

Histopathological changes in naturally-infected Chirruh snowtrout, *Schizothorax esocinus* (Heckel), with *Adenoscolex oreini* (Caryophyllidea: Capingentidae)

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Abstract. The aim of this study was to carry out histopathological studies on the intestines of Chirruh snowtrout, *Schizothorax esocinus* (Heckel), naturally infected with the caryophyllidean cestode, *Adenoscolex oreini*. The specimens of *S. esocinus* (25–40 cm) were collected from the Jhelum River, Kashmir, India. Heavily infected fish (> 50 worms per individual host) with only *A. oreini* were chosen for the histopathological investigation. The intestinal tissues with parasites *in situ* were fixed and processed for routine histological investigations. About 12.7% of the snowtrout were found to harbor *A. oreini* infection. With heavy worm burden, the aggregation of worms and increased mucus secretion were noted in infected intestines. The worm scolex lacked a specialized attachment organ and penetrated deeply into the intestinal wall. The pathology induced by this worm in the gut included mucosal damage at the site of attachment and the compression of villi adjacent to the strobila. Intense cellular response induced by the worm at the site of attachment was also observed.

Keywords: *Adenoscolex*, Caryophyllidea, histopathology, *Schizothorax*, scolex glands

The adverse effects of caryophyllidean cestodes on their piscine hosts include compression, damage, and atrophy of epithelium, the production of lesions, the loss of microvilli, the shedding of host tissues, irritation, inflammation, and the perforation of the gut mucosa, and subsequent mortality is well documented (see references in Morley and Hoole 1995). The degree of pathology in the gut is closely related to the morphology of the scolex and the mode of attachment (Mackiewicz et al. 1972, Hayunga 1979). Histopathology due to caryophyllidean cestode infections in fishes has been reported by Ahmed and Sanaullah (1979) for *Lytocestus indicus* and *L. parvulus* in catfish, *Clarias batrachus* (L); Hayunga (1979) for *Glaridacris catostomi*, *G. laruei*, and *Huntrella nodulosa* in white sucker, *Catostomus commersoni* (Lacepède); Karanis and Taraschewski (1993) for *Caryophyllaeus laticeps* in bream, *Abramis brama* (L.), chub, *Leuciscus cephalus* (L.), and roach, *Rutilus rutilus* (L.); Morley and Hoole (1995) for *Khawia sinensis* in carp, *Cyprinus carpio* (L.), and Mackiewicz et al. (1972) for 15 caryophyllid species in carp. However, there are no reports on histopathology from *Adenoscolex oreini* (Fotedar 1958) infection in Chirruh snowtrout, *Schizothorax esocinus* (Heckel). The present study was undertaken to determine the histopathology resulting from natural infection by this parasite in Chirruh snowtrout.

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Intestines with attached parasites were removed from naturally-infected, freshly-killed *S. esocinus* (25-40 cm) captured in the Jhelum River, Kashmir, India and fixed in Bouin's fluid for 8 h. After thorough washing with distilled water, dehydration was carried out in a graded series of ethanol before embedding in paraffin (melting point 58°C). The specimens were serially sectioned at about 7 µm on a rotary microtome, stained with the haematoxylin-eosin and observed under a light microscope (Getner, India, GTR 15416). In the present study, only fish heavily infected (> 50 worms per individual host) with *A. oreini* were selected. For comparative purposes, the intestinal tissues from uninfected fish were also processed simultaneously. The detached adult worms were fixed, stained with Borax carmine, and mounted on glass slides to study the structure of the scolex.

During our studies on the fish of the Kashmir Valley, India, Chirruh snowtrout was found to be frequently parasitized by the caryophyllid *A. oreini*. This parasite was found in clusters similar to those reported for *K. sinensis* (Morley and Hoole 1995). The lumen of the intestine was almost blocked due to the aggregation of the worms, which possibly affected the movement of food as was suggested for pseudophyllidean cestodes (Shostak and Dick 1986). Increased deposition of mucous was noted in the lumen of infected in comparison to uninfected intestines, which is a common phenomenon with intestinal helminth parasites and which may provide defense to the host. It was also reported that mucus forms in a continuous layer of variable thickness in response to parasitic infection and acts as a physical barrier against microorganisms, parasites, and their toxins (see references in Bosi et al. 2005).

The scolex of *A. oreini* was found to be conical in shape and lacking in specialized attachment organs (Fig. 1a). The histological examination of the infected intestines revealed damage to and atrophy of the mucosal epithelium at the point of scolex contact (Fig. 1b), which could have resulted from the pressure exerted by the scolex during penetration (Jara and Szerow 1981). Due to the absence of attachment organs, the *A. oreini* scolex penetrates deep into the

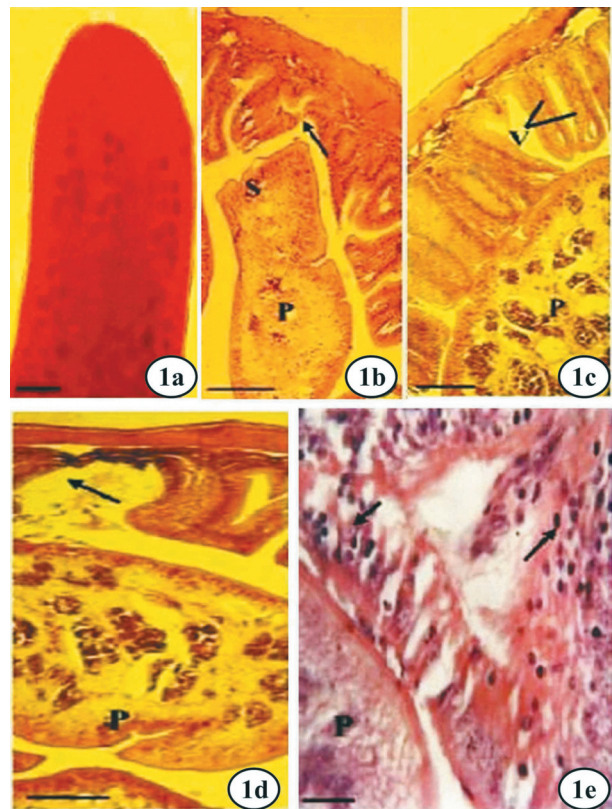


Figure 1. *A. oreini* scolex lacking an attachment organ (Scale bar = 50 µm) (a). Photomicrographs depict *A. oreini* damage to intestinal wall of Chirruh snowtrout, *Schizothorax esocinus*. Note damage of mucosal epithelium (arrow) at attachment site of scolex (S), compression of villi (V) and deep lesion in intestinal wall adjacent to parasite strobila (P) (Scale bar = 50 µm) (b-d). Intense cellular response (arrows) at parasite attachment site (P) (Scale bar = 10 µm) (e).

intestinal wall to maintain helminth position in the gut mucosa thus causing more damage. Similarly, the greatest degrees of damage are reported for those caryophyllidean species which lack specialized attachment organs (Mackiewicz et al. 1972, Hayunga 1979). Karanis and Taraschewski (1993) reported that because of the absence of specialized attachment organs, the caryophyllid *C. laticeps* develop intimate contact with host tissues that results in the compression and ultimately the degeneration of epithelial tissues. Damage to gut mucosa at the attachment site of *K. sinensis* in carp has also been reported (Morley and Hoole 1995, Jara and Szerow 1981).

Compression of villi (Fig. 1c) and deep lesions in the intestinal wall (Fig. 1d) were observed adjacent to strobila, which might lead to impaired digestion and

absorption of food, water, and electrolytes as it is suggested that intestinal helminths often provoke structural modification of host tissue and induce alterations in normal intestinal physiology (Hoste 2001). The lesions noted in the intestinal mucosa near the strobila could have been previous sites of attachment rather than irritation caused by the strobila and probably indicated the worm was either detached during fixation or migrated to other locations. Kapustina (1978) also noted damage to intestinal mucosa adjacent to the strobila of *K. sinensis*, which was attributed to cestode feeding strategies, migration of the parasite in the gut, and previous sites of attachment. In the present study, intense cellular response to the parasite was evident, which is a normal non-specific reaction of fish against parasites (Rowley et al. 1988). Leukocyte infiltration has also been reported in carp infected with *K. sinensis* (Morley and Hoole 1995) and in bream, roach, and chub infected with *C. laticeps* (Karanis and Taraschewski 1993). Although intense cellular response against caryophyllid cestodes has been noted previously, its role has not yet been investigated. Thus, future studies are required to assess the role of cellular and humoral components against this parasite.

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Streszczenie

Zmiany histopatologiczne wywołane przez *Adenoscolex oreini* (Caryophyllidea: Capingentidae) u *Schizothorax esocinus* (Heckel)

Celem pracy było określenie zmian histopatologicznych wywołanych przez *Adenoscolex oreini* (Caryophyllidea: Capingentidae) u *Schizothorax esocinus* (Heckel). Naturalnie zainfekowane osobniki *S. esocinus* (25-40 cm) zostały złowione w rzece Jhelum (Kaszmir, Indie). Do badań histopatologicznych wybrano ryby, u których intensywność zarażenia

przekraczała 50 osobników. Skoleks *A. oreini* nie posiada wyspecjalizowanych organów wykorzystywanych do przytwierdzenia się do ścian jelita. W efekcie infekcji *A. oreini* zaobserwowano głęboką penetrację ścian jelita i uszkodzenie nabłonka. Obserwowany stan zapalny był odpowiedzią organizmu na infekcję wywołaną przez *A. oreini*.