were released by lifting the cage up slowly without disturbing them, and the pike was allowed to eat one of the two fish during the next 12 h. The same set of pike was used in two trials on consecutive days before replacing them with

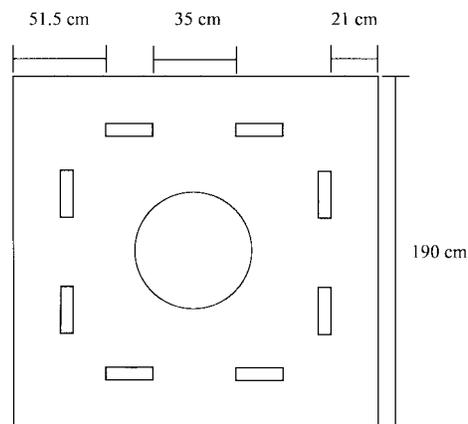


Fig. 1. Overhead schematic view of the experimental arena used in the experiment. Rectangles indicate the locations of the bricks and the circle denotes the position in the cage where the pair of rainbow trout was allowed to settle before the experiment. The pike was allowed to select its location freely.

another group. This was done to reduce stress caused by transfers of fish between tanks. A total of 96 trials were conducted.

After the experiment, the remaining rainbow trout were removed from the tanks and killed with an overdose of 0.01% MS 222 fish anaesthetic (Sigma Chemical Co., St Louis, USA). We measured the coverage of parasite-induced cataracts from the lenses of each fish with a Kowa Portable Slit Lamp SL-14 microscope (see Wall and Bjerkås, 1999; Karvonen *et al.*, 2004) using a subjective scale: 0 = no cataracts, 1 = cataracts covering less than 25%, 2 = cataracts covering 25–50%, 3 = cataracts covering 50–75%, 4 = cataracts covering 75–100%, 5 = cataracts covering 100% of the lens area. The thickness of the cataracts was not considered. We counted the *D. spathaceum* metacercariae by dissecting the lenses, and measured the length (± 1 mm) and mass (± 0.1 g) of each fish. Since the intensity of infection, cataract coverage, and body size could not be measured from rainbow trout that were eaten in the experiment, we estimated these parameters from random samples of 50 infected and 50 control fish taken from the storage tanks during the experiment. We killed the fish and studied them as mentioned above. The experiment was conducted with the permission of the Lab-Animal Care and Use Committee of the University of Jyväskylä.

RESULTS

In 74 of 96 trials, the pike ate one of the two rainbow trout. In the rest of the trials, they did not eat in 12 h. Infected and control fish did not differ in their susceptibility to predation (38 infected and 36 control fish eaten; Sign test, $P = 0.908$). The test was able to detect a relative difference of 24% in the susceptibility of fish to predation. Preference of pike for infected and uninfected fish did not differ between the first and second trial conducted in a single round of the experiment (only the cases where pike ate in both trials are included; McNemar test, $P = 0.616$, $n = 32$). All sampled fish that were exposed to the parasite were infected and the mean (\pm standard error) intensity of *D. spathaceum* was 65.3 ± 2.3 , which caused parasite-induced cataract formation in the lenses of fish. Cataract coverage in infected fish varied from less than 25% to 100% (median 25–50%). Control fish were free of eye flukes and no cataracts were observed. The distribution of infection intensity and cataract coverage did not differ between the random sample of infected fish and infected fish that were not eaten in the experiment [Kolmogorov-Smirnov test: intensity of infection, $Z = 0.463$, $P = 0.983$, $n_1 = 50$, $n_2 = 36$ (Fig. 2a); cataract coverage, $Z = 0.503$, $P = 0.962$, $n_1 = 50$, $n_2 = 36$ (Fig. 2b)]. The mean (\pm standard error) body length and mass of the random sample of infected fish were 157.7 ± 2.1 mm and 36.3 ± 1.5 g respectively. Corresponding values for control fish were 156.3 ± 1.3 mm and 35.1 ± 0.9 g respectively, which did not differ from the infected fish (independent samples *t*-test: length, $t_{98} = -0.589$, $P = 0.557$; mass, $t_{98} = -0.676$, $P = 0.501$). Body length and mass did not differ between the random sample of study fish (both infected and uninfected) and fish that were not eaten in the experiment (sample fish: 157.0 ± 1.2 mm and 35.7 ± 0.9 g; study fish: 155.4 ± 1.8 mm and 35.7 ± 1.1 g; independent samples *t*-test: length, $t_{172} = -0.795$, $P = 0.428$; mass, $t_{172} = -0.019$, $P = 0.985$).

DISCUSSION

The ability of trophically transmitted parasites to manipulate the behaviour or other phenotypic traits of their hosts has usually been considered an adaptation to enhance parasite transmission. Several studies have shown increased susceptibility of infected hosts

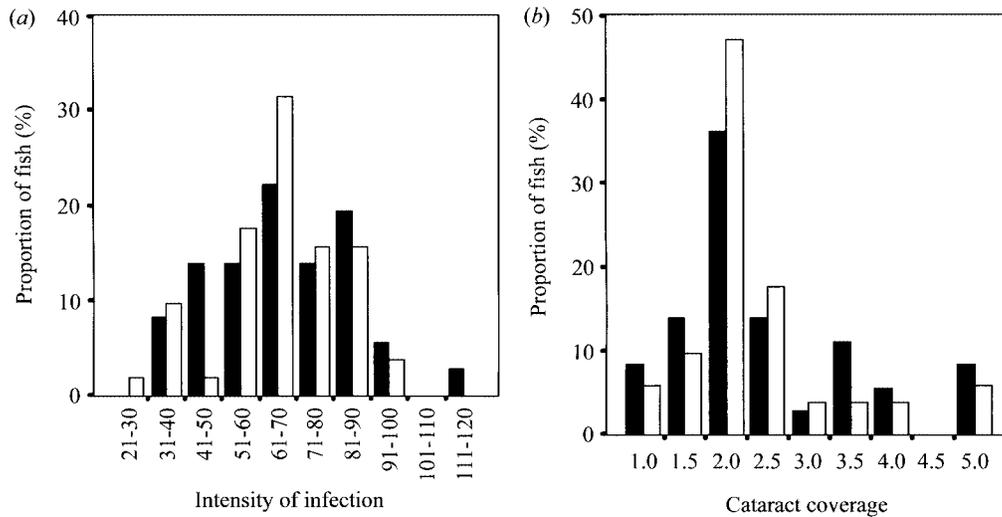


Fig. 2. Proportional distribution of (a) intensity of infection and (b) cataract coverage for the random sample of infected fish (□) and infected fish that were not eaten in the experiment (■). Cataract coverage is presented as a mean value for both lenses of fish determined using the scale: 0 = no cataracts, 1 = cataracts covering less than 25%, 2 = cataracts covering 25–50%, 3 = cataracts covering 50–75%, 4 = cataracts covering 75–100%, and 5 = cataracts covering 100% of the lens area.

to predation by target hosts subsequent to parasite-induced alterations in their phenotype (reviewed by Moore, 2002). However, manipulation may also include costs for parasites, such as an increased energy demand (Poulin, 1994) or vulnerability to being caught by non-host predator species (Mouritsen and Poulin, 2003; Tompkins *et al.*, 2004). In this study, we examined non-host predation as a cost of manipulation in an eye fluke–fish interaction, where the parasite *D. spathaceum* impairs the vision of fish by inducing cataract formation (e.g. Rushton, 1937, 1938; Shariff *et al.*, 1980; Karvonen *et al.*, 2004). This reduces the fish escape response (Seppälä *et al.*, 2004) and crypsis (Seppälä *et al.*, 2005a), and increases their susceptibility to simulated avian predation (Seppälä *et al.*, 2004, 2005b). Earlier, Brassard *et al.* (1982) had shown that injuries caused by penetration of *D. spathaceum* cercariae into the fish increase their vulnerability to piscine predation. This, however, is not related to parasite transmission, because at this stage parasites are not yet infective to birds. When we exposed uninfected fish and fish carrying fully developed metacercariae to predation by pike, we found no difference in their susceptibility to piscine predation. Furthermore, infection intensity and cataract coverage of the infected fish that were not eaten in the experiment did not differ from those of the random sample, indicating that neither the number of parasites nor cataract coverage affected the results. These results, together with our previous findings (Seppälä *et al.*, 2004, 2005b), suggest that in this particular parasite–host interaction, host manipulation may enhance parasite transmission to the target hosts by predisposing infected fish to bird predators but not to non-host species. Thus, the ability of *D. spathaceum* eye flukes to manipulate fish can be considered as a potential adaptation to increase the probability of successful transmission.

Nevertheless, the experiment was conducted under laboratory conditions and it could be speculated that the conditions may have affected the behaviour of the prey or the predators. It is possible, for example, that fish are able to detect pike from several metres in nature and

the size of the experimental arenas may have reduced their chances of avoiding predation. This, however, appears unlikely because fish avoided pike mainly by sheltering next to the bricks and by escaping when an attack from a pike took place. Furthermore, in addition to predation by pike, fish may also be exposed to other non-host predator species in the wild. These include other piscivorous fish and mammals, which might differ from pike regarding their predatory behaviour. Thus, it is possible that manipulation predisposes fish to other non-host predator species than pike. However, in this study we focused on pike as it typically is the most common predatory fish species in Finnish water bodies.

Why does manipulation not also predispose fish to piscine predators? Since manipulation by *D. spathaceum* parasites is probably caused by impaired vision of fish (Seppälä *et al.*, 2005b), the ability of infected fish to avoid predators could be decreased in general, not just in the case of aerial predators. In our earlier studies (Seppälä *et al.*, 2004, 2005b), we used simulated avian predation to imitate predation by gulls (Laridae) and terns (Sternidae), which attack from the air. In this case, successful predator avoidance probably depends on visual detection of an approaching predator. However, when avoiding underwater attacks, fish are able to use other senses such as the lateral line and olfaction to detect predators, and thus the role of vision may be reduced. Furthermore, in several parasite–host relationships, the activity of infected hosts is changed compared with that of uninfected individuals (e.g. Hurd and Fogo, 1991; Maynard *et al.*, 1998; McCarthy *et al.*, 2000; Pulkkinen *et al.*, 2000). This may also be true in the present system, where impairment in fish vision may, for example, reduce the activity and movement of fish leading to lower contact probability with an ambush-type predator such as pike. Further studies are needed to examine which factors lead to specificity of host manipulation in this system.

Based on our studies with the present system (Seppälä *et al.*, 2004, 2005b, this study), we suggest that manipulation predisposes fish only to avian predation. Thus, the ability of *D. spathaceum* parasites to manipulate fish phenotype may enhance their transmission to the target hosts. In other systems, infected hosts have been reported to also be more vulnerable to being caught by non-host predators (Mouritsen and Poulin, 2003; Tompkins *et al.*, 2004), which may override the benefits of manipulation. This, however, does not necessarily mean that manipulation cannot be favoured by natural selection also in these systems (Cézilly and Perrot-Minnot, 2005). Even if the increase in host vulnerability to non-host predators is higher in relation to target hosts, it is still possible that manipulation has evolved to increase the probability of parasite transmission. This depends on the likelihood of successful parasite transmission in the absence of manipulative effort. Thus, quantitative comparisons with varying manipulative effort are needed to estimate the net fitness outcome of host manipulation (see also Poulin *et al.*, 2005). These studies are especially important because it is possible that in many interactions associated with host manipulation, dead-end predators kill most parasite individuals, but parasite transmission to the target hosts is still enhanced through manipulation.

ACKNOWLEDGEMENTS

We thank V. Kupari and E. Tawast for assistance in the laboratory. We are grateful to D. Benesh, J. Jokela, and R. Poulin for their helpful comments on the manuscript. Konnevesi Research Station provided the facilities for the experiment. The study was funded by the SUNARE research programme of the Academy of Finland, the Biological Interactions graduate school, and the Finnish Cultural Foundation.

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