**Galaxias maculatus** (Galaxiidae, Salmoniformes) infected with *Acanthostomoides apophalliformis* (Digenea, Platyhelminthes) in Southern Argentina. Pathology and absence of parasite induced mortality

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**Abstract**

This study was conducted to analyze at histopathological level, lesions produced by *Acanthostomoides apophalliformis* in the native fish *Galaxias maculatus*, and relate them to quantitative results on parasite-induced fish mortality, in Lake Moreno, Southern, Argentina. Absence in most lesions of inflammatory reaction, unaltered hepatic parenchyma at a short distance from the foci of the lesions and viable appearance of parasites, all suggest a good reciprocal adaptation. This hypothesis is reinforced by data showing the absence of parasite-induced mortality in the fish population.

**Key-words:**
Galaxias maculates.
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Histopathology.
Parasite-induced host mortality.

**Introduction**

Lesions produced by parasites on wild fish are usually studied at histopathological level, i.e. at individual level. Nevertheless, only a mortality study (a population level study), can show the ecological significance of individual lesions.

Anderson and Gordon1 introduced a quantitative analysis for detecting signs of parasite-induced host mortality (PIHM) in fish populations. They used simulation experiments (Monte Carlo) and considered a decline in older fish of both parasite abundance and degree of aggregation of parasite frequency distribution (measured as the variance to mean ratio), to be clear signs of such a mortality. Furthermore, these authors pointed out that fish-metacercaria systems, are more reliable to detect PIHM by using their method, because parasite life spans are long in relation to that of their hosts, so that death of parasites results mainly from hosts death1.

*Acanthostomoides apophalliformis* (Szidat) is a digenetic trematode whose liver dwelling metacercaria parasitizes “puyenes” *Galaxias maculatus* (Jenyns), a small native fish under protection in Nahuel Huapi National Park and Reserve. Several papers report biological data both on *A. apophalliformis*’s and *G. maculatus*2,3,4,5,6,7,8.

Although a high abundance of *A. apophalliformis* infecting “puyenes” in Lake Moreno (mean intensity range, 1.8 – 4.4; prevalence range, 41.4 ~96.7 %), has been reported4, there are no studies on pathology and PIHM in this host-parasite system.

We analyzed at histopathological level, lesions produced by *A. apophalliformis* in *G. maculatus* in Lake Moreno, Southern Argentina, and related them to parasite-induced host mortality.

**Materials and Methods**

The study was carried out at Lake Moreno, in the Nahuel Huapi National Park and Reserve (40° 08′-41° 35′ S; 71° 02′-71° 37′ W), Southern Argentine. A total of 477 *G. maculatus* were captured using baited traps on January 2001 and processed in the laboratory by one of us1. One part of the information gathered from these fish was...
Figure 1 - Section of *Acanthostomoides apophalliformis* metacercaria showing inner (il) (PAS +, parasite origin) and outer (ol) (PAS -, produced by the host) layers of the capsule (PAS, x 100).

Figure 2 - Section showing a lesion produced by *Acanthostomoides apophalliformis* in the liver of *Galaxias maculatus*, il as in Figure 1, h = normal hepatocytes, nt = host tissue with necrotic aspect, fr = fibroblastic reaction, mo = mononuclear cells (H & E, x 200).
Figure 3 - Variation of parameters for detecting parasite-induced host mortality according to host size (taken as age indicator) for Galaxias maculatus from Lake Moreno. a: abundance; b: variance to mean ratio.
used for an other study\(^9\), and the rest was used for the present one. In the lab, all the fish were dissected under stereomicroscope to collect and count *A. apophalliformis* but only 20 of them were studied for histoapathology. The livers were fixed in 10\% buffered formalin, or in Carnoy’s fluid. After fixation, they were embedded in paraffin wax, sectioned at 5-7\,\mu m and stained with haematoxylin and eosin (H&E), or Schiff’s periodic acid (PAS).

In the present study we define abundance as Nº parasites/examined fish (including fish without parasites in the calculation) and mean intensity as mean Nº parasites/infected fish\(^2\).

## Results and Discussion

Up to 13 encapsulated metacercariae of *A. apophalliformis* were found in the hepatic parenchyma of “puyenes”. Metacercarial capsules showed two layers, the inner layer being a cyst wall of parasite origin, with a positive PAS reaction, and the outer layer being formed by the host and consisting of fibrous connective tissue (Figure 1).

Independently of the number of metacercariae, inflammatory reaction was absent in most cases (17 of 20 fish) and the hepatic parenchyma was unaltered a short distance from the foci of the lesions (Figure 2). Weak inflammatory reactions were observed in three specimens (Figure 2).

Pathological finding differ form others on “puyenes” infected with the same parasite in Chile where lesions with more degenerative changes in the hepatic parenchyma were observed\(^7\). Absence of a chronic inflammatory response and the viable aspect of the parasites in all the lesions strongly suggest a good mutual adaptation between host and parasite. This hypothesis is further reinforced at population level with the PIHM analysis. Figure 3 shows that mean abundance of parasites increased steadily with size (as age indicator) of fish (a), whereas variance to mean ratio increased from size class 36-45 to size class 46-55 mm, decreased in size class 36-45 mm and increased again in size class > 65 (b). This pattern clearly differ from that shown by Anderson and Gordon\(^1\), of “convex” or “peaked” curves of parasite abundance, declining concomitantly with the variance to mean ratio in older fish. It is therefore concluded that lesions do not induce significant mortality in the “puyenes” population studied. This result agrees with findings for *Tylodelphys barilochensis*, another native trematode infecting “puyenes” in the metacercaria stage\(^12\). Nevertheless it does not agree with the mortality suggested by results of Revenga\(^13\) for rainbow trout *Oncorhynchus mykiss*, infected by the cestode *Diphyllobothrium dendriticum*, both fish and parasite species being introduced.

The fact that both fish and parasites are native, high parasite abundance (prevalences: 41.4 - 96.7 \%) reported\(^9\), lack of inflammatory reaction in most cases and absence of PIHM, all suggest that the system has coevolved according to a mutualistic model\(^14\).

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**References**


