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Bacterial kidney disease

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Susceptible species
All species of the family Salmonidae are considered susceptible to bacterial kidney disease (BKD), in particular chinook salmon (Oncorhynchus tshawytscha), coho salmon (O. kisutch), rainbow trout (O. mykiss), Atlantic salmon (Salmo salar), brown trout (S. trutta) and brook trout (Salvelinus fontinalis). The Danube salmon (Hucho hucho), grayling (Thymallus thymallus) and whitefish (Coregonus lavaretus) can also become infected (Pfeil-Putzien et al., 1985; Rimaila-Päränen, 2002). Differences in susceptibility are reported between species, for example pink (O. gorbuscha), sockeye (O. nerka) and Chinook salmon (O. tshawytscha) are regarded as more susceptible than Atlantic salmon or rainbow trout. Clinical BKD has also been reported in the non-salmonids including the, ayu (Plecoglossus altivelis) and Pacific hake (Merluccius productus; Kent et al., 1988; Nagai and Iida, 2002). Experiments show the sablefish (Anoplopoma fimbri) is susceptible with morbidity or death from uncomplicated infections following intraperitoneal injection of the pathogen (Bell et al., 1990).

Disease name
The first description of what is now understood to be BKD was presented in the 1930s and followed from infection in adult Atlantic salmon from the River Dee, Scotland. BKD was recorded on many occasions in wild Atlantic salmon from Scottish rivers until the early 1960s (Mackie et al., 1933, Smith, 1964). However, since the mid-1960s BKD has not been reported from wild salmon in Scotland (Bruno, 2004).

Other names in the literature include Dee Disease, corynebacterial disease, white boil disease and kidney disease, but today bacterial kidney disease or BKD is in common use.

Aetiological agent
The bacterium Renibacterium salmoninarum is a small (~ 1.0 μm), non-motile diplobacillus that is slow growing and a fastidious pathogen (Benediktsdóttir et al., 1991; Bruno and Munro, 1986). The bacterium stains Gram positive, is non-acid fast with optimal growth in L-cysteine supplemented medium at 15°C. Growth is slow and may take 2 to 12 weeks for colonies to appear. Both horizontal and vertical transmission (i.e. from parent to progeny via the egg) of R. salmoninarum is recorded (Bruno, 1986; Evelyn et al., 1986).

Geographical distribution
BKD is widespread where salmonids are farmed and reported within Europe, North and South America as well as from Asia. At present, Ireland, Australia and New Zealand are declared free from R. salmoninarum.

Associated environmental conditions
The detection of R. salmoninarum depends upon seasonality and fish age and these factors should be taken into consideration before determining the most appropriate diagnostic method.
Significance
BKD has global importance primarily in cultured salmonids from fresh and saltwater environments with chronic losses ranging from 5–40%. Fish can carry the bacterium from freshwater to the marine phase with latent infection emerging months after seawater transfer. Similarly, stressful conditions may increase mortality with decreased growth and consequent impact on production costs. The cell surface is hydrophobic and associated with a soluble glycoprotein (Bruno, 1988) and this has a role in suppressing hosts defence mechanisms (Grayson et al., 2002).

Gross clinical signs
Clinical observations and external lesions are variable but include loss of balance, darkening and mottled appearance of the skin, distended abdomen, exophthalmia and focal or diffuse haemorrhaging around the base of the pectoral fins and lateral line. Internally the kidney is swollen, greyish in appearance with white nodules throughout the tissue (Figure 1). Generally, splenomegaly, pale liver and presence of a clear or cloudy ascitic fluid, and pale pyloric caeca with a ‘fatty-like’ appearance are noted (Figure 2). An opaque, pseudomembrane may form around the heart, intestinal tract, liver, spleen and swimbladder. A yellow viscous fluid occurs in the intestine and may contain blood.

Light microscopy
R. salmoninarum is an obligate intracellular pathogen with an integral affinity to the haematopoietic tissues including the kidney. Infection comprises extracellular bacteria with limited host response or intra- and extracellular bacteria accompanied by focal necrosis, glomerular oedema and granuloma formation. Multiple granulomas develop with a central caseous zone and circumscribed by epitheloid and other infiltrating lymphoid cells (Figures 3 and 4). The pancreatic fat becomes heavily infiltrated by bacteria that colonize the muscularis externa of the pyloric caeca with an accompanying infiltration of leucocytes. Deposits of fibrin and collagen accumulate around the swimbladder and intestine with hypertrophy and phagocytic cells containing bacteria. Macrophages containing bacteria are correspondingly associated with a marked epicarditis of the heart and spleen. Small foci containing phagocytised bacteria are located within organs, which coalesce and become the centre of an inflammatory reaction (Smith, 1964; Fryer and Saunders, 1981; Bruno, 1986).

Control measures and legislation
The control of BKD can be cost-effective by enhanced biosecurity measures, limiting the spread of infection through the detection of BKD-affected sites, imposition of movement restrictions and a requirement to eradicate R. salmoninarum before movement constraints are completely removed (Hall et al., 2014). One ‘live’ vaccine has been licensed for prevention of BKD in some countries; however, only variable efficacy has been demonstrated.

Diagnostic methods
For many countries including the UK, policies have changed, and advancement in reliable diagnostic tests have driven testing regimes. For example, the primary statutory screening tool for detection in the UK is a polymerase chain reaction (PCR). For confirmation of the disease as opposed to the presence of the pathogen, evidence is required for morbidity and/or mortality in the affected population. In addition, the
presence of clinical signs consistent with BKD and/or internal gross pathology consistent with BKD and evidence of the presence of *R. salmoninarum* in one or more laboratory tests, namely PCR or enzyme-linked immunosorbant assay (ELISA). In cases where there are signs consistent with BKD, but laboratory tests prove negative the disease is not confirmed. However, histological evidence of a Gram-positive bacterial infection or pathology consistent with BKD in the absence of ELISA and PCR is insufficient evidence of confirmation.

**Key references**


Figure 1. Rainbow trout with bacterial kidney disease. Note grey swollen kidney, pale pyloric caeca and liver, splenomegaly and internal haemorrhage.

Figure 2. Atlantic salmon with bacterial kidney disease. Note ascites and splenomegaly with opaque pseudomembrane.
Figure 3. Histological section of liver from Atlantic salmon with bacterial kidney disease. Note focal granuloma. H&E, bar scale = 200μm.

Figure 4. Histological section of kidney from Atlantic salmon with bacterial kidney disease. Note presence of extracellular *Renibacterium salmoninarum*. Gram stain, bar scale = 30μm.
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