ICES Identification Leaflets for Diseases and Parasites of Fish and Shellfish

Leaflet No. 37

Furunculosis

Original by A. L. S. Munro. Revised and updated by David W. Bruno

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Susceptible species

Furunculosis has been reported from most regions rearing salmonids including all species of charr, grayling, salmon and trout, as well as from wild salmonids (Bernoth *et al.*, 1997). Furthermore, wild non-salmonid species are susceptible and overall there are few cultured or feral fish that are immune (Cipriano and Bullock, 2001).

Disease name

The first documented evidence of furunculosis came from work by Emmerick and Weibel (1894) who observed lesions on moribund brown trout. Subsequently they isolated a bacterium, experimentally established infection in other fish and named the bacteria, *Bacterium salmonicida*. A few years later, this was changed to *Bacterium truttae* (Marsh, 1902), however this taxonomic anomaly was corrected and the original name reinstated. Finally, with the erection of a new genus its taxonomic position was finally established as *Aeromonas salmonicida* (Griffin *et al.*, 1953).

Aetiological agent

Furunculosis is caused by *Aeromonas salmonicida* subsp. *salmonicida*, a Gram negative, non-motile, facultative anaerobic rod measuring 1-2 x 0.5 μm (Cipriano and Austin, 2011). Most strains are oxidase positive and produce a water-soluble brown pigment on media containing tryptone with optimum growth between 22 and 25°C. The term 'atypical *Aeromonas salmonicida*' has been used for bacterial strains that are distinguished through morphological and biochemical differences comprising colony size, growth-rate, haemolysis and sucrose fermentation. Recognized subspecies of A. *salmonicida* include *achromogenes, masoucida, smithia* and *pectinolytica*. The hosts for atypical strains include a wide variety of non-salmonid fish, as well as salmonids.

Geographical distribution

Aeromonas salmonicida and subspecies are found worldwide.

Associated environmental conditions

Outbreaks of furunculosis are triggered by interrelated stressors such as higher water temperatures in the spring or during periods of rapid temperature change, handling including smolting and spawning, crowding and poor water quality.

Significance

Furunculosis is an important disease of cultured salmonids and can have significant negative economic impacts on aquaculture operations. Pathogenicity depends on the external surface layer, mainly composed of the A-protein (so-called A-layer) which provides protection against the defence mechanisms of the host. In addition, lipopolysaccharides (LPS), another major cell envelope antigen, occurs on the bacterial surface and helps resist the host's normal bactericidal mechanisms. During bacterial growth, extracellular products (EPC) are released and induce lesions leading to mortalities. In some fish, a carrier state may be established after infection. The bacterium is carried by fish from freshwater to the marine environment, and overall

infectivity, certainly for Atlantic salmon in seawater, is similar to that reported for infections in freshwater.

Gross clinical signs

Chronic and acute furunculosis occur and generally outcomes dependant on water temperature, age of the fish and pathogenicity of the strain of *A. salmonicida*. Fish may die acutely with few or no prior signs of disease and limited pathological changes, or in the chronic stage, the fish show lethargy, inappetence and darkening of the skin, however these are common in most bacterial septicaemias. Ventral haemorrhage in particular near the base of the pectoral, pelvic and anal fins in addition to exophthalmia can be recorded. At necropsy, ascites, splenomegaly and subcapsular haemorrhage in the liver maybe observed.

Liquefactive, haemorrhagic 'boil' lesions in superficial muscle are often reported in chronic cases and raised, fluctuating lesions that may rupture may be seen at the skin surface (Figure 1). Bacteria are released from skin ulcers and this contributes to the spread of infection.

Light microscopy

Histopathological lesions are characterized by aggregates or dense micro-colonies in organs such as heart, kidney, liver, spleen and muscle (Figure 2). Horizontal spread with subsequent bacterial colonization may be seen in the capillaries of the gills (Bruno, 1986). Mural thrombi with bacterial colonization are often seen on the intimal aspect of vessels and ellipsoids. There is generally little tissue reaction around such aggregates in the early stages of the disease, but tissue necrosis and liquefaction may be widespread in late and chronic phases.

Control measures and legislation

Prophylactic measures against furunculosis have been available for a long time and successful vaccines are available, hence furunculosis is currently rare in salmonid farming. Avoidance through effective management and following best practice is the most effective for managing infection, but where clinical outbreaks occur, medicated feed can be prescribed.

Diagnostic methods

Diagnosis is based upon gross and histopathological lesions, isolation of the causative agent, immunohistochemistry or polymerase chain reaction (PCR). The appearance of *A. salmonicida* microcolonies in haematoxylin and eosin stained tissue sections is considered pathognomonic. Furuncles are characteristic, but they are not always present in diseased fish and therefore not necessarily a diagnostic characteristic. Primary isolation of the pathogen is achieved from the kidney and other organs using media such as tryptic soy agar (TSA) or brain heart infusion agar (BHIA).

Key references

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Figure 1. Large subcutaneous furuncle attributed to furunculosis in Atlantic salmon.

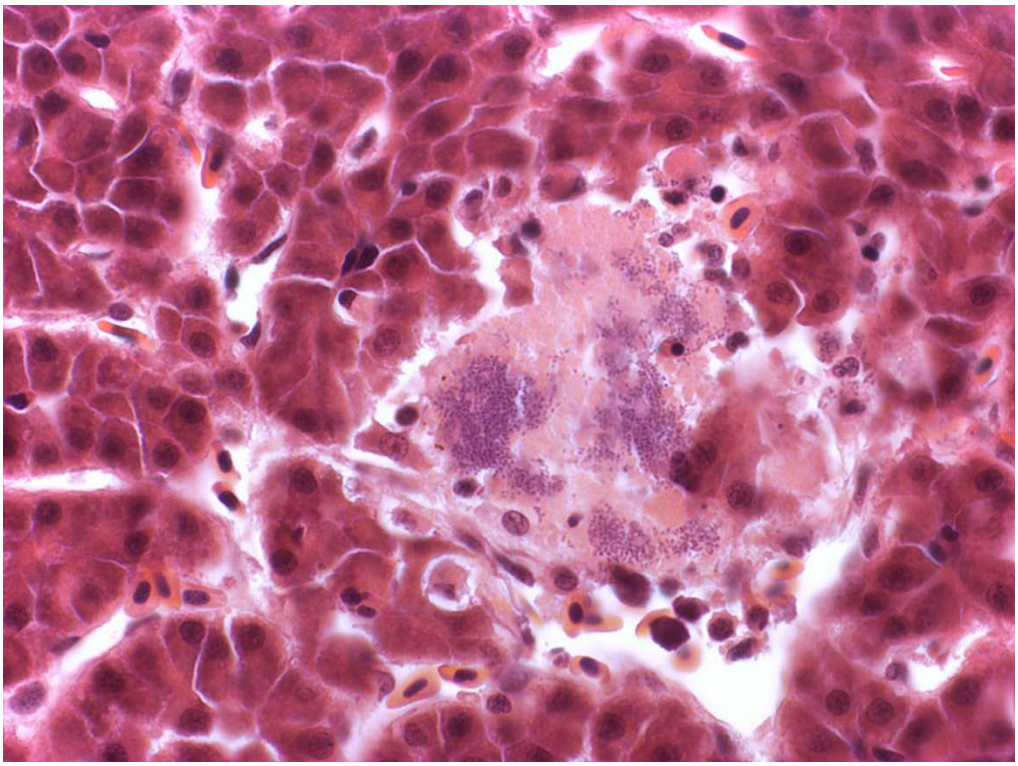

Figure 2. Diffuse necrotic lesion in liver of rainbow trout showing typical staining of *Aeromonas salmonicida*, HE stain.

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