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PROLIFERATIVE KIDNEY DISEASE OF SALMONIDS

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Proliferative kidney disease (PKD) is a condition of salmonid fishes first recognized in North America following an outbreak of the disease in late 1981 at the Hagerman State Fish Hatchery in Idaho. The disease has been known in Europe and the British Isles for many years where it is recognized as a major problem affecting rainbow trout production, especially in France and Italy (Ferguson and Ball 1979). On severely-affected farms, 100% of the fish may be affected and mortality may approach 50-100%. Although the cause is unknown, it is believed that a protozoan parasite is involved, either an amoeba-like organism (trophozoite?) (Ferguson and Needham 1978) or possibly a haplosporidian protozoan (Seagrave et al. 1980). The potential impact of the disease on North American fish culture is, as yet, undetermined.

SIGNS OF INFECTION

The following description of external and internal signs has been adapted from Ferguson and Needham (1978).

External Signs

Affected fish typically have a distended abdomen with longitudinal swelling of the body wall at the level of the lateral line. Some fish may show dark body coloration with varying degrees of mono- or bilateral exophthalmia. Prior to death, respiratory distress is obvious, probably due to a pronounced anemia. In the final stages, there is also a marked nervous agitation with a loss of equilibrium.

INTERNAL SIGNS

Internally, the most obvious change is a gross enlargement of the kidneys into swollen, greyish, bulbous ridges. This condition invariably involves the posterior portion of the kidney but, in very severe cases, it can extend along the whole length of the kidney and include the anterior hematopoietic tissue. The swim bladder may be laterally displaced and distorted and the abdominal swelling may be compounded by excess peritoneal fluid. The spleen may be smaller than normal or massively enlarged with patches of greyish mottling beneath the capsule and throughout the stroma. The liver may show a similar greyish mottling.

DIAGNOSIS

Diagnosis of the disease requires histopathological examination of kidney and other tissues for the presence of the distinctive protozoan-like agent and associated tissue reaction. Gross lesions must be consistent with those described above and with the histopathological lesions described by Ferguson and Needham (1978). For the Great Lakes Basin, it is recommended that no diagnosis be made without consultation with a recognized authority who is familiar with the disease.

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

PKD has only been reported in rainbow trout although it has been observed in brown trout and Atlantic salmon in Europe (Ferguson, unpublished observations). The disease occurs in Europe and the British Isles (Ferguson and Needham 1978). In North America, a single outbreak has occurred near Hagerman, Idaho.

SOURCES AND Reservoirs OF Infection

A possible secondary host has been suggested by Seagrave et al. (1980) but further studies on the causative agent and its life cycle are required.

MODES OF TRANSMISSION

The mode of transmission is unknown, but Ferguson and Ball (1979) were unable to transmit the disease to rainbow trout reared for several months in water circulating through a tank of infected trout.

SUSCEPTIBILITY AND RESISTANCE FACTORS

The disease has usually been associated with soft, acidic water (Ferguson and Adair 1977). However, the outbreak in Idaho occurred in alkaline (pH 8.0) water similar to a minor outbreak of the disease on a chalk stream in southern England (Scott 1979). Fish that have recovered from an outbreak of PKD appear to be immune to further clinical disease in subsequent years.

INCUBATION PERIOD

The incubation period is probably temperature dependent (Ferguson and Ball 1979; Ferguson 1981). Clinical disease may not develop if water temperatures are low (Ferguson 1981). At a northern Irish fish farm where summer water temperatures exceeded 15°C the incubation period was approximately 2 months (Ferguson and Ball 1979).

SEASONAL INCIDENCE

In Northern Ireland and Scotland, the disease occurs during summer months (mid-July to early September) when water temperatures exceed 15°C (Ferguson and Ball 1979). The outbreak in Idaho occurred in November when water temperatures were approximately 15°C. Similarly, an outbreak was diagnosed in West Germany in mid-winter: again, the water temperature was 15-18°C (W. Korting, Tierärztliche Hochschule, personal communication).

METHODS OF CONTROL

PREVENTION

Until the cause of PKD disease is better understood, restricting the transfer of fish (and water) from areas where the disease is known to occur appears to be the best preventive measure.

THERAPY

Chemotherapy has proved unsuccessful although work continues in Europe (Ferguson and Ball 1979). Changes in management have been successful in reducing losses from the disease but not in eliminating infections (Ferguson and Ball 1979). Avoiding prolonged exposure (greater than 2 months) to water temperatures in excess of 15°C during the fingerling stage appears to be the key to controlling clinical disease (Ferguson 1981).

KEY STEPS TO REMOVE THE DISEASE And/Or AGENT FROM FISH POPULATIONS

As indicated above, PKD can be controlled in certain circumstances by controlled management practices. Methods to eliminate the disease after it has become established are unknown and await a better understanding of the causative agent's life history. The success of the eradication procedures (stock destruction and chlorination) employed in Idaho are not yet known. The Great Lakes Fishery Commission recommends that emergency disease eradication procedures be applied if PKD is diagnosed.

REFERENCES

- 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.
- Ferguson, H. W., and B. McC. Ada?. 1977. Protozoa associated with proliferative kidney disease in rainbow trout (*Salmo gairdneri*). Vet. Rec. 100: 158-159.
- 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.
- Ferguson, H.W., and E.A. Needham. 1978. Proliferative kidney disease in rainbow trout, *Salmo gairdneri* Richardson. J. Fish Dis. 1: 91-108.
- Seagrave, C.I., D. Bucke, and D.J. Alderman. 1980. Ultrastructure of a Haplosporean-like organism: the possible causative agent of proliferative kidney disease in rainbow trout. J. Fish Biol. 16: 453-459.
- Scott, P.W. 1979. The occurrence of proliferative kidney disease on a ch& stream. Vet. Rec. 105: 330-331.