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# Role of water temperature on the size, migration activity and pathogenity of *Diphyllobothrium dendriticum* (Cestoda) plerocercoids in brown trout *Salmo trutta* m. *lacustris* (L.)

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The size, migration activity and pathogenity of *Diphyllobothrium dendriticum* (Cestoda) plerocercoids in the intermediate host were studied experimentally in age 1+ brown trout *Salmo trutta* m. *lacustris* (L.). In the first experiment (A) which lasted 12 weeks, two water temperatures were used: heated water at about 15°C and non-heated at about 10°C. In the second experiment (B) which lasted 7 weeks, the water temperature was raised gradually to 24–26°C. Increased water temperature did not promote *D. dendriticum*-induced mortality in either of the experimental conditions. Experiment A revealed, however, that the worms were longer and migrated more actively outside the body cavity of fish in heated aquaria.

## 1. Introduction

In Europe and North America, *Diphyllobothrium dendriticum* (Nitzsch 1824) (syn. *D. norwegicum* Vik) (Cestoda) is a widespread and common tapeworm occurring with varying prevalences and intensities on stomachs and visceral organs of fish, such as salmonids, coregonids and three-spined stickleback (*Gasterosteus aculeatus* L.) (e.g., Halvorsen 1970, Henricson 1977, 1978, Kennedy 1978, Ching 1988, Andersen & Valtonen 1992). Heavy infections resulting in chronic granulomatous peritonitis were reported in some northern lakes (Vik 1957, Bylund 1966, Curtis 1983, Rahkonen & Koski 1997), and *D. dendriticum* (or species closely resembling *D. dendriticum*) was found to cause mortality of salmonids in fish farms (Hoffman & Dunbar 1961, Rahkonen *et al.* 1996). Moreover, reports by Duguid and Sheppard (1944), Hickey and Harris (1947), Fraser (1960), and Bylund (1972) suggested that *D. dendriticum* may induce a host mortality also in a natural environment in summer. *D. dendriticum* killed fish in experimantal infections by Kuhlow (1953), and Bylund (1972) but not by Rahkonen and Valtonen (1997).

Salmonid hosts were shown to react to a

*D. dendriticum* infection with an inflammatory response resulting in encapsulation of the worms within about two months (e.g., Sharp *et al*. 1989, Sharp *et al*. 1992, Rahkonen & Valtonen 1997). This reaction is essential for a fish host since freely migrating larvae were found to cause considerable mortality in hatchery-reared trout (*Salmo trutta* L.) (Rahkonen *et al*. 1996). An indication of temperature-related pathogenity was obtained since a clearly higher mortality as well as water temperature were observed in summer 1991 as opposed to 1992 (Rahkonen *et al*. 1996). Hickey and Harris (1947) indicated that free plerocercoids placed in physiological saline were non-motile at 10°C, sluggish at 12°C, and active at 14°C. The reported mortalities in a natural environment and in farms (see references above) caused by *D. dendriticum* occurred only in summer when water temperature exceeds 15°C, even in northernmost habitats.

Experimental studies of the effect of water temperature on the migration pattern and size of plerocercoids as well as *D. dendriticum*-induced mortality in fish are lacking. However, this kind of information could help us to evaluate the effect of *D. dendriticum* on wild fish populations. Our prediction, based on previous observations (*see* Duguid & Sheppard 1944, Hickey & Harris 1947, Hoffman & Dunbar 1961, Bylund 1972, Rahkonen *et al*. 1996), is that temperature has an effect on *D. dendriticum*-induced mortality. However, we were interested whether this is due to a larger size and a greater activity of plerocercoids in fish in warm water.

The objectives of our experiments were (1) to examine the size and site of *D. dendriticum* plerocercoids introduced as procercoids into stomachs of brown trout (*Salmo trutta* m. *lacustris* (L.)), and the effect of the infection on the mortality of the fish at two water temperatures (10°C and 15°C) (experiment A), and (2) to study *D. dendriticum*-induced mortality in brown trout subjected to heat stress (experiment B).

## 2. Materials and methods

Infective procercoids of *D. dendriticum* were obtained using the method described by Rahkonen and Valtonen (1997). In brief, plerocercoids originated from brown trouts from Lake Inari, northern Finland, had been introduced into the stomachs of golden hamsters (*Mesocricetus auratus*). Eleven days later the hamsters were killed, and mature worms extracted from their intestines. Eggs removed from the worms were incubated in dark, aerated bottles at room temperature for 10 days, and then stored in the dark at 4°C. Hatching took place immediately when eggs were exposed to light. Hatched coracidia were poured into an aquarium containing a laboratory culture of copepodite stages of *Cyclops strenuus*.

An infected copepod culture was kept at 14–15°C. In experiments A and B respectively, a total of 112 and 60 copepods were examined 17 and 24 days post infection, and about 60 and 70% of them were found to be infected with 1–4 well-developed procercoids. A few days later, doses of 15 (exp. A) and 25 (exp. B) copepods, representing about 8 and 18–20 procercoids, respectively, were diluted in a drop of 0.3% (weight/volume) pepsin in physiological saline (0.9%), pH 2, and introduced by means of intubation into the stomachs of anaesthetised (MS-222) brown trout (exposed fish). Unexposed, control fish received only a drop of 0.3% pepsin/saline solution. The age 1+ (mean weight about 8 g) brown trout used in the experiments originated from a fish farm in northern Finland (Lake Inari stock). All fish were bathed in 1:4000 formalin for 20 min. four days before stomach intubation to avoid protozoan infections during the experiment.

### 2.1. Experiment A

The experiment was carried out between 16 October 1996 and 8 January 1997. Twelve plastic 60-l aquaria receiving active carbon-filtered tap water were arranged in two rows of six each. The water in the aquaria was aerated, and in every second aquarium it was possible to heat the inflowing water. Each of the aquaria contained 17 fish fed with commercial pellets once a day. The experimental design included four aquaria with heated, and four with non-heated water all containing exposed fish. Additionally, unexposed, control fish were placed in two randomly chosen aquaria with heated and two with non-heated water to monitor possible mortality without *D. dendriticum*.

The water temperature was 11.5°C on the day of infection. The temperature was raised to approximately 15°C in every second aquarium on the following day, and varied between 14–15°C during the 11-week maintenance. In the last week (12 weeks post infection), it was necessary to drop the temperature to about 13.5°C because of the problems with oxygen oversaturation. Water temperature in the non-heated aquaria decreased slowly from 11.5°C to 10°C during the first month post infection, to 9°C two months post infection, and to about 7.5°C by the end of the experiment (12 weeks post infection) as an effect of the natural decrease in the temperature of tap water in winter.

Oxygen, pH and electrical conductivity were measured once a week in every aquarium. The oxygen saturation level in the heated aquaria was usually about 100%, but during the fourth and fifth weeks it increased up to 104% producing the symptoms of the gas-bubble disease. After building a more effective aeration system, the saturation level was

maintained at about 100%. In non-heated aquaria, the oxygen saturation level varied between 97 and 101%. The pH was 7.6–7.8, and the conductivity about 175–190 µS $cm^{-1}$ throughout the experiment.

The fish that died ($n$ = 17) during the experiment were studied fresh. All the inner organs were compressed between glass plates (8 × 15 cm), and examined under 10× magnification in transmitted light. Skin mucus was examined (100× magnification) for protozoans to exclude them as a cause of mortality. At the end of the 12-week experiment, the alive fish ($n$ = 187) were killed, measured, weighed, dissected and examined for *D. dendriticum* plerocercoids. The heart and liver were compressed between glass plates, and examined at 10–20× magnification in transmitted light. The pericardium was studied with the aid of tweezers. The intestine and other inner organs were placed individually in 0.5% (w/v) pepsin in physiological saline (0.9%), pH 2 for at least three hours, and the rest of the fish remains in another bowl in a similar solution for at least five hours. The solution was sieved and studied at 10–20× magnification in transmitted light. The worms found were relaxed in tap water in a refrigerator overnight and measured.

### 2.2. Experiment B

The experiment was carried out between 7 February and 28 March 1997. Altogether 60 exposed and 60 unexposed brown trout were placed in eight flow-through 60-l plastic aquaria, 15 fish in each, receiving active-carbon filtered tap water. The aquaria were arranged in two rows, 4 in each, alternating between exposed and unexposed. The water in every aquarium was aerated, and the fish were fed daily with commercial pellets. However, in the last week when the water temperature was 24°C and above, the fish did not eat anymore and feeding was stopped.

The water temperature was 11–12°C for 3 weeks post infection, and after that it was raised as follows: 4th week to 15–16°C, 5th week to 16–17°C, 6th week to 20–21°C, and 7th week to 24–26°C

The experiment was terminated 7 weeks post infection. The oxygen saturation level was monitored throughout the experiment. It was 90–100% at temperatures up to 16°C, 80–90% at temperatures 16–21°C and 70–80% at 24–26°C.

The method of studying the fish was the same as in experiment A, except that after the examination of the heart and pericardium, the whole fish with a loosened intestine was placed in the pepsin solution in a bowl for about 5 hours. Therefore, in this experiment the proportions of plerocercoids inside the body cavity and muscles could not be separated.

### 2.3. Calculations

The SYSTAT statistical software (SYSTAT 1996) was used to analyse the results. Frequency data were tested with the $G^2$-test of independence, while mean intensity (worms per infected fish) and abundance (worms per fish studied) data were tested with the non-parametric Kruskall-Wallis test. A nested ANOVA model (fish nested within aquaria) was used for the size data of worms. For the ANOVA model, the normality of the error residuals was tested with Lilliefors' test, and the homogeneity of variances with Cochran's test (Day & Quinn 1989).

## 3. Results

### 3.1. Experiment A

The infection level was almost equal among the exposed fish kept in the four heated and four non-heated aquaria (Table 1). *D. dendriticum* larvae

Table 1. Prevalence (%) and mean intensity of *D. dendriticum* in 1+ brown trout exposed to 8 procercoids in heated and non-heated aquaria (heated 14–15°C, non-heated 11 to 7.5°C) (Experiment A).

| | No. of fish | Preval. % | Intensity of infection | | |
|---|---|---|---|---|---|
| | | | mean | SD | min.–max. |
| Heated aquaria | | | | | |
| 1 | 17 | 41 | 2.4 | 1.5 | 1–5 |
| 2 | 17 | 65 | 1.6 | 1.0 | 1–4 |
| 3 | 17 | 35 | 2.0 | 1.1 | 1–4 |
| 4 | 17 | 53 | 2.9 | 1.9 | 1–6 |
| Total | 68 | 49 | 2.2 | 1.4 | 1–6 |
| Non-heated aquaria | | | | | |
| 1 | 17 | 53 | 2.1 | 1.6 | 1–6 |
| 2 | 17 | 29 | 2.4 | 1.3 | 1–4 |
| 3 | 17 | 53 | 2.6 | 1.6 | 1–5 |
| 4 | 17 | 53 | 2.1 | 1.3 | 1–4 |
| Total | 68 | 47 | 2.3 | 1.5 | 1–6 |

were found in roughly every second fish with approximately two (1–6) worms per infected fish. There were no statistically significant differences in the mean intensities or abundances among the four heated, as well as among the four non-heated aquaria (Kruskal-Wallis test, $p > 0.05$). None of the unexposed fish were found to be infected with *Diphyllobothrium* species.

Most of the *D. dendriticum* plerocercoids were in capsules around the digestive tract. However, 1–2 worms had migrated to the heart area (the atrium and/or pericardium) in 8.8% and 7.4% of the exposed fish in aquaria with heated and non-heated water, respectively. Plerocercoids occurred inside the heart atrium in 2.9% of the fish at both temperature levels.

At the end of the experiment, when the migration pattern included all larvae found (dead fish not included), the proportion of plerocercoids found in the heart region (pericardium and atrium) was somewhat greater in the fish in heated (15.5%) as compared to the fish in non-heated water (7.2%), but the difference was not statistically significant ($G^2 = 2.28$, d.f. = 1, $p > 0.05$) (Table 2). However, when larvae outside the body cavity (in heart atrium, pericardium and muscles) were considered, the proportion was statistically significantly greater in the fish in heated (31%) as compared to the fish in non-heated water (14.3%) ($G^2 = 5.218$, d.f. = 1, $p = 0.022$) (Table 2).

After 12 weeks post infection, the worms in the fish of the aquaria with heated water were significantly longer than these in the fish of the aquaria with non-heated water, 22 mm ($n = 52$, SD = 7.0) and 10 mm ($n = 66$, SD = 3.4), respectively (nested ANOVA for log-transformed data, $F_{(1,6)} = 226.915$, $p < 0.001$).

Table 2. Distribution of *D. dendriticum* plerocercoids in different organs in 1+ brown trout at two temperature levels after 12 weeks post infection (heated 14–15°C, non-heated 11 to 7.5°C) (experiment A). A = intestines, B = liver, C = muscles, D = pericardium, E = heart atrium.

| | No. of pleroc. | Proportion (%) at different sites A*) | B | C | D | E |
|---|---|---|---|---|---|---|
| Heated | 58 | 63.8 | 5.2 | 15.5 | 12.1 | 3.4 |
| Non-heated | 70 | 71.4 | 14.3 | 7.1 | 4.3 | 2.9 |

*) including gonads and swim-bladder

Seventeen fish (8.3%) died in the experiment. Heating of the water promoted mortality resulting in the death of 8 exposed and 7 unexposed fish, as compared to two exposed fish from aquaria with non-heated water. Mortality was associated with either a shock reaction caused by the rapid warming of the water two days post infection (4 fish), the gas-bubble disease within 16–20 days post infection (12 fish) or the *Flavobacterium* infection 11 weeks post infection (1 fish). The *D. dendriticum* larvae occurred in 7 out of 10 exposed fish found dead (with 1 to 6 larvae), but because the *D. dendriticum* infection was not the primary cause of the death, no statistical analyses were done. Plerocercoids were not found in the heart region of the dead fish.

### 3.2. Experiment B

The total prevalence of the *D. dendriticum* infection in the four aquaria with exposed fish was 70.9% with a mean number of 2.7 plerocercoids per infected fish (Table 3). The number of plerocercoids in the infected fish ranged from 1 to 15, and showed an aggregated distribution (variance/mean = 3.0). There was no statistically significant variation in prevalences ($G^2 = 2.2895$, d.f. = 3, $p > 0.05$), and mean intensities (Kruskal-Wallis test = 4.491, d.f. = 3, $p > 0.05$) among the exposed fish (Table 3). The mean length of all measured plerocercoids ($n = 104$) was 10.7 mm (SD = 4.1).

The worms around the alimentary canal were found to be tightly encapsulated. However, 20% of the exposed brown trout harboured *D. dendriticum* larvae in their heart region (the atrium and/or pericardium), while 7.5% had one plerocercoid inside a thin capsule in the heart atrium.

Altogether four unexposed control fish in two aquaria were found to be naturally infected with 1, 1, 2 and 6 encapsulated *D. dendriticum* plerocercoids, respectively, attached to their visceral organs. Seven fish were excluded from further analysis; two unexposed fish (one per aquarium) died on the day of infection, and 5 exposed fish escaped from one aquarium.

Mortality did not occur in any of the aquaria during the experiment. However, the fish showed symptoms of heat stress; they lost their appetite, were apathetic and gasped for air. Both the ex-

posed and unexposed fish demonstrated a good external condition.

## 4. Discussion

Unlike the expectations based on previous observations (Duguid & Sheppard 1944, Hickey & Harris 1947, Kuhlow 1953, Bylund 1972, Rahkonen *et al*. 1996), the *D. dendriticum*-induced mortality was not found in either of the present experiments. In experiment A, the gas-bubble disease was clearly the main cause of the deaths in both exposed and unexposed fish, and in experiment B, heat stress did not promote mortalities of the infected fish.

The optimum temperature range for brown trout was demonstrated to be 4–19°C, and the upper critical range 19–30°C (Elliot 1981), so both temperature levels in experiment A were within the optimum range. As the temperature increased into the upper critical range in experiment B, the stress responses followed the phases described by Elliot (1981). If the infected fish had been more susceptible to heat stress this should have emerged during the last couple of weeks of this experiment. After the termination of experiment B, the water temperature was raised for few days to 27–28°C, which is not possible in natural environment in northern areas. This rapidly resulted in total losses, particularly in the control aquaria, indicating that the upper lethal temperature level was reached. No evidence of an increased mortality of the infected fish was observed even in these extreme conditions.

The low pathogenity of *D. dendriticum* demonstrated in the experiments (*see* also Rahkonen & Valtonen 1997) is somewhat surprising, however, since every trout (2–3 cm in length) harbouring one to three plerocercoids succumbed within one month in the experiment by Kuhlow (1953). Unfortunately, the temperature of Kuhlow's experiment was not given. Moreover, Bylund (1969, 1972) found a high mortality in sea trout (6–10 cm in length) at 10–15°C. Nearly every (13/14) infected fish died, most within two months. In the experiment by Bylund (1972), however, the mean number of plerocercoids per infected fish was as high as 23 (range 2–77). In our experiment B, the two fish with the highest number of plerocercoids, 11 and 15 (one in the pericardium), survived until the end of the experiment, although the fish were about the same size as those in the experiment by Bylund (1972).

On the other hand, temperature had an effect on the size and migration activity of the plerocercoids; they were longer, and a greater proportion of them migrated outside the body cavity (to the heart region, and muscles) in fish in the aquaria with heated water in experiment A. In addition, the mean length of the worms in experiment B was about the same after approximately 8 weeks as in fish in the aquaria with non-heated water (exp. A) after 12 weeks. The activity of *D. dendriticum* plerocercoids was shown to be temperature dependent by Hickey and Harris (1947) as well. It could be assumed that the damage to the host tissues will increase with the increasing sizes and movement intensity of plerocercoids. As a consequence, the worms could be expected, for

Table 3. Prevalence (%) and mean intensity of *D. dendriticum* in 1+ brown trout exposed to 18–20 procercoids (Experiment B).

| | No. of fish | Preval. % | Intensity of infection | | |
|---|---|---|---|---|---|
| | | | mean | SD | min.–max. |
| Aquarium | | | | | |
| 1 | 15 | 86.7 | 3.2 | 2.9 | 1–11 |
| 2 | 10* | 60.0 | 1.5 | 0.8 | 1– 3 |
| 3 | 15 | 66.7 | 2.8 | 4.4 | 1–15 |
| 4 | 15 | 66.7 | 2.8 | 1.6 | 1– 6 |
| Total | 55 | 70.9 | 2.7 | 2.9 | 1–15 |

* The 5 fish that escaped were excluded from the further study.

example, to cause *D. dendriticum*-induced mortality in heated water in experiment A, where the worms were twice as long as in the fish kept at the lower temperature. This prediction was not confirmed in this case, however.

The proportion of the exposed fish where *D. dendriticum* had penetrated the heart region seemed to increase along with the temperature: 7.4% at about 10°C, and 8.8% at 15°C in experiment A, while it was 20% in experiment B. When comparing the proportions we need to remember that the procercoid dose was clearly higher in experiment B as compared to experiment A.

In general, in the present study we were unable to create such conditions as found at the fish farm in northern Finland (Rahkonen *et al*. 1996), where *D. dendriticum* migrated commonly into the heart of brown trout and caused mortality. In our experiments as well as at the fish farm the fish host and *D. dendriticum* were sympatric in their origin.

In the present experiments, the brown trout seemed to be well protected against the harmful effects of *D. dendriticum*. Freeland (1986) suggested that those host genotypes which are able to develop resistance to certain common parasites of intermediate to high lethality are likely to gain selective advantage. Accordingly, the widespread occurrence of *D. dendriticum* and also its ability to develop occasional strong infections (see the references in the introduction) suggest that the *D. dendriticum*-brown trout association has reached a low degree of pathogenity during their long co-evolution. Observed mortality cases caused by the *D. dendriticum* heart infections at the northern fish farm indicate, however, that this balance may collapse in certain circumstances (Rahkonen *et al*. 1996). The present results demonstrate, however, that in experimental conditions it is apparently difficult to generate and mimic interactions of various factors typical at fish farms and in the natural environment.

In conclusion, increased water temperature did not promote *D. dendriticum*-induced mortality in the present experiments. However, the plerocercoids were longer and a greater proportion of them migrated outside the body cavity of fish in heated aquaria (14–15°C) than in non-heated (11 to 7.5°C). In addition, some indication was obtained that the proportion of heart-infected fish increases along with temperature.

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