NON-SALMONID FISH INHABITING A HATCHERY WATER SUPPLY ENZOOTIC FOR PROLIFERATIVE KIDNEY DISEASE INFECTED WITH ONE MYXOSPOREAN, Myxobilatus spp.

By E. MacConnel and C. Smith

Proliferative kidney disease (PKD), caused by an unclassified myxosporean (PKX), is a serious disease of salmonids. Although partial spore development occurs to a limited extent in fish infected with PKX, complete sporulation has not been observed and precise taxonomic status of PKX remains uncertain. The severe inflammation seen in infected fish, unusual for most myxosporean infections, suggest salmonid species are aberrant hosts for PKX. Recently, there has been considerable speculation that PKX belongs to the genus Sphaerospora (Feist, 1988; Hedrick et al., 1986). Sphaerospores have been found in tu chub (Gila bicolor) inhabiting the water supply of PKD-infected Hot Creek State Hatchery, California and stickleback (Gasterosteus aculeatus) from Quinault Lake, Washington, which is the water supply of a PKD-infected steelhead hatchery (Hedrick et al., 1988). Similar characteristics shared between PKX and Sphaerospora have been described by Kent and Hedrick (1986). Odening et al. (1988) reported the simultaneous occurrence in rainbow trout of PKX and Sphaerospora spp., and unidentified myxosporean forms in swim bladder and intestinal walls. This report presents evidence that disputes the implication that PKD is caused by cross transmission of Sphaerospora spp.

The first reported outbreak of PKD in North America was at the Hegerman, Idaho, State Fish Hatchery (SFH), rainbow trout (Onchorhyncus mykiss)(Smith et al., 1984). PKD was also diagnosed during an extensive examination of wild rainbow trout inhabiting Riley Creek, one hatchery water supply. At that time, intraluminal sporogenic forms similar to those report-

Figure 1. Myxosporean presporogenic forms (arrows) in kidney tubule (TU) lumen of sculpin. (1000×).
Figure 2. Presporogonic forms (arrows) and spores (arrowhead) of *Myxobilatus* spp. in kidney tubule (TU) in bow trout. (Giemsa, 1000×)

ed to occur in rainbow trout infected with PKX (Kent and Hedrick, 1986) were observed in at least one fish.

Riley Creek originates from springs about 2 km above the hatchery. Water temperature is constant at 15°C, water hardness is about 140 mg/l and pH ranges from 7.3 to 7.6. Only two fish species inhabit Riley Creek above the hatchery, rainbow trout and Shoshone sculpin (*Cottus greenei*). PKD seasonally occurs in May to September at the Hagerman SFH. Rainbow trout and sculpins were collected for histological examination on two occasions in February and March 1986 by electrofishing Riley Creek above the hatchery. Tissue was collected from posterior kidney, preserved in Bouin's fixative, transferred to 70% ethanol, embedded in paraffin and sectioned at 5 μm. Sections were stained with hematoxylin-eosin, Giemsa, and hematoxylin-periodic acid-Schiffs. A total of 14 subyearling and yearling rainbow trout and 57 sculpins were examined.

Histological sections of several rainbow trout showed small, granulomatous lesions in the kidney interstitium typical of the later stages of PKD. Interstitial intraluminal myxosporeans were not observed. Myxosporean presporogonic forms and spores were, however, found in kidney tubule lumina of 18 sculpin examined (Fig. 1). Characteristic of *Myxobilatus*, the elongate spores with caudal projections contained two paddle-shaped polar capsules located laterally at one pole (Fig. 2). Sphaerospores were not found in rainbow trout or sculpin from Riley Creek. One other parasite, a coccidian, was observed in liver from two sculpins.

Although *Sphaerospora* spp. has been implicated as the possible etiologic agent of PKD, cross-transmission experiments: conducted by Rafferty (1985) with *Sphaerospora* infected roach (*Rutilus rutilus*) and carp (*Cyprinus carpio*), by Hedrick (personal communication) with tushub and stickleback, and by Feist (1988) with stickleback have been inconclusive. Using the indirect immuno-fluo-
escent test, Rafferty and Mulcahy (1988) demonstrated no recognition of *Sphaerospora renicola* by anti-PKX serum in heavily infected tissues from brown trout, *Salmo trutta*). Feeding homogenized preparations of infected kidneys to healthy fish, and holding disease-free fish in aquaria with feces from infected fish, or with infected fish have been unsuccessful in transmitting PKX (Ferguson and Ball, 1979; D'Silva *et al.*, 1984). Similar to most myxosporeans, there is no evidence of fish transmission of PKX.

The lack of sphaerospores in either species of fish inhabiting Riley Creek suggests that the etiologic agent of PKD is not transmitted directly from fish harboring spores of the genus *Sphaerospora*. Chilmonczyk *et al.* (1989) reported the experimental infection of PKX-free rainbow trout by exposure to contaminated sediments in an indoor recirculating unit. Perhaps the involvement of an alternate life stage in an invertebrate host demonstrated by Wolf and Markiw (1984) for *Myxobolus cerebralis* may also be true for PKX.

**Summary**

Recent speculation that the causative agent of PKD is *Sphaerospora* spp., commonly found in non-salmonid species inhabiting water enzootic for PKD, is disputed. Examination of non-salmonids inhabiting hatchery water supply enzootic for PKD revealed the presence of only one myxosporean, *Myxobilatus* spp.

**References**


**Authors' address**

U.S. Fish and Wildlife Service, Fish Technology Center, 4050 Bridger Canyon Road, Bozeman, Montana 59715, USA.