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The differential effects of *Ligula intestinalis* (L.) plerocercoids on host growth in three natural populations of roach, *Rutilus rutilus* (L.)

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Abstract – This study reports the differential effect of the pseudophyllidean cestode, *Ligula intestinalis*, on the growth rates of three fish populations of the roach, *Rutilus rutilus*, under field conditions. Here, we show that only one host-population is affected by parasite-induced gigantism in the first 2 years of host life. Paradoxically, this increase in fish growth is strongly correlated with the parasitic load in plerocercoid stages, in that only the roach population subjected to the heaviest parasitic pressure shows an enhanced growth of cestode-parasitized individuals. This host gigantism observed in only one locality is associated with parasite-induced fish mortality occurring, on average, one year earlier in this fish population when compared to other populations. We discuss the different reasons for this differential parasite effect on growth enhancement across host-population in natural settings. Whether the growth effects represent an adaptive response by the host or a manipulation by *L. intestinalis* of the host, remains to be elucidated.

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Key words: roach; *Ligula intestinalis*; plerocercoids; host growth; parasite-induced gigantism; mortality

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Un resumen en español se incluye detrás del texto principal de este artículo.

Introduction

One important question in host-parasite evolutionary ecology today (Poulin 1998) is whether parasite-induced host modification observed in both natural and experimental conditions is adaptive or simply coincidental (Williams 1992). Some authors believe that the high degree of complexity of behavioural alterations induced by parasites implies a selected design for a specific function, as observed in parasites with complex life-cycles (Holmes & Bethel 1972; Dawkins 1986). Strategies of host exploitation by parasite are diverse, and host gigantism, which is often linked with host castration, may represent a specific adaptive solution to a difficulty in the parasite life-cycle (Baudoin 1975; Dawkins 1982; Minchella 1985; Poulin 1998; Taskinen 1998). However, other explanations are possible, and Taskinen (1998) reviewed three different hypotheses to explain host gigantism. Firstly, host gigantism might be simply the result of a non-adaptive side-effect of the destruction of the gonads by the parasite (Sousa 1983; Keas & Esch 1997), or of experimental conditions such as *ad libitum* food supply (Fernandez & Esch 1991; Keas & Esch 1997). Secondly, gigantism could be a host strategy to enhance its own survival by increasing the probability of outlasting the infection and then recovering a higher fertility rate (Minchella 1985; Ballabeni 1995). Thirdly, gigantism could be a parasite strategy which enhances host growth in a way that favours parasite survival and transmission (Baudoin 1975; Poulin 1998). Trematode-induced

gigantism in marine and freshwater snails or bivalves has been observed in many laboratory studies. However, when comparing the findings on different mollusc species (Sousa 1983; Minchella 1985; Goater et al. 1989; Lafferty 1993; Mouritsen & Jensen 1994), one is left wondering why such similar hosts do not always display a gigantism response to parasite infection. Thus, one additional factor adding confusion to our understanding of gigantism may be a statistical or taxonomic bias. In other words, studies on parasite-associated growth enhancement in hosts from different groups of organisms are still dramatically lacking, thus preventing a global overview of what really happens in nature.

In fish, very few studies have tried to detect the existence of host gigantism, and discussed the possible effects exerted by parasites on host growth. For instance, Arnott et al. (2000) have demonstrated that infected whitefish and three-spined sticklebacks, respectively, had a higher growth rate than their unparasitized counterparts. More specifically, the work of Pulkkinen & Valtonen (1999) has shown that infected fish grew faster in size in the first year of life when compared to their unparasitized conspecifics. Ballabeni & Ward (1993) found no changes in growth of European minnows caused by parasitic infection. On the contrary, other studies have shown that infected fish may grow more slowly than unparasitized specimens in whitefish (Miller 1945), bream (Garadi & Biro 1975), rainbow trout (Wolf & Markiw 1983), and chinook salmon (Hauck 1984). Thus, unambiguous examples of parasite-associated growth in fish are very rare.

Therefore, the purpose of this study was to evaluate the existence, or not, of host gigantism in roach *Rutilus rutilus* (L.) and the possible influence of infection by plerocercoids of the pseudophyllidean cestode, *Ligula intestinalis* (L.), on host growth. Spurred by both conflicting results (see references above) and the work of Pulkkinen & Valtonen (1999) which has shown accelerated growth rates in parasitized fish only during the first year of life, we decided to examine the possible effect of parasitism as a function of host age in roach. In order to examine it in the field, we surveyed during the year 1998 three distinct fish populations in the southwest of France.

In addition to studying the possible quantitative effects of parasites on host growth rates, we also investigated the variation in parasitic load with host age across the three fish populations: one factor potentially of great importance in the case of a parasite-mediated host alteration aimed at increasing transmission to definitive hosts (Poulin 1998).

Materials and methods

Study sites

The roach specimens used in this study originated from three different fish populations in southwestern France: (1) Lavernose–Lacasse gravel pit, (2) Muret gravel pit and (3) Lake Pareloup (see Fig. 1). Fish populations were surveyed monthly from the beginning of January 1998 to the end of December 1998.

Lake Pareloup, located in the Massif Central mountains, is the fifth largest hydroelectric reservoir in France (surface of 1260 ha, volume of water of $168 \times 10^6\,m^3$, location at 800 m above sea level, average annual water temperature of 9 °C). The Muret gravel pit (surface of 17 ha, 175 m above sea level, maximum depth of 4 m with a mean depth of around 2 m, average annual water temperature around 14°C) is situated on the alluvial plain of the Garonne river, 2500 m away from the river. Finally, the Lavernose–Lacasse gravel pit (surface of 23 ha, same water conditions as the Muret pit) is also situated on the alluvial plain of the Garonne river 2000 m from the river channel.

Animals

Roach specimens were gill-netted during monthly over-night samplings with nets of different mesh openings (10, 12, 14, 17, 21, and 27 mm measured between two adjacent knots) in order to cover the maximum size and age range of fish specimens (see Loot et al. 2001a). Unfortunately, we were unable to include in the analyses fish specimens of 4-year-old in both Pareloup and Lavernose–Lacasse localities, and of 2-year-old at Muret, due to the very low numbers of specimens we collected for these specific age classes. After their capture, fish were measured (total body length) to the nearest mm. Both total wet weight and somatic weight of fish were measured to the nearest gram, and then the fish were dissected to determine the number of plerocercoid larval forms occurring in the abdominal cavity. The parasitic load of each fish when plerocercoids occurred was quantified using the index of parasitization (IP) of Kennedy & Burrough (1980). This index is calculated as the ratio of the total weight of plerocercoids per host to the total weight of the host minus the total weight of parasites, with this ratio then being multiplied by 100.

Scalimetry

Several scales were taken from the left side above the lateral line of each fish. The scales were

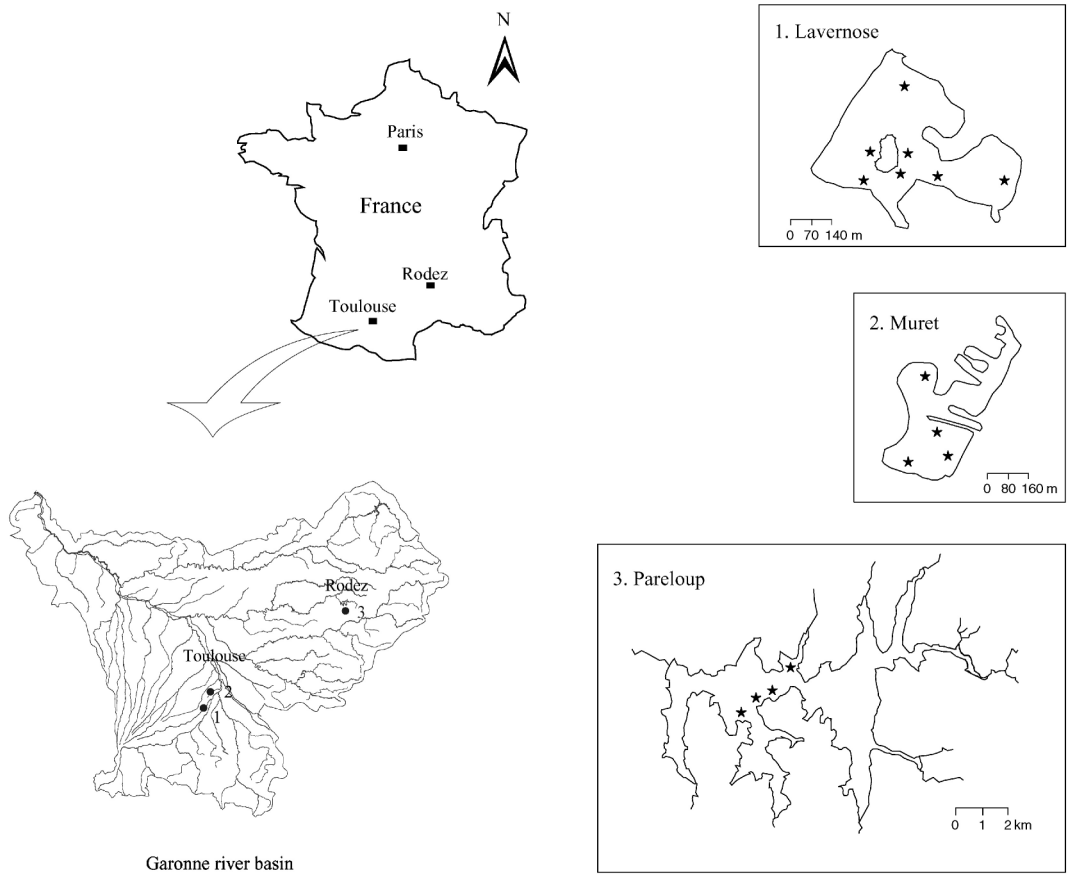

Fig. 1. Geographical location of the three study sites (1) Lavernose–Lacasse gravel pit; (2) Muret gravel pit and; (3) Lake Pareloup. Stars on maps of the different bodies of water represent the different stations sampled within each locality.

cleaned by soaking in a 5% KOH solution before rubbing off the adherent tissues with a small brush. Then, they were rinsed with water and placed between two microscope slides for viewing on a microfiche viewer (Baglinière & Le Louarn 1987). For each fish individual, we examined eight different scales and averaged their measurements. All the measurements were made on the same area of the scale as recommended by Boët & Le Louarn (1985).

First, we measured the total radius of each scale in order to model the relationships between the total body length of the fish (TL) and the total scale radius (TR) across the different fish populations and groups of unparasitized and parasitized roach specimens. Second, we used the non-linear back-calculation technique of Monastyrsky (1930) which allows the use of a set of measurements of marks (R_i) present on one fish individual at one time i (expressed in year) to infer its length (L_i) at the time of formation of each mark (Francis 1990). For a given fish specimen of say 3 years, the body sizes are calculated with the different values taken by R_i for i equalling 1, 2 and 3 years. Then, back-calculated lengths were used to fit the different growth curves for the three fish populations and for uninfected and infected fish specimens (Hickling 1933). The back-calculation method is, however, subject to some validation problems (Francis 1990), and does not necessarily result in the actual lengths at given ages. Nevertheless, when applied to fish from the same population, this method is efficient in revealing the relative differences between growth rates. The application of this method for comparisons of host growths between control and parasitized specimens within the same fish population as we did in this study thus seems to be entirely adequate (Pulkkinen & Valtonen 1999).

Statistics

Data were analysed using S.P.S.S. Version 8.0 for Windows (Norusis 1993). Differences in growth between unparasitized and parasitized fish hosts were analysed using non-parametric statistics (Mann–Whitney's U-tests). In addition, in order to estimate the impact potentially exerted by

plerocercoids on the three roach populations, we analysed the dynamics of infestation levels as a function of host age (Anderson & Gordon 1982; Rousset et al. 1996). Differences in parasite prevalence, mean parasite abundance and IP between the different host age classes were estimated using non-parametric Kruskal–Wallis' K statistics (Sokal & Rohlf 1994; Zar 1996).

Results

Host body size

Prior to the examination of variation in growth rate, the best fits for modelling the relationships between the total length (TL) and the total scale radius (TR) were given by power functions: for Lake Pareloup, unparasitized hosts $TL = 6.490 \times TR^{0.763}$ ($r^2 = 0.680$, $P < 0.001$) and parasitized hosts $TL = 5.1397 \times TR^{0.809}$ ($r^2 = 0.790$, $P < 0.001$); for Muret gravel pit, unparasitized hosts $TL = 6.377 \times TR^{0.735}$ ($r^2 = 0.673$, $P < 0.001$) and parasitized hosts $TL = 7.506 \times TR^{0.687}$ ($r^2 = 0.740$, $P < 0.001$); and for Lavernose–Lacasse gravel pit, unparasitized hosts $TL = 6.604 \times TR^{0.723}$ ($r^2 = 0.806$, $P < 0.001$) and parasitized hosts $TL = 9.067 \times TR^{0.644}$ ($r^2 = 0.640$, $P < 0.001$).

Then, back-calculation of infected and uninfected fish lengths for the three localities were: for Lake Pareloup, for unparasitized hosts $L_i = TL \times (R_i/TR)^{0.763}$ and for parasitized hosts $L_i = TL \times (R_i/TR)^{0.809}$; for Muret, for unparasitized

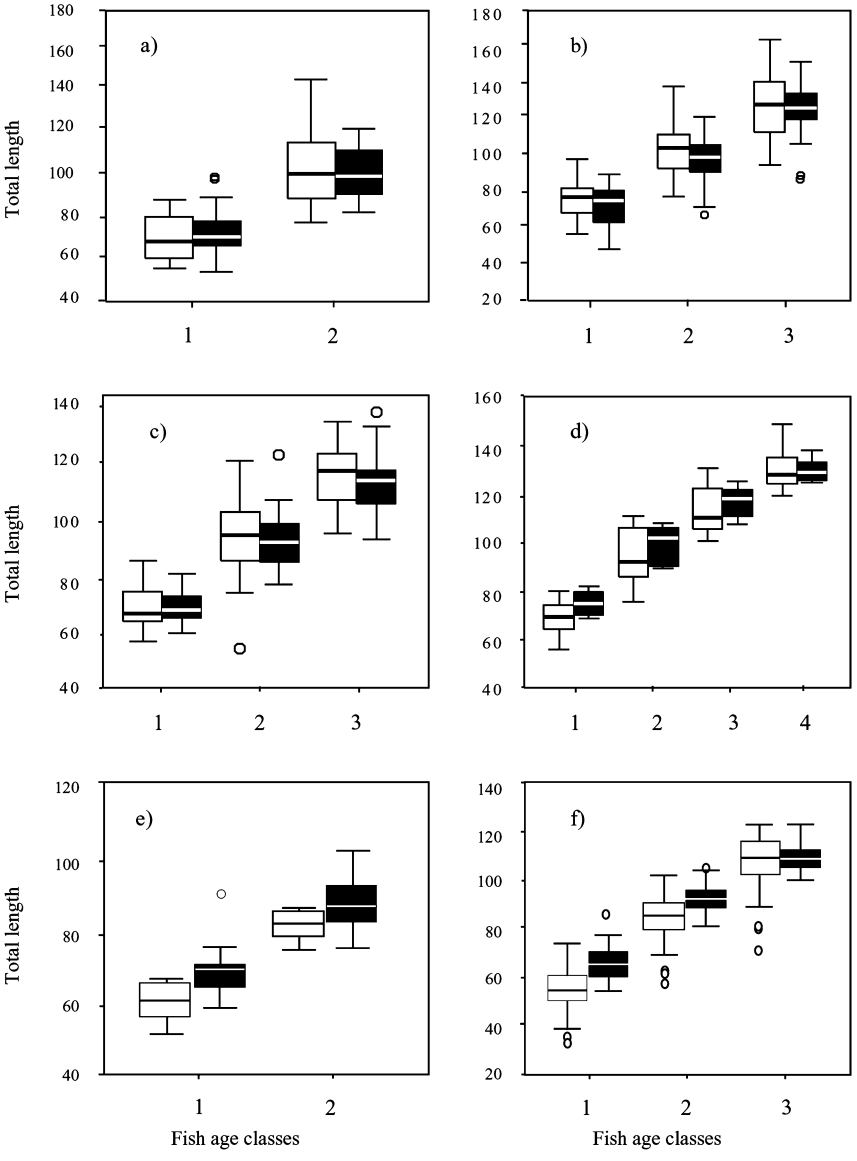

Fig. 2. Boxplots of back-calculated roach lengths. (a) Back-calculated length of 2+ infected (black boxes) and uninfected (open boxes) roach for lake Pareloup; (b) back-calculated length of 3+ infected and uninfected roach for lake Pareloup; (c) back-calculated length of 3+ infected and uninfected roach for Muret gravel pit; (d) back-calculated length of 4+ infected and uninfected roach for Muret gravel pit; (e) back-calculated length of 2+ infected and uninfected roach for Lavernose–Lacasse gravel pit; and (f) back-calculated length of 3+ infected and uninfected roach for Lavernose–Lacasse gravel pit. The top, mid-line and bottom of each boxplot represent the 75th, 50th and 25th percentiles, respectively. The horizontal lines represent the 10th and the 90th percentiles. The open circles represent units in which values were more than 1.5 box-lengths from the 75th percentile (outliers).

Table 1. Results of Mann–Whitney's *U*-tests for comparisons of back-calculated fish body lengths between infected and uninfected roach specimens in (a) Pareloup Lake, (b) Muret gravel pit, and (c) Lavernose–Lacasse gravel pit

Observed fish age	Age classes, *i*	*U*	*P*
(a) Pareloup Lake			
2+	1	397.00	0.856 (ns)
	2	375.50	0.367 (ns)
3+	1	161.00	0.276 (ns)
	2	335.00	0.703 (ns)
	3	394.00	0.330 (ns)
(b) Muret gravel pit			
3+	1	510.00	0.658 (ns)
	2	471.00	0.346 (ns)
	3	441.00	0.184 (ns)
4+	1	22.00	0.059 (ns)
	2	39.00	0.541 (ns)
	3	35.00	0.367 (ns)
	4	45.00	0.858 (ns)
(c) Lavernose–Lacasse gravel pit			
2+	1	300.00	0.000 (***)
	2	540.00	0.047 (*)
3+	1	35.00	0.000 (***)
	2	90.00	0.000 (***)
	3	85.00	0.130 (ns)

Fish lengths are calculated for each *i* year class, and then parasitized and unparasitized groups are compared using the *U* statistics. *Legend*: ns, not significant; $^{*}P < 0.05$; $^{***}P < 0.001$.

hosts $L_i = \mathrm{TL} \times (R_i/\mathrm{TR})^{0.735}$ and for parasitized hosts $L_i = \mathrm{TL} \times (R_i/\mathrm{TR})^{0.687}$; and for Lavernose–Lacasse, for unparasitized hosts $L_i = \mathrm{TL} \times (R_i/\mathrm{TR})^{0.723}$ and for parasitized hosts $L_i = \mathrm{TL} \times (R_i/\mathrm{TR})^{0.644}$, with L_i and R_i being the fish length and the scale radius, respectively, at the time of the formation of the *i*th annulus. Differences between median back-calculated lengths at *i*th year for uninfected and infected roach are illustrated for the three fish population in Fig. 2 and Table 1.

For Lake Pareloup, considering the two fish age classes, i.e., 2+ and 3+ years, no significant differences in growth rates were found between unparasitized and parasitized roach (Table 1a). Similarly, in the Muret gravel pit, for 3+ and 4+ fish, no significant differences were observed between control and infected hosts (Table 1b). For the Lavernose–Lacasse gravel pit, however, we observed a significant difference in back-calculated length estimates between uninfected and infected roach specimens of the 2+ and 3+ age classes (see Table 1c). For the first 2 years of life, infected fish specimens from this gravel pit were estimated to be slightly longer than uninfected fish; after that age, we found no difference (see Table 1c).

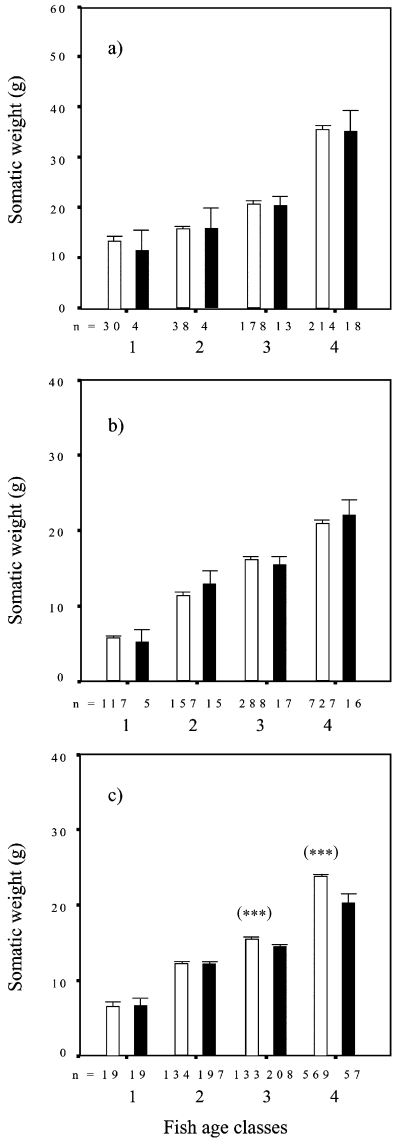

Fig. 3. Age-specific somatic weight differences between infected (black bars) and uninfected (open bars) roach for the three study localities: (a) lake Pareloup; (b) Muret gravel pit; and (c) Lavernose–Lacasse gravel pit. The total number of roach specimens surveyed for each age class is indicated. Asterisks indicate that comparisons are significant (same levels of significance as in Table 1); note the different scales on the *y*-axis (see Results for further details).

Somatic host weight

Figure 3 illustrates differences between uninfected and infected hosts with respect to somatic weight for the three roach population surveyed. For Lake Pareloup (Fig. 3a) and the Muret gravel pit (Fig. 3b), all unparasitized and parasitized fish age classes showed no difference in their somatic weight (*t*-test for all roach age classes, $P > 0.05$). In Lavernose–Lacasse roach specimens (Fig. 3c), no significant difference was observed for the youngest fish 1- and 2-year old. However, we

observed a difference in host somatic weight between uninfected and infected fish for 3-year, 4-year and 5-year-old age classes with parasitized fish having, on average, a lower specific somatic weight (for 3+ roach, $t = 3.599$, $P < 0.001$; for 4+ roach, $t = 5.289$, $P < 0.001$).

Parasite-induced mortality

Figure 4 illustrates the changes in parasite infestation levels with fish age for the three localities concerned. Variations in prevalence (Fig. 4a), in mean parasite abundance (Fig. 4b) and IP (Fig. 4c) estimates as a function of fish age show a similar curvilinear pattern. For the three roach populations, a maximum level of prevalence, mean parasite abundance and IP occurs in the medium-size fish individuals, thus indicating that a parasite-induced mortality is likely to occur. Trends in changes in mean parasite abundance with fish age were significant, i.e., Lake Pareloup (Kruskal–Wallis' test, $K = 21.59$, d.f. $= 6$, $P < 0.01$), Muret gravel pit ($K = 20.90$, d.f. $= 6$, $P < 0.01$) and Lavernose–Lacasse gravel pit ($K = 125.58$, d.f. $= 5$, $P < 0.001$). Changes of IP with host age were also significant for the three populations, i.e., Pareloup Lake ($K = 32.02$, d.f. $= 6$, $P < 0.001$), Muret gravel pit ($K = 16.35$, d.f. $= 6$, $P < 0.05$), and Lavernose–Lacasse gravel pit ($K = 124.30$, d.f. $= 5$, $P < 0.001$).

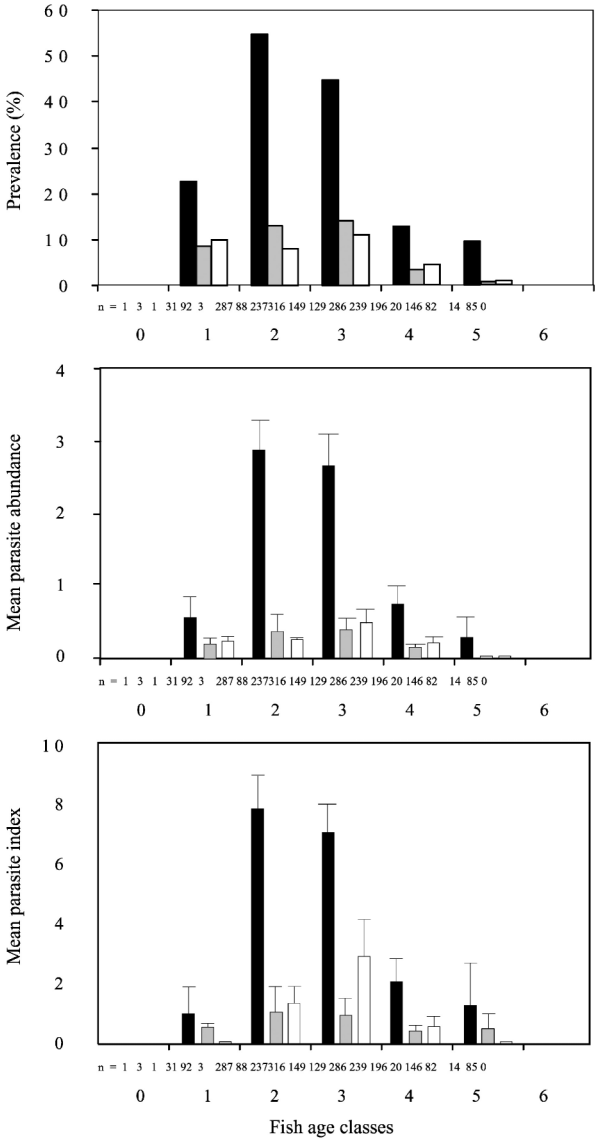

Fig. 4. Age-specific parasitic load for the three study localities: lake Pareloup (open bars), Muret gravel pit (grey bars) and Lavernose–Lacasse gravel pit (black bars): (a) variability of parasite prevalence (%) for *L. intestinalis* plerocercoids in roach as a function of host age classes; (b) variability of mean abundance (±SE) with host age classes; and (c) variability of mean parasite index (±SE) with host age classes. The total number of roach specimens surveyed for each age class is indicated.

At Lake Pareloup the maximum parasite prevalence (11%) was seen in 3-year-old roach, at Muret (14%) also in 3-year-old roach, and at Lavernose–Lacasse (55%) in 2-year-old roach specimens. The maximum mean parasite abundance reached 0.43 (SD, ±0.09) plerocercoids at Pareloup for 3-year-old roach, 0.32 (SD, ±0.09) plerocercoids at Muret for 3-year-old roach as well, and 2.85 (SD, ±0.21) at Lavernose–Lacasse for 2-year-old roach. The maximum IP reached 2.77 g (SD, ±0.64) at Pareloup for 3-year-old roach, 0.82 (SD, ±0.28) at Muret for 2-year-old roach and 7.80 (SD, ±0.56) at Lavernose–Lacasse for 2-year-old fish. The most important finding is that roach at Lavernose–Lacasse were suffering more heavily from plerocercoid worms and were affected, on average, at a younger age, i.e., 1 year earlier, than the two other populations of fish.

Discussion

The present findings confirmed Pulkkinen & Valtonen (1999) study of a parasite-associated host growth rate enhancement in the first 2 years of life in fish. However, this seems likely to happen in only one roach population, i.e., the Lavernose–Lacasse gravel pit, among the three fish localities we surveyed. The host gigantism observed in the first 2 years of life of parasitized roach specimens was correlated with the heaviest levels of infection, in terms of prevalence (up to 55%), of mean abundance (up to 2.85, SD, ±0.21) and of parasite index (up to 7.80, SD, ±0.56), observed in any of the three localities. For instance, the very high level of prevalence, two times higher than that in the experiment of Arnott et al. (2000), would tend to indicate that roach individuals at Lavernose–Lacasse might be relatively more susceptible to *L. intestinalis* infestation when compared to fish in

the two other populations (Pareloup, prevalence up to 11% and Muret up to 14%). Another alternative, but not exclusive, explanation could be that phenotypic differences between parasite population and/or possible differences in the probability of completion of the parasite's life cycle may affect their ability to infect the different host compartments and establish within the different fish populations. The fact that more than half of the fish can be parasitized at Lavernose–Lacasse might implicate mutually stronger selective pressures on hosts and parasites. In addition, in contrast to Pulkkinen & Valtonen's (1999) work, we found a cestode-induced host mortality in the three fish populations, with the Lavernose–Lacasse roach population being by far ($K = 125.58$, $P < 0.001$) the most heavily harmed by parasitic effects. Examination of mortality curves across the three different roach populations showed that fish in the Lavernose–Lacasse gravel pit were impacted earlier in their life, i.e., when 2-year old, by the debilitating effects of parasites, than fish from the Pareloup and Muret localities.

There are four different possible explanations for this pattern of results. First, the results are consistent with the idea that plerocercoids could secrete growth enhancers resulting in accelerated host growth rates. Interestingly, plerocercoids of the pseudophyllidean tapeworm, *Spirometra mansonoides*, in mice produce and release a protein, a plerocercoid growth factor (PGF) which binds and activates host growth hormone (GH) receptors (Phares 1996, 1997). Whereas PGF may obviously stimulate mouse growth in the case of *S. mansonoides*, we are unaware of the existence of such growth protein enhancers in *L. intestinalis*. In addition, the other related pseudophyllidean tapeworm, *Schistocephalus solidus*, parasitizing the three-spined stickleback is another case where no such hormonal productions have been identified (see Arme & Owen 1967; Arnott et al. 2000). Hence, the cestode-associated gigantism of roach we observed in this study is unlikely to be due to such physiological manipulation, an explanation which is entirely consistent with the fact that not all three roach populations were affected by accelerated growth rates.

The second explanation for the enhanced growth of cestode-parasitized individuals at Lavernose–Lacasse may be that some parasites have become specialised for exploiting the reproductive organs of the hosts. This can be achieved directly by feeding on the gonads of the host or by usurping the space normally allocated to gonads and eggs, or indirectly either by diverting energy away from the gonad development towards somatic usage (Sorensen & Minchella 1998) or by the production of a peptide-like that inhibits the production of sexual organs in host (Coustau et al. 1991; Schallig et al. 1991) and in turn may stimulate the secretion of growth enhancers (Hordijk et al. 1992). Usually, castrated hosts divert energy towards somatic growth instead of allocating it to reproduction. Thus, parasitized hosts reach larger body sizes than their uninfected conspecifics (Poulin 1998). This phenomenon is best known from mollusc–trematode interactions but occurs in many other systems. It is likely that *L. intestinalis* plerocercoids inhibit host fish gonad development but they obviously do not fully castrate their hosts (Kerr 1948; Arme et al. 1982), as is commonly believed. This may appear to be counter-intuitive because young plerocercoids derive host energy to their own benefit (larger larval forms may reach 380 mm length and 2.2 g with up to 30 plerocercoids of different sizes observed in one roach specimen) and immune responses exist in cyprinids against *L. intestinalis* infection (Taylor & Hoole 1994). Although our findings might be explained by a host strategy for preferential allocation of energy to somatic growth in parasitized individuals, this hypothesis does not explain the fact that we did not observe a cestode-associated growth enhancement in the heaviest infected fish of the two other localities, i.e., Pareloup and Muret.

A third (but related to the previous one) explanation for the growth enhancement in parasitized hosts is that this phenomenon might result from a change in fish foraging behaviour, increased food conversion efficiency and/or reduced activity caused by parasite (Arnott et al. 2000). For instance, plerocercoid larval forms of *S. solidus*, through a nutrient-energy drain, increase the nutritional demand of infected sticklebacks and stimulate their foraging behaviour (Walkey & Meakins 1970; Pascoe & Mattey 1977; Giles 1983; Milinski 1985; Godin & Sproul 1988). Adamek et al. (1996) have shown that infestation of the roach by *L. intestinalis* strongly influenced both food intake and diet composition, with parasitized roach ingesting more animal food relative to plant food than did their non-infected counterparts. Walkey & Meakins (1970) have suggested that with a heavy parasite load, particularly if this is represented by a large number of small rapidly growing plerocercoids of *S. solidus*, there is a considerable depletion of endogenous host food reserves, a fact that is occasionally substantiated by apparent weight losses in parasitized sticklebacks under considerable energetic stress. Additionally, infected sticklebacks may prefer smaller prey items relative to their stomach capacity because the presence of *S. solidus* plerocercoids

can restrict the space available for food in and through the stomach (Milinski 1985; Cunningham et al. 1994). Furthermore, infected sticklebacks are poor competitors and should favour small prey because these are ignored by uninfected fish and so are not strongly contested (Milinski 1985).

Our findings have demonstrated that for the Lavernose–Lacasse roach population only, parasitized fish grew faster and reached a larger body size than do uninfected specimens. It can be suggested that *L. intestinalis* affects the behaviour of roach at Lavernose–Lacasse in a manner that makes parasitized younger fish spend more time foraging. Parasite-induced enhancement of feeding activity in hosts might then result in higher exposure to infected copepods. Then after the first 2 years of life, we observed a negative effect on fish growth and a decrease in somatic weight which could logically be explained by the energetic drain driven by the massive numbers of plerocercoids observed in 3-year-old roach specimens (see also Pulkkinen & Valtonen 1999). Thus, in the Lavernose–Lacasse locality, the hypothesis of enhanced host growth due to behavioural and/or physiological changes in parasitized individuals may be entirely relevant. However, it does not explain why in the Muret gravel pit which is only 22 km away from Lavernose–Lacasse, and which presents the same ecological conditions except for the degree of parasitism, we did not observe such a phenomenon of host gigantism. Possibly, physiological differences between the two host-populations might be invoked for the different outcomes in growth rates. The argument that parasite populations could be distinct does not hold here due to the close proximity of the two localities, and the possibility of parasite exchanges via avian definitive hosts.

The last two hypotheses propose that growth effects in hosts correspond to either an adaptative response by fish to parasitic infection, or a manipulation exerted by *L. intestinalis* to increase the likelihood of its transmission, respectively. First, gigantism observed in parasitized hosts might be a response which would promote host survival particularly over the winter season. The irrelevance of this hypothesis based on the parasite life-cycle dynamics has been discussed by Arnott et al. (2000) for another pseudophyllidean worm, *S. solidus*, parasitizing sticklebacks (Tierney et al. 1996). For *L. intestinalis* in roach this is also unlikely that enhancement of growth associated with cestodes might act as an 'insurance' for offsetting the risk of mortality during winter. In addition, the hypothesis formulated by Ballabeni (1995) suggesting that parasite-induced growth enhancement could correspond to a strategy for outliving infection is not valid in the case of *L. intestinalis* in roach because this cestode has an annual life-cycle and fish do not possess specific mechanisms to eliminate plerocercoids when they are completely mature inside the host body cavity. Furthermore, this hypothesis of a potential host adaptation does not explain why the two other roach populations, i.e., Pareloup and Muret, did not show enhanced growth in parasitized hosts because logically we could expect such a response in those localities at least for the most heavily infected individuals present.

Finally, host gigantism could be a parasite adaptation to enhance transmission. Previous studies have demonstrated that *L. intestinalis* strongly affects roach behaviour, i.e., parasitized specimens are preferentially found in shallow waters when compared with uninfected fish in natural conditions (Loot et al. 2001a). Moreover, this finding is supported by experimental studies which have demonstrated that infected roach individuals were more frequently observed near the water surface of a tank than control specimens found at the bottom of the water (Loot et al. 2002). In addition, the tapeworm modifies the normal swimming behaviour of fish into a jerky-style swimming and confers to infected roach a chubby-fat phenotype (Loot et al. 2001b); this may represent an efficient way to attract the attention of bird predators, a scenario entirely compatible with the adaptative parasite-induced manipulation of host to increase the parasite trophic transmission (Holmes & Bethel 1972; Lafferty 1997; Poulin 1998; Lafferty et al. 2000). The fact that enhancement of host growth only happens in the Lavernose–Lacasse population is consistent with the local level of infestation, by far the heaviest parasite load observed in this study. The absence of host gigantism in the two other localities we surveyed might be the consequence of different parasite life-cycle efficiencies, with the dynamics of *L. intestinalis* being more closely adapted to local population of their hosts at Lavernose–Lacasse, and/or the 3-host compartment dynamics being more stochastic than that observed at Lavernose–Lacasse.

Obviously, experimental research is required in order to understand the phenomenon of host fish growth in this host-parasite system. Unfortunately, the *L. intestinalis*-roach model does not easily allow experimental investigations compared with the *S. solidus*-stickleback model, notably because infected fish are very sensitive to any manipulation. Observational studies such as the present one are however, necessary, and the *L. intestinalis*-cyprinids model makes an interesting

choice for comparative studies in natural settings. In summary, our findings indicate that either close coevolutionary interactions may happen locally to drive parasite transmission success, or that some local host-populations may be more susceptible to *L. intestinalis* infection independently of coevolutionary processes.

Resumen

1. Este estudio evalúa los efectos diferenciales del cestodo pseudophyllideo, *Ligula intestinalis*, sobre las tasas de crecimiento de poblaciones de *Rutilus rutilus*. Mostramos que solo una población huésped está afectada por gigantismo inducido por el parásito durante los dos primeros años de vida.
2. Paradojicamente, el incremento en el crecimiento de los peces esta altamante relacionado con la carga parasitaria, en que solamente la población de *R. rutilus* sujeta a mayor presión parasitaria mostró un mayor crecimiento de los individuos parasitados por el cestodo. Este gigantismo observado en solamente una población está asociado a una mortalidad de los peces inducida por el parasito que ocurre, de promedio, un año antes en esta población.
3. Discutimos las varias razones de este efecto parasitario diferencial sobre el crecimiento de poblaciones salvajes. Que los efectos sobre el crecimiento representen una respuesta adaptativa del huésped o sean el resultado de una manipulación del huésped por *L. intestinalis* permanece todavia sin dilucidar.

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References

Adamek, Z., Barus, V. & Prokes, M. 1996. Summer diet of roach (*Rutilus rutilus*) infested by *Ligula intestinalis* (Cestoda) plerocercoids in the Dalesice reservoir (Czech Republic). Folia Zoologica 45(4): 347–354.

Anderson, R.C. & Gordon, D.M. 1982. Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite-induced host mortalities. Parasitology 85: 373–398.

Arme, C., Griffiths, D.V. & Sumpter, J.P. 1982. Evidence against the hypothesis that the plerocercoid larva of *Ligula intestinalis* (Cestoda: Pseudophyllidea) produces a sex steroid that interferes with host reproduction. Journal of Parasitology 68(1): 169–171.

Arme, C. & Owen, R.W. 1967. Infections of the three-spined stickleback *Gasterosteus aculeatus* L. with plerocercoid larvae of *Schistocephalus solidus*, with special reference to pathological effects. Parasitology 57: 301–304.

Arnott, S.A., Barber, I. & Huntingford, F.A. 2000. Parasite-associated growth enhancement in a fish-cestode system. Proceeding Royal Society of London 267(B): 657–663.

Baglinière, J.L. & Le Louarn, H. 1987. Caractéristiques scalimétriques des principales espèces de poissons de poissons d'eau douce de france. Bulletin Français de Pêche et de Pisciculture 306: 1–39.

Ballabeni, P. 1995. Parasite-induced gigantism in a snail: a host adaptation? Functional Ecology 9: 887–893.

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.

Baudoin, M. 1975. Host castration as parasitic strategy. Evolution 29: 335–352.

Boët, P. & Le Louarn, H. 1985. La croissance du poisson. Techniques d'étude. In: Gerdeaux, D. & Billard, R., ed. Gestion piscicole des lacs et retenues artificielles, Paris: Inra. pp. 125–142.

Coustau, C., Renaud, F., Delay, B., Robbins, I. & Mathieu, M. 1991. Mechanisms involved in parasitic castration. in vitro effects of the trematode *Prosorhynchus squamatus* on the gametogenesis and the nutrient storage metabolism of the marine bivalve mollusc *Mytilus edulis*. Experimental Parasitology 73: 36–43.

Cunningham, E.J., Tierney, J.F. & Huntingford, F.A. 1994. Effects of the cestode *Schistocephalus solidus* on food intake and foraging decisions in the three-spined stickleback, *Gasterosteus aculeatus*. Ethology 97: 65–75.

Dawkins, R. 1982. The extended phenotype. Oxford University Press, Oxford, UK.

Dawkins, R. 1986. The blind watchmaker. London, UK: Longman Publishers.

Fernandez, J. & Esch, G.W. 1991. Effect of parasitism on the growth rate of the pulmonate snail *Helisoma anceps*. Journal of Parasitology 77: 937–944.

Francis, R.I.C.C. 1990. Back-calculation of fish length: a critical review. Journal of Fish Biology 36: 883–902.

Garadi, P. & Biro, P. 1975. The effect of ligulosis on the growth of bream (*Abramis brama* L.) in lake Balaton. Annales Instituti Biologici (Tihany) Hungaricae Academiae Scientiarum 42: 165–173.

Giles, N. 1983. Behavioural effects of the parasite *Schistocephalus solidus* (Cestoda) on an intermediate host, the three-spined sticklebacks *Gasterosteus aculeatus* L. Animal Behaviour 31: 1192–1194.

Goater, T.M., Shostak, A.W., Williams, J.A. & Esch, G.W. 1989. A mark-recapture study of trematode parasitism in overwintered *Helisoma anceps* (Pulmonata), with special reference to *Halipegus occidualis* (Hemiuridae). Journal of Parasitology 75: 553–560.

Godin, J.-G.J. & Sproul, C.D. 1988. Risk taking in parasitized sticklebacks under threat of predation: effects of energetic need and food availability. Canadian Journal of Zoology 66: 2360–2367.

Hauck, A.K. 1984. A mortality and associated tissue reactions of ghinook salmon, *Oncorhynchus tshawytscha* (Waldbaum), caused by the microsporidan *Loma* sp. Journal of Fish Diseases 7: 217–229.

Hickling, C.F. 1933. The natural history of the hake. IV. Age-determination and growth-rate. Fishery Investment London Series 2: 13(2): 120 pp.

Holmes, J.C. & Bethel, W.M. 1972. Modification of intermediate host behaviour by parasites. In: Canning, E.U. & Wright, C.A. ed. Behavioural aspects of parasite transmission. London: Academic Press. pp. 123–149.

Hordijk, P.L., De Jong-Brink, M., Ter Matt, A., Pieneman, A.W., Lodder, J.C. & Kits, K.S. 1992. The neuropeptide schistosomin and heamolymph from parasitized snails induce similar changes in excitability in neuroendocrine cells controlling reproduction and growth in a freshwater snail. Neurosciences Letters 136: 193–197.

Keas, B.E. & Esch, G.W. 1997. The effect of diet and reproductive maturity on growth and reproduction of *Helisoma anceps* (Pulmonata) infected by *Halipegus occidualis* (Trematoda). Journal of Parasitology 83: 96–104.

Kennedy, C.R. & Burrough, R.J. 1980. The establishment and subsequent history of a population of *Ligula intestinalis* in roach *Rutilus rutilus* (L.). Journal of Fish Biology 19: 105–126.

Kerr, T. 1948. The pituitary in normal and parasitised roach (*Leuciscus rutilus* Flemm.). Quartely Journal of the Microscopy Sciences 89: 129–137.

Lafferty, K.D. 1993. Effects of parasitic castration on growth, reproduction and population dynamics of the marine snail *Cerithidea californica*. Marine Ecology Progress Series 96: 229–237.

Lafferty, K.D. 1997. The Ecology of Parasites in a Salt Marsh Ecosystem. In: Beckage, N.E., ed. Parasites and pathogens: effects on host hormones and behavior. London, UK: Chapman & Hall. pp. 316–332.

Lafferty, K.D., Thomas, F. & Poulin, R. 2000. Evolution of host phenotype manipulation by parasites and its consequences. In: Poulin, R., Morand, S. & Skorping, A., ed. Evolutionary biology of host-parasite relationships: theory meets reality. Amsterdam: Elsevier Science. pp. 117–127.

Loot, G., Aulagnier, S., Lek, S., Thomas, F. & Guégan, J.F. 2002. Experimental demonstration of a parasite (*Ligula intestinalis* L.) induced behavioural modification in a cyprinid fish (*Rutilus rutilus* L.). Canadian Journal of Zoology 80: in press.

Loot, G., Brosse, S., Lek, S. & Guégan, J.F. 2001a. Behaviour of roach (*Rutilus rutilus* L.) altered by *Ligula intestinalis* (Cestoda: Pseudophyllidea): a field demonstration. Freshwater Biology 46: 1219–1227.

Loot, G., Lek, S., Brown, S.P. & Guégan, J.F. 2001b. Phenotypic modification of roach (*Rutilus rutilus*) infected with *Ligula intestinalis* L. (Cestoda, Pseudophyllidea). Journal of Parasitology 87: 1002–1010.

Milinski, M. 1985. Risk of predation of parasitized sticklebacks (*Gasterosteus aculeatus* L.) under competition for food. Behaviour 93: 203–215.

Miller, R.B. 1945. Effect of *Triaenophorus* problem in Canadian lakes. Journal of the Fisheries Research Board of Canada 6: 334–337.

Minchella, D.J. 1985. Host life-history variation in response to parasitism. Parasitology 9: 205–216.

Monastyrsky, G.N. 1930. Über Methoden zur Bestimmung des linearen Wachstums der Fische nach der Schuppe. Abhandlungen des Wissenschaftlichen Institutes für Fischereiwirtschaft 5: 5–44.

Mouritsen, K.N. & Jensen, K.T. 1994. The enigma of gigantism: effect of larval trematodes on growth, fecundity, egestion and locomotion in *Hydrobia ulvae* (Pennant) (Gastropoda: Prosobranchia). Journal of Experimental Marine Biology and Ecology 181: 53–66.

Norusis, M.J. 1993. SPSS for Windows. Base system user's guide release 6.0., SPSS Inc., Chicago.

Pascoe, D. & Mattey, D. 1977. Dietary stress in parasitized and non-parasitized *Gasterosteus aculeatus* L. Zeitschrift für Parasitenkunde 51: 179–186.

Phares, C.K. 1996. An unusual Host-Parasite Relationship: the Growth Hormone-like Factor from Plerocercoids of Spirometrid Tapeworms. International Journal for Parasitology 26: 575–588.

Phares, C.K. 1997. The Growth Hormone-Like Factor from Plerocercoids of the Tapeworm *Spirometra mansonoides* is a Multifunctional Protein. In: Beckage, N.E., ed. Parasites and pathogens: effects on host hormones and behavior. London, UK: Chapman & Hall. pp. 3–36.

Poulin, R. 1998. Evolutionary ecology of parasites: from individuals to communities. Chapman & Hall Ltd, London, UK.

Pulkkinen, K. & Valtonen, E.T. 1999. Accumulation of plerocercoids of *Triaenophorus crassus* in the second intermediate host *Coregonus lavaretus* and their effect on growth of the host. Journal of Fish Biology 55: 115–126.

Rousset, F., Thomas, F., De Meeûs, T. & Renaud, F. 1996. Inference of parasite-induced host mortality from distributions of parasite loads. Ecology 77(7): 2203–2211.

Schallig, H.D.F.H., Sassen, M.J.M., Hordijk, P.L. & De Jong-Brink, M. 1991. *Trichobilharzia ocellata* influence of infection on the fecundity of its intermediate snail host *Lymnaea stagnalis* and cercarial induction of the release of schistosomin, a snail neuropeptide antagonizing female gonadotropic hormones. Parasitology 102: 85–91.

Sokal, R.R. & Rohlf, F.J. 1994. Biometry: the Principles and Practice of Statistics in Biological Research, 3rd edn. New York: Freeman.

Sorensen, R.E. & Minchella, D.J. 1998. Parasite influences on host life history: *Echinostoma revolutum* parasitism of *Lymnaea elodes* snails. Oecologia 115: 188–195.

Sousa, W.P. 1983. Host life history and the effect of parasitic castration on growth: a field study of *Cerithidea californica* (Gastropoda: Prosobranchia) and its trematode parasites. Journal of Experimental Marine Biology and Ecology 73: 273–296.

Taskinen, J. 1998. Influence of trelatode parasitism on the growth of a bivalve host in the field. International Journal for Parasitology 28: 599–602.

Taylor, M.J. & Hoole, D. 1994. Modulation of fish lymphocyte proliferation by extracts and isolated proteinase inhibitors of *Ligula intestinalis* (Cestoda). Fish Shellfish Immunology 4: 221–230.

Tierney, J.F., Huntingford, F.A. & Crompton, D.W. 1996. Body condition and reproductive status in sticklebacks exposed to a single wave of *Schistocephalus solidus* infection. Journal of Fish Biology 49: 483–493.

Walkey, M. & Meakins, R.H. 1970. An attempt to balance the energy budget of a host-parasite system. Journal of Fish Biology 2: 362–372.

Williams, G.C. 1992. Natural Selection: Domains, Levels, and Challenges. Oxford University Press, Oxford, UK.

Wolf, K. & Markiw, M.E. 1983. Salmonid whirling disease: new findings indicate that the infective stage produced in and released by tubificid oligochaete is a Triactinomyxon. Fish Health News 10: ii–iv.

Zar, J.H. 1996. Biostatistical Analysis. Prentice Hall International, Upper Saddle River, NJ, USA.