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Mortality in Atlantic Salmon (*Salmo salar*) Associated with Trichodinid Ciliates

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ABSTRACT: A protozoan infection (*Trichodina truttae*) was identified in captive Atlantic salmon (*Salmo salar*) kelts that died in spring of 1988 and 1989. Fish with intense infections showed signs of listlessness, erratic swimming and inappetence. The infection induced excessive mucus secretion, epithelial sloughing and lesions that probably permitted entry of opportunistic bacteria which eventually caused ulcers and death. A seawater bath for 30 min each week for 4 wk effectively controlled the parasite.

Key words: *Trichodina truttae*, ciliate, ectoparasite, *Salmo salar*, Atlantic salmon, mortality.

Species of *Trichodina* (Ciliophora: Peritrichida) are ectoparasitic ciliates that occur on the skin, fins and gills of fish and occasionally cause disease in cultured freshwater fish (Hoffman, 1967). Several species have been reported from salmonids and one (*Trichodina truttae*), noted in hatcheries, is pathogenic to Pacific salmon (*Oncorhynchus* spp.) and steelhead trout (*Salmo gairdneri*) in western Canada (Hoskins et al., 1976; Arthur and Margolis, 1984). Recently, a number of Atlantic salmon (*Salmo salar*) kelts which had originated from a brook (Noel Paul's) in Newfoundland, located in eastern Canada, died after being held for a period of about 8 mo. Examination of smears from the skin and gills revealed intense trichodinid infections. The purpose of this communication is to report (1) the occurrence of this ciliate in a salmonid fish, (2) the signs associated with the infection and (3) an effective method of control.

Thirty six adult Atlantic salmon kelts, about 53 cm in length, were obtained in central Newfoundland (47°52′N, 56°20′W) in October 1986 and held in the laboratory (Ocean Sciences Centre, Memorial University of Newfoundland, St. John's, Newfoundland, Canada A1C 5S7) in self-cleaning, flow-through aquaria (capacity 1,800 L, depth 100 cm, flow rate 8L/min). They were fed Atlantic silversides (*Menidia menidia*) impregnated with a vitamin supplement (Johnston et al., 1987) ad libitum thrice weekly. Water temperatures varied in tanks from 0 to 4 C in winter (January to March) and 6 to 16 C for the rest of the year.

Skin and gill smears from live or dead fish were prepared for microscopic examination by scraping with a 24 × 24 mm coverslip. Prevalence and intensities, estimated by means of a Neubauer hemocytometer, of the ciliate infection were determined from mucus obtained from the trunk region below the lateral line. Some air dried smears were stained using Klein's silver impregnation technique as modified by Lom and Laird (1969); others were stained with Heidenhein's iron hematoxylin. At necropsy, affected skin was fixed in buffered formalin and processed by conventional histological methods; 6 μm thick sections were cut and stained with hematoxylin and eosin.

About 1.5 yr following capture, a decrease in food intake in some individual salmon occurred. This was recognized by a loss of weight as indicated by their slender shape. It occurred in spring (May 1988), concomitant with the rise of the water temperature. There was evidence of partial rotation of their bodies while swimming and a tendency to rub themselves against the walls of the aquarium. Some fish had whitish pustules at intervals along the body, others appeared to be coated with an opaque film of heavy mucus, and in a few the skin was eroded and open

hemorrhagic lesions were apparent. Affected fish also were listless.

Microscopic examination of the whitish pustules and opaque films of mucus from nine salmon which died revealed numerous trichodinids. The latter also were observed on salmon with hemorrhagic lesions and were accompanied by large numbers of rod-shaped bacteria. Sixteen of 27 live salmon examined in 1988 were infected with trichodinids. One had an estimated infection of 1×10^6 organisms/ml; in three others the intensity exceeded 1×10^5/ml. In all cases the intensity of the infection was consistently greater on the skin than on the gills. In spring 1989, trichodinids were observed in only 12 of 15 salmon that died.

Microscopic lesions were compatible with the gross lesions. Epithelial hyperplasia accompanied by cellular sloughing and excessive mucus secretion was observed in salmon with intense trichodinid infections. Purulent and ulcerated lesions ($\leq$0.5 cm in diameter) on the trunk varied from colorless to a pale pink and were accompanied by an inflammatory response. These lesions also had bacterial infections in some fish. There was no evidence of trichodinids in the subcutaneous layers where the epithelium was eroded and had become infected with bacteria.

A seawater bath appeared to be an effective method of control for the trichodinid infection. The volume of freshwater in the aquarium was reduced to 50% and running, ambient seawater was introduced at a flow rate of about 5 L/min for 0.5 hr before resuming the flow of freshwater. In 1988 this was performed weekly for 4 wk. After this period, none of the remaining 14 fish had the trichodinid infection on the skin or gills. On reappearance of the infection in 1989 the treatment was repeated, with no subsequent appearance of the parasite.

The presence of trichodinids, tentatively identified at *T. truttae*, in a native population of Atlantic salmon suggests that the parasite is endemic on the island of Newfoundland. This also is the first report of the ciliate in *S. salar* in eastern Canada; to date, this is the only fish host in which the parasite has been observed. It appears unlikely that the parasite was the primary cause of death in the salmon because some fish had low grade infections at necropsy. The presence of ulcers and subcutaneous bacteria supports the view that unidentified opportunistic bacterial infections might have been the underlying cause of mortality. I suggest that stress induced by confinement and reduced food intake during winter might have impaired host resistance, mediated through elevated cortisol levels, to the trichodinid infections. Thus, the trichodinids could have increased in intensity and caused epithelial sloughing and excessive mucus secretion. Epithelial erosion permitted entry of the opportunistic bacteria which ultimately caused mortality. This scenario of corticosteroid-induced stress having a suppressive effect on the defense systems and subsequent injury in fish by trichodinids has been reported previously (Lom, 1973; Pickering and Pottinger, 1989).

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