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Accumulation of *Diplostomum* spp. (Digenea: Diplostomatidae) Metacercariae in the Eyes of 0+ and 1+ Roach (*Rutilus rutilus*)

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Abstract—McKeown C. A. & Irwin S. W. B. 1997. Accumulation of *Diplostomum* spp. (Digenea: Diplostomatidae) metacercariae in the eyes of 0+ and 1+ roach (*Rutilus rutilus*). *International Journal for Parasitology* 27: 377–380. Unlike other long-term studies of *Diplostomum* spp. metacercariae in fish eyes, this study investigated accumulation of the parasites in the eyes of very young roach for 3 consecutive years. Fish were caught by electro-fishing in the summers of 1990, 1991 and 1992 and the numbers of parasites located in the hosts' eyes were recorded. Two distinct peaks of parasite abundance were observed, 1 in late June and the other in mid-September each year. Significantly, parasite abundance decreased after each of the peaks. Results obtained in a parallel investigation of the accumulation of the same parasites in rainbow trout in an adjacent fish farm showed the same 2 periods of accumulation each year. However, in the fish farm, no decrease in parasite abundance was observed at any time. The decreases in parasite abundance in the wild population can be attributed to mortality of the most heavily parasitized fish. As this did not occur in the farmed fish, in which similar levels of parasitism existed, it is likely that mortality in the wild population was an indirect result of the parasites. © 1997 Australian Society for Parasitology. Published by Elsevier Science Ltd.

Key words: Diplostomum; metacercaria; host mortality; *Rutilus rutilus*.

INTRODUCTION

Although there have been many long-term studies of populations of diplostome parasites and their fish hosts (e.g., Kennedy & Burrough, 1977; Kennedy, 1981), none have concentrated on 0+ and 1+ fish. This study, which was designed primarily to establish the infective periods of diplostome cercariae, took place from 1990 until 1993 and dealt only with juvenile roach (*Rutilus rutilus*) which hatched in a navigational canal alongside the River Bann in N. Ireland. The canal is the water supply for the adjacent Movanaghan Fish Farm in which a parallel study was ongoing at the same time (Field & Irwin, 1994). One aspect of the fish farm study was that it monitored the accumulation of diplostome cercariae in the eyes of each year's cohort of rainbow trout (*Oncorhynchus mykiss*). The studies differ in that 1 population of fish was subject to predation and the rigors of an existence in the wild and the other was protected in an environment free of predators and provided with an adequate supply of food. Both roach and rainbow trout are known to be common hosts of *D. spathaceum* and very heavy intensities have been observed in roach by Bottomley & Woodiwiss (1969) and Wootten (1974) and in rainbow trout by Palmer (1939) and Stables & Chappell (1986). As both host species appear to be susceptible to infection by diplostome cercariae, any substantial differences in results obtained from the 2 studies could be contributed to the influences affecting a natural (wild) population as compared to those of a cultivated population of fish. On the other hand, the possibility that they could result from different responses by roach and rainbow trout to the presence of the parasites must be given consideration.

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MATERIALS AND METHODS

Fish were caught by electro-fishing every 2 weeks from approximately week 12 (April) to week 40 (October) in 1990, 1991 and 1992. Roach fry were chosen because they were the predominant species and easily obtained. Samples of 20, 1+ roach fry (i.e. spawned the previous year) were taken, and when numbers of 1+ fry fell beneath the sample size, 0+ roach were taken in order to continue the study. In the laboratory each fish was aged, its total length measured and its eyes removed and bathed in a salt solution in marked Petri dishes, stored in a refrigerator at 4°C until examined. Each eye was dissected separately and the number of parasites found in the lens was recorded. (Although other parts of each eye were investigated, no parasites were located in them.) Water temperature in the canal was recorded over the periods concerned.

The ecological terms in this paper are based upon a report by Margolis *et al.* (1982) on the use of ecological terms in parasitology. Parasite abundance is taken as the number of a parasite species divided by the total number of fish.

RESULTS

Roach fry acquired eyefluke over the summer period, from May to September in 1990, 1991 and 1992 (Fig. 1A–C). Accumulation of *Diplostomum* metacercariae began each year several weeks after the water temperature had risen above 10°C. 1+ roach fry were easily obtained in spring and early summer and were sampled in preference to the recently hatched 0+ fry. This meant, however, that a residual number of metacercariae were present in these fish from the previous summer.

1+ roach fry in the summer of 1990 (Fig. 1A) showed a peak in abundance of parasites in late June, reaching 8.55 metacercariae per fish in week 25. Abundance levels then dropped before a second peak occurred in mid-September, week 37, reaching an abundance of 10.85 metacercariae per fish.

In 1991 (Fig. 1B) 1+ roach fry showed a pattern of infection which corresponded to that observed in 1990. Parasite abundance rose from a background count of 3 to a distinct peak in week 25. Following this peak of infection the 1+ fry disappeared from the canal and it was necessary to follow accumulation of eyefluke in the younger fish. The parasites in these fish showed a peak of abundance in mid-September, weeks 35–37, before numbers of metacercariae once again decreased as the onset of winter approached.

In 1992 (Fig. 1C), maximum abundance in 1+ fry again occurred in week 25, and was followed by a decline in abundance in week 31. The 0+ fry of 1992 showed similarly low numbers in late July/early August, week 31, but by late August, week 35, both the 1+ and the 0+ fry showed a distinct increase in metacercarial accumulation. In early September, week 37, abundance levels began to drop in 0+ fish, after which time sampling was suspended.

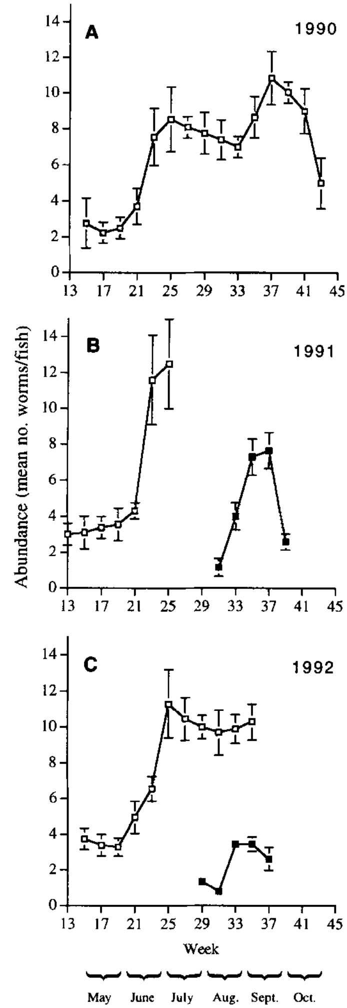

Fig. 1. Accumulation of metacercariae by roach in (A) 1990, (B) 1991 and (C) 1992, showing standard error bars. Open squares, 1+ fry; solid squares, 0+ fry. In 1991, 1+ fish were no longer present in the canal after week 25; following this, 0+ fish were sampled. In 1992, both 1+ and 0+ fish were sampled from week 29 until sampling was suspended.

DISCUSSION

This study has shown 2 distinct periods during which diplostome cercariae accumulated in fishes' eyes. The periods were late May until the end of June and late August until mid-September. In itself this is not new. Two periods of annual infection of fish by diplostome cercariae have been recorded by numerous researchers (e.g., Sweeting, 1974; Brassard *et al.*, 1982a,b; Pennycuick, 1971; Kennedy, 1987). Burrough (1978) interpreted this phenomenon by suggesting that the first period of accumulation was of cercariae which had been released by snails which had survived through the winter, whereas the second period of accumulation resulted from cercariae shed by snails which had hatched in the spring and become infected by diplostome miracidia in the summer.

However, a major, and perhaps very significant, finding is apparent when the results of the present study are compared with those of Field & Irwin (1994). The present study shows that the abundance of parasites in wild roach juveniles fell after the first peak in week 25 and again after the second peak in week 37. These reductions were particularly apparent in 1990 when 1+ fish were sampled throughout the summer, but the same trend was clear in 1991 for 0+ fish and in 1992 for both 1+ and 0+ fry. The results obtained by Field & Irwin (1994) did not show reductions in abundance of diplostome metacercariae in the cultivated rainbow trout at any time during the study. Their results showed an increase from May until week 25, abundance remained at a plateau until the second period of increase and it continued at the second level until the end of the sampling period. The reason why a wild population of roach should seemingly partially lose its parasite burden whereas a cultivated population of rainbow trout should not, must be addressed. Possible reasons for apparent parasite loss from a host population include the effect of immunological response by the hosts, or loss of the more heavily infected hosts from the population. It is possible that roach may have a more effective immune response than rainbow trout to diplostome metacercariae. However, the fact that these parasites accumulate in the transparent parts of the eyes, sites which are considered to be immunologically privileged because they are devoid of blood vessels (Shariff *et al.*, 1980), renders this an unlikely explanation. It is more likely that the more heavily infected young roach were lost due to the indirect effects of the parasites. The more heavily infected specimens may have been increasingly susceptible to predation, a factor which is not encountered in a fish farm. Similarly they may have been less capable of scavenging an adequate supply of food. Various authors, such as Crowden & Broom (1980) and Brassard *et al.* (1982c), have suggested that the presence of diplostome metacercariae increases predation by birds and fish, and alters feeding behaviour in infected fish. Ferguson & Hayford (1941) and Ferguson (1943) stated that diplostome cercariae penetration of fish could result in petechial haemorrhages and exophthalmia. Whatever the effect, it would appear that the more heavily infected roach were being continuously lost from the population because they were less fit to cope with the pressures of a natural environment. The Field & Irwin (1994) results indicated that a similar level of parasitism, with its resulting lack of fitness, was not a problem in the artificial environment of a fish farm. Together these studies of parasite recruitment in juvenile fish may not have provided answers to the questions posed by Kennedy (1981) regarding host mortality induced by heavy diplostome infection, but they appear to lend support to the suggestion that these parasites do result in host mortality, and that mortality is an indirect result of the parasitism.

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