# Bacterial kidney disease (Renibacterium salmoninarum)



#### AETIOLOGY

#### **CLASSIFICATION OF THE CAUSATIVE AGENT**

Eubacteria, family Micrococcaceae, genus Renibacterium, species R. salmoninarum. Renibacterium salmoninarum is a Gram-positive nonsporulating, nonmotile coccobacillus that seems to act as an obligate parasite. It develops chronically in salmonid fish and results in late clinical disease.

#### RESISTANCE TO PHYSICAL AND CHEMICAL ACTION

Temperature: Optimum temperature for culture: 15°C.

Preserved by refrigeration, freezing, freeze-drying.

Chemicals/ Chlorine, quaternary ammonia, formalin, Disinfectants: may be useful for disinfecting materials.

However, transmission *in ovo* and the wide distribution of the pathogen in natural habitats limit the effectiveness of disinfection proceedures.

disinfection procedures.

Survival: Survival of this organism in the environment is poorly understood. It has been reported that the bacterium could remain viable in seawater for up to 1 week, and in the absence of other microflora, even longer. This duration of survival could be significant in a hatchery or netpen. May be hosted by nonsusceptible nonsalmonid species.

# EPIDEMIOLOGY

- Morbidity rate is high, due to vertical transmission.
- Mortality is delayed but may become serious in highly infected populations.

# Hosts

· All salmonid species.

# **TRANSMISSION**

- Horizontal transmission may be direct, through contact with diseased fish and contaminated water, or indirect, through handling materials or feeding with fisheries residues.
- Vertical transmission is the most frequent route of contamination. The bacteria are present in the ovarian fluids and are likely to be the source of the R. salmoninarum that have been detected in eggs from some infected female salmonids.

# **SOURCES OF THE AGENT**

- Internal organs or skin tissues in cases of external lesions.
- · Eggs and coelomic fluid.
- Inanimate vectors.

 Many fish species, and even invertebrates, may be hosts of the pathogen without clinical manifestation.

#### **OCCURRENCE**

Occurs in feral and farmed populations of salmonids in almost all areas where they are naturally distributed or have been acclimatised. No conclusive evidence has been reported for the presence of *R. salmoninarum* in the salmonid population of Australia, New Zealand, Russia, or some Mediterranean, countries.

For detailed information on occurrence, see recent issues of *World Animal Health* and the OIE Web site.

# DIAGNOSIS

Incubation period is long, and the clinical signs, often prompted by environmental causes, usually occur in fish over 1 year old.

#### **CLINICAL DIAGNOSIS**

- Dark coloration, exophthalmia and abdominal distension result from the destruction of interstitial kidney tissues.
- Externally, haemorrhages may be observed at the base of the fins or at the vent, and the rupture of small cutaneous vesicles results in small ulcerations.
- Greyish nodules or diffuse masses are generally observed in the kidney, spleen and liver. The kidney appears enlarged and the abdominal cavity is generally filled with fluid.

#### **LESIONS**

 Typical chronic infection characterised by granuloma scattered in the internal organs. A phagocytic reaction associated with more or less complete encapsulation may be observed. Caseous necrosis may be observed in advanced cases.

# **DIFFERENTIAL DIAGNOSIS**

 External manifestations are nonpathognomonic, but the course of the disease and the nature of the kidney lesions may provide presumptive indications. The disease must be differentiated from other kidney diseases of chronic progression including pseudo-kidney disease (Carnobacterium piscicola), nephrocalcinosis, and proliferative kidney disease. Confirmation must be by the observation and identification of the bacteria.

# **LABORATORY DIAGNOSIS**

#### **Procedures**

# Identification of the agent

- Isolation and culture under appropriate conditions, completed by bacteriological identification or specific agglutination, require several weeks.
- Antigen detection in infected tissues is more rapid.
- Immunofluorescence (direct/indirect fluorescent antibody test [FAT and IFAT]).
- Enzyme-linked immunosorbent assay (ELISA).
- Polymerase chain reaction (PCR).

# Serological tests

 Not recommended. No positive correlation has been demonstrated between the antibody response and the course of the infection in fish populations.

# **Samples**

# Identification of the agent

- Internal organs from diseased fish: kidney and spleen are the most convenient.
- Tissue prints or smears for the detection of the antigen.
- In maturing females, coelomic fluid is a reliable material and does not need to kill the fish.

# PREVENTION AND CONTROL

# **TREATMENT**

 No therapy has really proved conclusive in cases of overt disease.

#### SANITARY PROPHYLAXIS

- Identification and culling of carrier broodfish.
- Destruction or slaughtering of infected fish.
- · Disinfection of premises, materials and raceways.
- Introduction of controlled pathogen-free stocks.
- In infected areas, screening of asymptomatic carriers before spawning, and selection of breeders with the lowest levels of infection associated with erythromycin treatment have noticeably reduced the prevalence of the disease.

# **MEDICAL PROPHYLAXIS**

There is presently no possibility of active immunisation.

# REFERENCES

Chapter 2.2.6. in the OIE *Diagnostic Manual for Aquatic Animal Diseases*, OIE, Paris, France.

Chapter 2.2.6. in the OIE *International Aquatic Animal Health Code*, OIE, Paris, France.

# OIE Reference Experts and Laboratories in 2000

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