

Profilerative Kidney Disease in Salmonids

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Background

Proliferative kidney disease (PKD) is one of the most economically important diseases of salmonid production in Europe and North America, including the United Kingdom. Infection has been identified in a wide variety of salmonid species in the UK, including rainbow trout, brown trout, brook trout, arctic char, atlantic salmon and grayling. Although the first ever description of the condition was by staff of the Veterinary Sciences Division in 1978, this disease had not been seen in recent years in Northern Ireland. However, in 2002 PKD was detected in wild fish in two rivers and on two freshwater farms on different river systems.

Description of Disease

PKD is an infectious condition caused by a malacosporean parasite. For many years the exact identity and nature of the disease was unknown, so that the name PKX (X for unknown) was given to the parasite. There have been a number of significant recent advances in the understanding of the disease, and the parasite has now been identified and named *Tetracapsulae bryosalmonae*.

Although all ages of fish are susceptible, disease usually occurs only in juvenile fish, with up to 100% of fish become infected.

Following entry through the gills and skin, the parasite may be found in several organs, particularly the kidney and spleen. The immune system of the fish mounts a very strong inflammatory response to the parasite resulting in visible swelling of the kidney. Associated with this, affected fish lose the ability to regulate their levels of salt and water, and become markedly anaemic. Mortality levels vary widely from 10% to almost 100%, under the influence of secondary factors such as the standard of husbandry, presence of other diseases and oxygen levels.

The disease shows a marked seasonality related to water temperature. Infection occurs in early summer associated with rising temperatures. This is followed by multiplication of the parasite and an associated inflammatory response, resulting in clinical disease two to three months later in late summer and early autumn. PKD is most severe at temperatures above 15°C and is rarely seen below 12°C. Outbreaks subside in the autumn with falling temperatures and surviving fish have a strong immunity that protects against reinfection the following year.

Transmission and lifecycle

The parasite does not appear to develop to full maturity in infected fish, and as a result transmission from fish to fish is considered not to occur. Instead, recent research has shown that the parasite grows and develops to maturity in tiny aquatic creatures called bryozoans or moss animals. Individual bryozoans are less than 1mm in size, but exist as larger colonies with an inconspicuous, branching, plant-like appearance. These colonies grow on submerged surfaces such as the underside of stones, roots, pipes and channels. Outbreaks of disease therefore require both the presence of infected bryozoan hosts and susceptible fish at a temperature that allows multiplication of the parasite in the bryozoans and/or fish. It may be the case that *T. bryosalmonae* is primarily a parasite of bryozoans, with fish being a “dead-end” accidental host. This could explain the strong immune response in fish which produces the clinical signs.

Clinical Signs

Following infection, clinical signs develop over a period of weeks. Typical signs include darkening of the skin, “pop-eye”, swollen belly and lethargy. Due to the development of anaemia, the gills may be visibly pale.

Post-Mortem Findings

The characteristic finding in fish with PKD is enlargement of the kidney, which may reach ten times its normal size (Figure 1). The kidney may have a grey/pink colour and a spongy feel. The spleen may also be enlarged, and there may be fluid, possibly bloody, present in the abdomen. On microscopic examination of the kidney, the

parasite may be observed associated with characteristic inflammatory changes, particularly in the kidney. Use of specific stains may aid detection of the parasite (Figure 2).

Diagnosis

A presumptive diagnosis may be based on the post-mortem findings. However, microscopic examination of tissues is required to confirm the diagnosis.

Treatment, Prevention and Control

Formerly malachite green has been used for the treatment of PKD, but following the ban on its use there is now no recognised treatment available. Effective control of bryozoans is almost impossible. Given their role in the disease it is very difficult to prevent fish becoming infected on affected farms, although the severity of the disease may vary markedly from year to year. Control on such farms is based around careful timing of when infection occurs. Delaying the introduction of susceptible fish until July still results in infection, but there is not enough time for disease to develop before temperatures drop, and these fish have a solid immunity the following year. In addition, fish should be managed in a way that minimizes stress during the critical summer months. Ideally, grading and movement should be avoided and feeding reduced. Fish should not be overstocked, good oxygen levels maintained and efforts made to avoid other diseases developing. While careful management may keep losses below 10% these measures also contribute to the overall economic loss.



Figure 1. Enlarged, discoloured kidney of a fish with PKD.

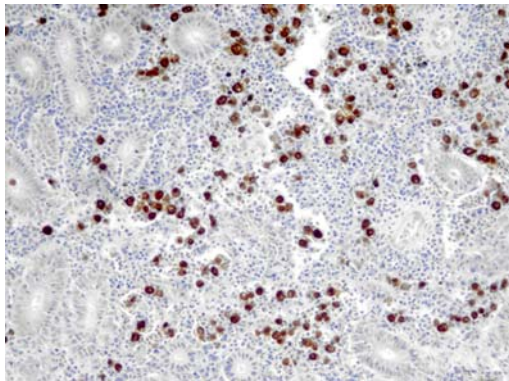


Figure 2. Microscopic view of the kidney of a fish with PKD stained to show presence of *T. bryosalmonae* (stained reddish-brown).

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