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## Introduction

Proliferative kidney disease (PKD) in salmonid fishes is caused by a poorly understood protozoan, the vegetative stages of which are known as PKX cells. The organism was initially believed to be an amoeba (phylum Sarcomastigophora) by Plehn (1924) and later by Ghittino et al. (1977) and Ferguson and Needham (1978). Similarities between PKX and oyster pathogens of the genus *Marteilia* led Seagrave et al. (1980) to suspect that the parasite was instead a haplosporidan (phylum Acetospora). More recent studies by Hedrick et al. (1984) and Kent and Hedrick (1985*a*, *b*) indicated that the PKX cells are presporogonic forms of a myxosporidan (phylum Myxozoa). Kent and Hedrick (1986) showed that polar capsule formation occurs but that the spores fail to fully mature in the salmonid host.

Although PKD has been recognized as a major problem among farm-reared trout in Europe for many years, it was not detected in North America until the early 1980's. Of major concern has been the appearance of the disease among Pacific salmon in California, Washington, and British Columbia. Whether the increase in prevalence reflects a spread of the disease or an improvement in its recognition (Hedrick et al. 1985*a*) has yet to be determined.

## Geographic Distribution

Proliferative kidney disease (PKD) has been reported from salmonid fishes in several countries in Europe, including Italy, France, Germany, England, Wales, Scotland, Ireland, Norway, Sweden, and Denmark (Clifton-Hadley et al. 1984*a*). In North America, it was first reported by Smith et al. (1982) and Smith et al. (1984) from rainbow trout (*Salmo gairdneri*) at the Hagerman State Hatchery in Idaho (Fig. 1). Later reports by Hedrick et al. (1984), Hoskins (1985), and Hoskins and Kieser (1986) demonstrated the first known occurrences of PKD among certain Pacific salmon.

At least three locations in British Columbia (one on the mainland), have been involved, and diagnoses of PKD have been confirmed at eight locations in California. Recently the disease was identified in the State of Washington (J. Morrison, personal communication) in steelhead trout and Pacific salmon.

## Host Range

The disease is principally confined to salmonid fishes, and the protozoan PKX has been found in only one species outside the family Salmonidae: Seagrave et al. (1981) reported finding PKX cells in the interstitium of the kidney of northern pike (*Esox lucius*). The most commonly infected species is the rainbow trout, although the disease has been frequently encountered in chinook salmon (*Oncorhynchus tshawytscha*) and coho salmon (*O. kisutch*) in North America (Hedrick et al. 1984; Hoskins and Kieser 1986). Outbreaks in Atlantic salmon (*Salmo salar*) and brown trout (*S. trutta*) have been reported in Europe (Ellis et al. 1985). Other potential hosts are the grayling, *Thymallus thymallus* (Seagrave et al. 1981), the brook trout, *Salvelinus fontinalis* (Plehn 1924; however, no other observations have been made and this report may be a translation error), and the Artic char, *S. alpinus* (Bucke et al. 1985).

## Effects of Temperature and Seasonal Occurrence

Most epizootics of PKD occur at temperatures of 15 °C and higher; however, natural infections have been reported at 13 °C (Hedrick et al. 1984) and experimentally induced infections at 7 °C (Rafferty 1986). Ferguson (1981) compared the progress of PKD in fish exposed to the infective stage and then transferred to pathogen-free water in the laboratory, at 5-7 and 16 °C. The disease manifested itself at 16 °C but not at 5-7 °C. Foott et al. (1986) showed that fish exposed to the infective stage at temperatures as low as 10 °C and then transferred to water at 18 °C developed typical PKD. Clifton-Hadley et al. (1985) found that the disease was slower in onset and less severe among rainbow trout held at 12 °C and lower, following infection. Clearly, more studies of the effects of temperature on the initial and later stages of infection are needed.


**Fig. 1.** Locations in North America where proliferative kidney disease (PKD) has been found in salmonid fishes.

## Immunity to Reinfection

An epizootiological study at a fish farm in North Ireland by Ferguson and Ball (1979) showed that survivors of a summer PKD outbreak were resistant to infection during the next year, but that yearling fish not previously exposed were susceptible. Foott et al. (1986) also demonstrated that fish surviving initial infections became strongly resistant to reinfection. The reason for this resistance has yet to be determined, but it most likely involves a strong humoral response (as suggested by Olsen and Jorgensen 1985 and Klontz et al. 1985), and cellular response to infection.

## Diagnosis and Identification

### *History and Environmental Conditions*

In any diagnosis of a pathogen, a history of the fish stock and the geographical location should be considered. At nearly all locations where

salmonids have contracted PKD, the episodes have recurred annually, often with a marked seasonality. As in myxosporidans such as *Ceratomyxa shasta* and *Myxosoma cerebralis*, the presence of the infective stage of PKD can be established by exposing sentinel fish; this exposure enables a determination of waters that are enzootic for the pathogen.

The seasonality of PKD may also be useful in diagnosis. The disease occurs most frequently at temperatures of 15.6°C or higher, which usually occur from midsummer through early fall (June to October).

The disease is most often seen at hatcheries that use impounded river water and seldom at those using spring water, and primarily in underyearling trout and salmon. Stocks, sites, and environmental conditions should therefore be considered by fish health specialists in making a diagnosis of PKD.

## *External Signs of PKD*

The signs shown by affected fish include darkening body color, distended abdomen due to ascites, pale gills indicating anemia (Hoffman and Lommel 1984), pronounced lateral body swelling, and bilateral exophthalmia. These signs are unfortunately similar to those caused by other chronic diseases that impair kidney function, and should not be relied on even in a presumptive diagnosis of PKD.

## *Internal Signs of PKD*

Enlargement of the kidney and spleen are the most notable internal signs (Fig. 2). The kidney may be grayish throughout or mottled, and is markedly swollen (sometimes more pronounced posteriorly); in severe cases the capsule may have a folded or corrugated appearance. Ascites may be present or absent and is usually clear.

These signs can be confused with kidney hypertrophies caused by *Renibacterium salmoninarum* and *Ichthyophonus hoferi*. However, PKD can be readily distinguished from the other conditions by histological and other microscopical studies.

## *Microscopic Examination*

Three aids to the diagnosis of PKD are light microscopy, histological examination, and detection of later stages of PKX in the kidneys. Wet mounts made from affected kidney tissues and examined by

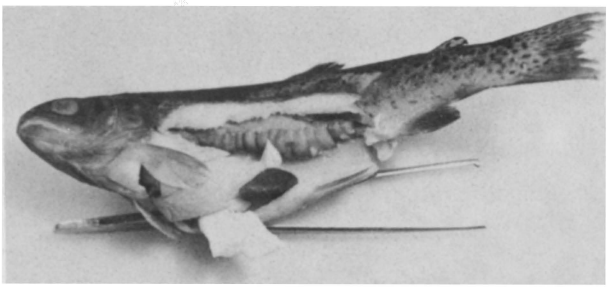

**Fig. 2.** Rainbow trout (preserved in formalin) with proliferative kidney disease, showing grossly swollen kidney and hypertrophy of the spleen.

brightfield or phase microscopy can be used for the diagnosis (Fig. 3*A*). However, adequate numbers of PKX, as well as experience in identifying the forms, are required to enable one to distinguish trophozoites of PKX from host macrophages—particularly when the macrophages are laden with cellular debris. Imprints of affected kidney tissues stained with Giemsa or Leishman-Giemsa (Clifton-Hadley et al. 1983; Klontz and Chacko 1983) provide a useful tool for detection of PKX (Fig. 3*B*). As in wet mounts, however, confusion with macrophages sometimes occurs, particularly when the staining between cytoplasm and nuclear material is not differential. When both imprints and wet mounts are used, the attachment of macrophages to PKX aids in distinguishing the parasite from other cells present in the interstitium of the kidney. It should be stressed that fresh kidney tissue is needed because degenerative changes (such as extensive vacuolation) in the normal cells may interfere with or preclude the diagnosis of PKX.

Observation of the PKX parasite in histological section is currently the method of definitive diagnosis of PKD (Fig. 3*C*). Sections from affected kidney tissues provide an opportunity to observe the parasite and the accompanying cellular response it provokes in the host. The lesions are characterized by areas of diffuse granulomatous reaction that often surround one or more PKX parasites. Early in infection there may be a hyperplasia of the hematopoietic cells, but as the disease progresses, these cells are replaced by macrophages and lymphocytes. Degeneration and subsequent loss of the kidney tubules are evident. The lesions may persist for several weeks after the PKX parasites have been eliminated from the fish. These lesions, in combina-

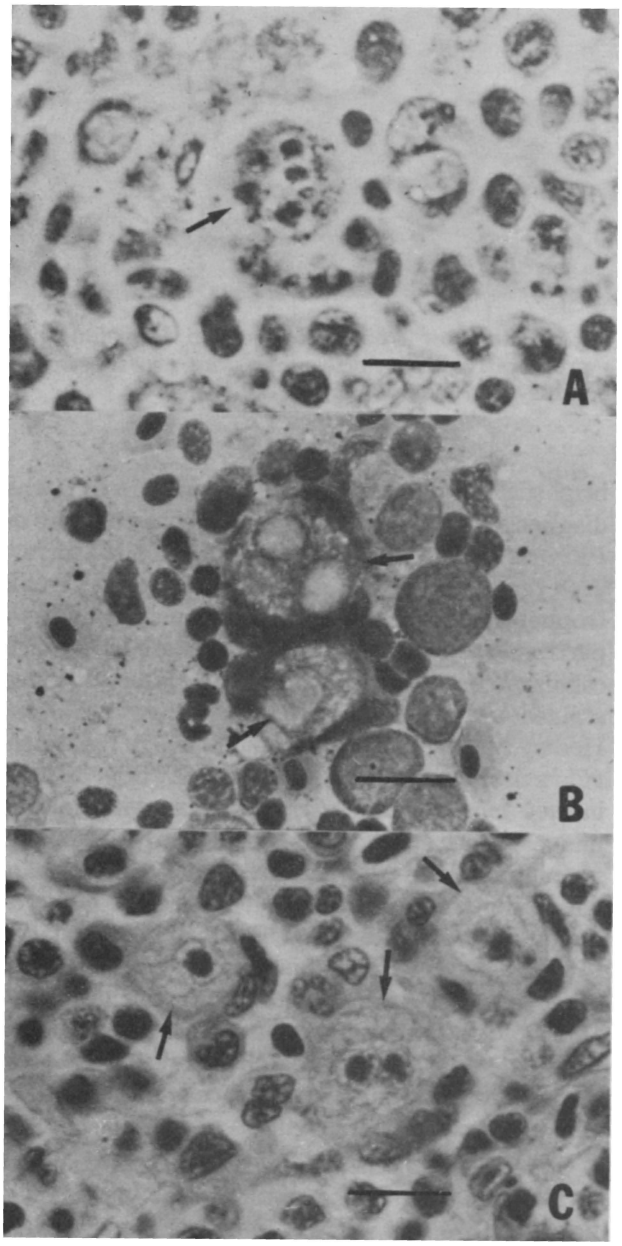


**Fig. 3.** Light micrographs of PKX, the causative agent of proliferative kidney disease (PKD). (*A*) phase contrast wet mount, (*B*) Leishman-Giemsa stained imprint, and (*C*) paraffin section stained with hematoxylin and eosin. Bars = 10 μm. Arrows indicate PKX cells.

tion with the observation of later stages of the PKX parasite, can also be used to demonstrate late infections or recent convalescence from PKD.

As described by Kent and Hedrick (1985*b*), Kent (1985), and Kent and Hedrick (1986), late developmental stages of the PKX myxosporidan have been identified in the lumens of the kidney tubules. They may persist for several months after recovery from clinical PKD and can therefore be useful indicators of previous infection (Figs. 4 and 5). Although the early sporogonic stages can be confused with other myxosporidans, certain characteristics are unique for PKX. Spores develop in pseudoplasmodia within the lumens of kidney tubules (Fig. 4*B–E*), are monosporous, and contain two anteriorly located spherical polar capsules about 2 μm in diameter (Fig. 4*C,D* and Fig. 5*B*). No distinct valve formation has been observed. In contrast, other known myxosporidans, such as *Parvicapsula* and *Myxidium*, found in the lumens of the kidney tubules of salmonid fishes, form true plasmodia and are polysporous (i.e., many spores develop within a pansporoblast); also, valvogenesis is evident. In *Parvicapsula*, sporogenesis occurs in the epithelium of the tubules as well. Observation of these unique spores of PKX therefore considerably extends the period in which PKD can be diagnosed.

## Mode of Transmission

Exposure to the infective stage in the water is the natural mode of transmission for the parasite that causes PKD. Exposures as short as 3 days have resulted in infection (Foott et al. 1986). Artificial transmission has been effected by injections into parasite-free fish of homogenates made from kidney (D'Silva et al. 1984; Clifton-Hadley et al. 1984), spleen (Kent and Hedrick 1985*a*), or blood (Kent and Hedrick 1985*a*) from infected fish. Feeding of infected tissue (Ghittino et al. 1977) or cohabitation with uninfected fish has failed to demonstrate fish-to-fish transmission.

## Prevention and Control

Infections with PKX have not yet been controlled by any chemotherapeutics tested (Bucke et al. 1981), and in this respect represent the same problem as do infections with other myxosporidans and viral pathogens. Avoidance of contact between the pathogen and the fish has been the most successful method of ameliorating the effects of PKD in hatchery-reared salmonids. Ferguson and Ball (1979) tested a stocking strategy that involved moving fish into waters (enzootic for PKD) in fall, when neither

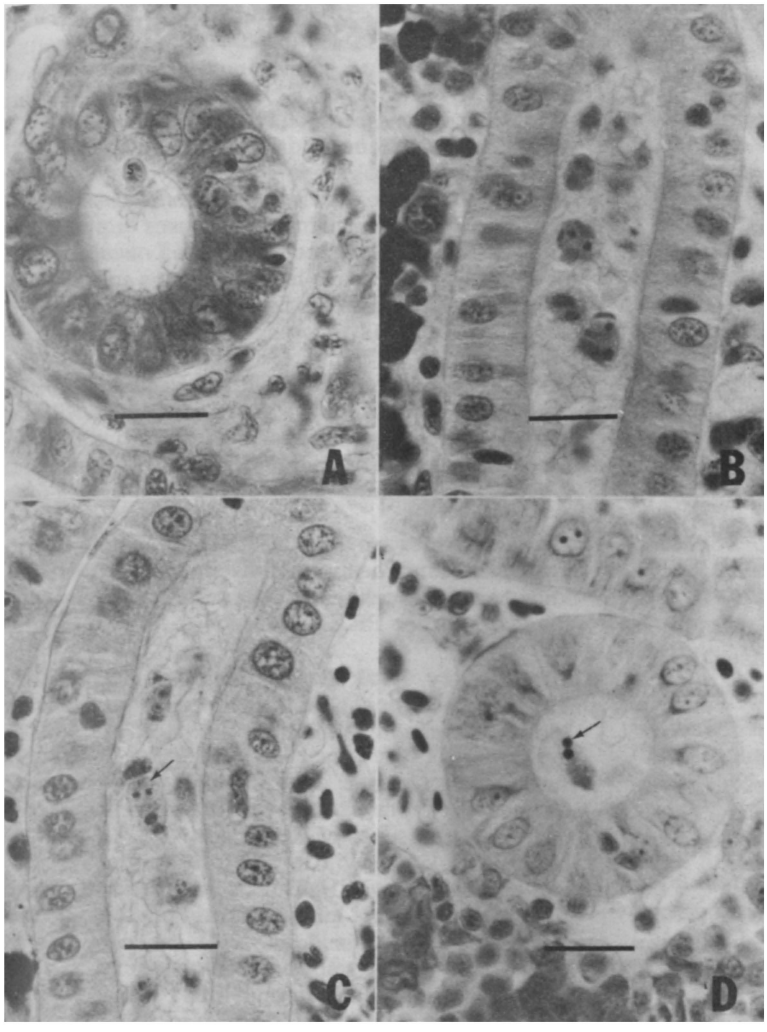


**Fig. 4.** Later developmental stages of PKX (presporogonic) in the kidney tubules of a rainbow trout recovering from proliferative kidney disease. (*A*) PKX between epithelial cells of tubule, (*B*) early presporogonic stages that have reached the lumen of the tubule, (*C*) development of polar capsules (arrows), and (*D*) most fully developed spores with polar capsules (arrows) but still without distinct valve formation. All stages are from paraffin sections; (*A-C*) stained with hematoxylin and eosin, and (*D*) with giemsa. Bars = 10 μm.

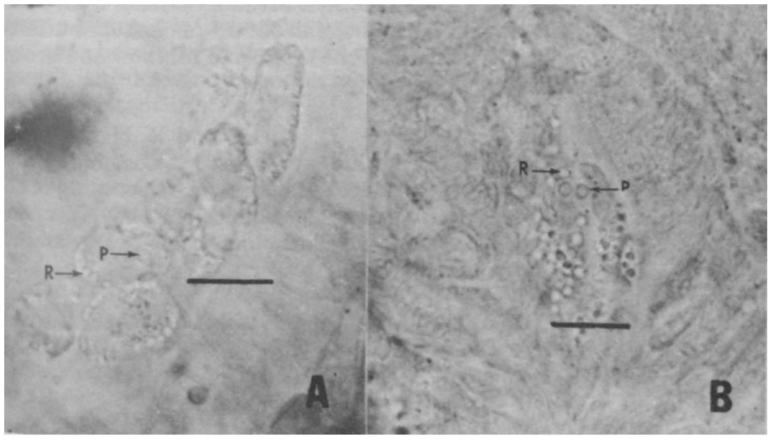


**Fig. 5.** Wet mounts made from kidney tissues of rainbow trout recovering from PKD, showing (*A*) later stages of PKX trophozoites with refractile granules (*R*), and (*B*) spore with polar capsules (*P*) within the lumens of the tubules. Bar = 10 μm.

water temperature nor perhaps abundance of the infective stage was conducive to infection. The fish then showed a manageable involvement the following year, although some infection and mortality occurred.

Reduction in temperature during expected times of peak occurrence of the disease has also been employed when possible (Hoskins and Kieser 1986). Although this treatment may not prevent infection, it seems to lessen the effects of the disease by reducing complications caused by both environmental and secondary pathogens.

Disinfection of the water supply to remove the infective stage of the parasite has shown promise. Observations made at Mad River (California) Hatchery in 1983 indicated the potential efficacy of ultraviolet treatment of the water to prevent PKD. Coho salmon held in the treated water were apparently completely protected, whereas other groups of coho salmon, steelhead, and chinook salmon in untreated water contracted the disease (R. P. Hedrick, unpublished data). Ultraviolet treatment of water effectively controls other myxosporidan pathogens such as *Myxosoma cerebralis* (Hoffman 1975) and *Ceratomyxa shasta* (Sanders et al. 1972).

The potential spread of the disease by the transfer of actively infected fish has not been thoroughly examined. Although the salmonid may be an aberrant host in which PKX fails to develop, a cycle of transmission of PKX might become established in new watersheds if the appropriate host is infected by contact with the parasite (by way of the water or the salmonid).

## Acknowledgments

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## Annotated Bibilography

Bucke, D., D. McGregor, E. B. Hudson, and P. Scott. 1981. Control measures fail to stop the spread of PKD. Fish Farmer 4:25.

A description of research at the Fish Disease Laboratory in Weymouth, U.K., on the causes or prevention of PKD. The pathogen causing PKX was shown to have affinities to certain haplosporidans and could not be controlled by tested antiprotozoal and antibacterial compounds.

Bucke, D., S. Feist, and R. S. Clifton-Hadley. 1985. The occurrence of proliferative kidney disease in wild fish stocks. Proceeding of the second international conference of the European Association of Fish Pathologists. 2-5 September, Montpellier, France.

PKX, the causative agent of PKD, was found in grayling, pike, and roach in addition to the previously known occurrences in salmonids. Artic char were also found to harbor PKX cells.

Clifton-Hadley, R. S., R. H. Richards, and D. Bucke. 1983. Method for the rapid diagnosis of proliferative kidney disease in salmonids. Vet. Rec. 112:609.

Impression smears stained with May-Grünwald-Giemsa were found to provide rapid means of diagnosing the presence of PKX cells.

Clifton-Hadley, R. S., D. Bucke, and R. H. Richards. 1984*a*. Proliferative kidney disease of salmonid fish: a review. J. Fish Dis. 7:363-377.

The most comprehensive review of PKD as it occurs in Europe.

Clifton-Hadley, R. S., R. H. Richards, and D. Bucke. 1984*b*. Experimental transmission of proliferative kidney disease—preliminary report. Vet. Rec. 114:90.

Intraperitoneal injections of kidney tissues from PKD-infected fish, homogenized in phosphate-buffered saline, transmitted the disease to rainbow and brown trout.

Clifton-Hadley, R. S., D. Bucke, and R. H. Richards. 1985. Proliferative kidney disease: its possible control by water temperature manipulation. Proceeding of the second international conference of the European Association of Fish Pathologists. 2-5 September, Montpellier, France.

A preliminary report on the effect of temperature on development of PKD.

D'Silva, J., M. F. Mulcahy, and P. DeKinkelin. 1984. Experimental transmission of proliferative kidney disease in rainbow trout, *Salmo gairdneri* Richardson. J. Fish Dis. 7:235-239.

The disease was found to be transmitted by placing trout in water containing the infectious stage or by intraperitoneal injections of PKX cells. No transmission resulted from feeding infected tissues, or by way of water from aquaria or feces from infected fish.

Ellis, A. E., A. H. McVicar, and A. L. S. Munro. 1985. Proliferative kidney disease in brown trout *Salmo trutta* L. and Atlantic salmon *S. salar* L. parr; histopathological and epidemiological observations. J. Fish Dis. 8:197-208.

A thorough description of the disease in Atlantic salmon and brown trout in Scotland.

Ferguson, H. W. 1981. The effects of water temperature on the development of proliferative kidney disease in rainbow trout, *Salmo gairdneri* Richardson. J. Fish Dis. 4:175-177.

Low or declining water temperatures were found to speed the recovery or inhibit the onset of PKD.

Ferguson, H. W., and E. A. Needham. 1978. Proliferative kidney disease in rainbow trout *Salmo gairdneri* Richardson. J. Fish Dis. 1:91-108.

Detailed account of PKD outbreaks in North Ireland. Excellent description of the parasite and kidney lesions. Although the authors speculated that the agent was an amoeba, they also indicated the possibility of a myxosporidan etiology.

Ferguson, H. W., and H. J. Ball. 1979. Epidemiological aspects of proliferative kidney disease amongst rainbow trout *Salmo gairdneri* Richardson in Northern Ireland. J. Fish Dis. 2:219-225.

This study examined the seasonality of the disease (midsummer to fall) at a farm in North Ireland. Losses were minimized by delaying stocking of fish until July and then harvesting before onset of disease in the next year. Fish surviving infection in year one were found to be resistant in year two.

Foott, J. S., R. Rosemark, and R. P. Hedrick. 1986. Seasonal occurrence of the infectious stage of proliferative kidney diesase (PKD). Bull. Eur. Assoc. Fish Pathol. In press.

Describes the seasonality of PKD and its dependence on the presence of the infective stage, rather than solely on suitable water temperatures.

Ghittino, P., S. Andruetto, and E. Vigliani. 1977. L'amebiasi della trota iridea d'Allevamento. Riv. Ital. Piscic. Ittiop. 12:74-89.

A description of PKD among rainbow trout in Italy. An amoeba cultured from infected trout originally believed to be PKX was later determined to be a contaminant.

Hedrick, R. P., M. L. Kent, J. S. Foott, R. Rosemark, and D. Manzer. 1985*a*. Proliferative kidney disease (PKD) among salmonid fish in California, USA: a second look. Bull. Eur. Assoc. Fish Pathol. 5:36-38.

A retrospective analysis of an unknown kidney disease that has been seen in rainbow trout and chinook salmon in California since at least 1966 indicated that some of the cases were PKD.

Hedrick, R. P., M. L. Kent, R. Rosemark, and D. Manzer. 1984. Occurrence of proliferative kidney disease (PKD) among Pacific salmon and steelhead trout. Bull. Eur. Assoc. Fish Pathol. 4:34-37.

This the first report of PKD in Pacific salmon.

Hedrick, R. P., M. L. Kent, R. Rosemark, and D. Manzer. 1985*b*. Proliferative kidney disease (PKD) in Pacific salmon and steelhead trout. J. World Mar. Soc. 15:318-325.

A detailed account of the first PKD outbreak at the Mad River Hatchery in California.

Hoffman, G. L. 1975. Whirling disease (*Myxosoma cerebralis*): control with ultraviolet irradiation and effect on fish. J. Wildl. Dis. 11:505-507.

Hoffman, R., and R. Lommel. 1984. Haematological studies in proliferative kidney disease (PKD) of rainbow trout, *Salmo gairdneri* Richardson. J. Fish Dis. 7:323-326.

An account of blood analyses of PKD infected fish. The authors concluded that the anemia resulting from infection was chronic and hemolytic rather than hypoplastic.

Hoskins, G. 1985. Brief reports. Am. Fish. Soc., Fish Health Sect. Newsl. 13:8.

First account of Canadian outbreaks.

Hoskins, G., and D. Kieser. 1986. Occurrence of proliferative kidney disease in British Columbia. *In* R. P. Hedrick, ed. Proceedings of a workshop on proliferative kidney disease (PKD) in North America. 25-26 April 1985, University of California, Davis.

A detailed report on the locations, species involved, and management of the disease in Canada.

Kent, M. L. 1985. The development of the PKX myxosporean, the causative agent of proliferative kidney disease, in rainbow trout, *Salmo gairdneri* Richardson. Ph.D. thesis, University of California, Davis.

A study of the sequential development of PKX from the earliest recognizable trophozoites to immature spores in the lumens of the kidney tubules.

Kent, M. L., and R. P. Hedrick. 1985*a*. Transmission of the causative agent of proliferative kidney disease (PKD) with the blood and spleen of infected fish; further evidence that the PKX parasite belongs to the phylum Myxozoa. Bull. Eur. Assoc. Fish Pathol. 5:39-42.

Intraperitoneal injections of blood or spleen containing PKX cells into parasite-free rainbow trout resulted in the appearance of typical PKX cells, PKD, and intraluminal stages with polar capsules in the kidney tubules of the recipient trout.

Kent, M. L., and R. P. Hedrick. 1985*b*. PKX, the causative agent of proliferative kidney disease (PKD) in Pacific salmonid fishes and its affinities with the Myxozoa. J. Protozool. 32:254-260.

The presence of immature spores in fish surviving PKD infections is documented.

Kent, M. L., and R. P. Hedrick. 1986. Development of the PKX Myxosporean in rainbow trout, *Salmo gairdneri*. Dis. Aquat. Organisms 1:169-182.

A light and electron microscopical documentation of the evidence supporting the myxozoan nature of the etiological agent.

Klontz, G. W., and A. J. Chacko. 1983. Methods to detect the organism causing proliferative kidney disease in salmonids. Bull. Eur. Assoc. Fish Pathol. 3:33-36.

Three methods are described for detecting PKX in kidney tissues: staining of fixed impression smears by (1) Leishman-Giemsa and (2) methylene blue, and (3) phase contrast microscopy of fresh tissue mounts.

Klontz, G. W., A. W. Rourke, and W. Eckblad. 1985. The immune response in rainbow trout to a systemic parasitism. Proceeding of the international meeting on fish immunology. 8-12 September 1985, Sandy Hook, New Jersey.

Rainbow trout systemically infected with PKX had decreased hematocrits, hemoglobin, and serum albumin fractions but significant increases in betaglobulins.

Olsen, N. J., and P. E. V. Jorgensen. 1985. Rainbow trout total immunoglobulin: estimate of concentration in serum under various holding conditions. Proceeding of the second international conference of the European Association of Fish Pathologists. 2-5 September, Montpellier, France.

Rainbow trout recovering from PKD were shown to have increased immunoglobulins, presumably directed to the PKX parasite.

Plehn, M. 1924. Praktichum der Fischkrankheiten. Schweizerbart'sche, Stuttgart.

One of the earliest descriptions of PKD; the condition is referred to as "amoeba infection" in rainbow and brook trout.

Rafferty, M. D. 1985. The influence of temperature on experimentally-induced proliferative kidney disease. Ph.D. thesis, National University of Ireland, Cork.

Rainbow trout experimentally infected by intraperitoneal injections of PKX cells were found to develop PKD at 7, 11, and 17 °C, although the disease was later in onset and the PKX parasite more difficult to identify at the lowest temperature.

Sanders, J. E., J. L. Fryer, D. A. Leith, and K. D. Moore. 1972. Control of the infectious protozoan (*Ceratomyxa shasta*) by treating hatchery water supplies. Prog. Fish-Cult. 34:13-17.

Ultraviolet light treatment of water was found to be effective in eliminating the infectious stage of the myxosporidan *Ceratomyxa shasta*.

Seagrave, C. P., D. Bucke, and D. J. Alderman. 1980. Ultrastructure of a haplosporean-like organism: the possible cause of proliferative kidney disease in rainbow trout. J. Fish Biol. 16:453-459.

The authors were led to conclude that PKX was most probably a haplosporidan like the *Marteilia* spp. of oysters and mussels, on the basis of similar developmental stages; the presence of haplosporosomes, multivesicular bodies, striated inclusions, and a similar cell wall; and division by internal cleavage (endogeny).

Seagrave, C. P., D. Bucke, E. B. Hudson, and D. McGregor. 1981. A survey of the prevalence and distribution of proliferative kidney disease (PKD) in England and Wales. J. Fish Dis. 4:437-439.

The seasonal nature of the disease in underyearling rainbow trout in the U.K. is documented, as well as infections in northern pike and grayling.

Smith, C. E., J. K. Morrison, and H. W. Ramsey. 1982. Proliferative kidney disease (PKD): first outbreak in North America. Fish Health News 10:3-5.

First description of PKD in North America, in rainbow trout in Idaho.

Smith, C. E., J. K. Morrison, H. W. Ramsey, and H. W. Ferguson. 1984. Proliferative kidney disease: first reported outbreak in North America. J. Fish Dis. 7:207-216.

A detailed account of PKD in rainbow trout from the Hagerman State Hatchery in Idaho.

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