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The Development of the PKX Myxosporean, the Causative Agent of Proliferative Kidney Disease, in Rainbow Trout Salmo gairdneri Richardson

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The Development of the PKX Myxosporean, the Causative Agent of Proliferative Kidney Disease, in Rainbow Trout Salmo gairdneri Richardson.

## Abstract

The endogenous development of PKX, the causative agent of proliferative kidney disease, is described in rainbow trout, <u>Salmo gairdneri</u>, by light and electron microscopy. Parasites multiplied by endogeny, binary fission and possibly plasmotomy in the renal interstitium, and elicited an interstitial nephritis. As the disease progressed parasites were found in the lumens of the tubules, and these represented daughter cells released from PKX parasites that had migrated to the tubules. Internal sporoblasts formed within these daughter cells (enveloping cells). The internal sporoblasts contained up to six daughter cells and some sporoblasts organized into spores with two spherical polar capsules. Although the spores persisted for several months after the interstitial PKX and associated inflammation had subsided, they did not complete their development as indicated by the fact that they remained within the enveloping cell and did not form valves.

The relationship of this spore to the vegetative stages was studied further by a transmission experiment. The myxosporean forms were only observed in the kidney tubules, while typical PKX also occurred in the blood and spleen. Parasite—free fish injected with the blood and spleen of infected fish developed both forms of the parasite, which further substantiates that PKX is the prespore form of the intraluminal myxosporean.

Because only incomplete spores were observed, the more precise taxonomic status of PKX was not determined. However, intraluminal stages of PKX show similarities to the genera Sphaerospora, Mitraspora and Parvicapsula. Furthermore, interstitial stages of PKX resemble the early stages of Sphaerospora.

The incomplete spore development and severe inflammatory response to PKX indicate that salmonids may be abnormal hosts. Fish were immunosuppressed with cortisol implants, and they exhibited greater densities of interstitial and intraluminal PKX but less interstitial hypercellularity. However, spores did not develop further than those observed in natural epizootics. This study supports the hypothesis that PKX is an early form of a myxosporean and that salmonids are abnormal hosts. A non-salmonid fish may be the primary host for PKX in which sporulation is completed.

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