## PATHOGEN INFORMATION

# 1. CAUSATIVE AGENT

### 1.1. Pathogen type

Virus.

### 1.2. Disease name and synonyms

Salmon pancreas disease (SPD), pancreas disease (PD), sleeping disease (SD).

### 1.3. Pathogen common names and synonyms

Salmonid alphavirus (SAV), salmon pancreas disease virus (SPDV), pancreas disease virus (PDV), sleeping disease virus (SDV),.

### 1.4. Taxonomic affiliation

1.4.1. Pathogen scientific name (Genus, species, subspecies or type)

*Salmonid alphavirus* (SAV) (Weston *et al.,* 2002) is used as species name in scientific publications, although the only species name that is approved by the International Committee on Taxonomy of Viruses is *Salmon Pancreas Disease Virus* (SPDV) (ICTV Feb. 2013).

Different gentotypes of SAV are named SAV subtypes 1, 2, 3, 4, 5 or 6 (Graham *et al.,* 2011).

1.4.2. Phylum, class, family, etc.

Family: *Togaviridae* Genus: *Alphavirus*

### 1.5. Description of the pathogen

SAV is an enveloped, single-stranded RNA virus, approximately 55–65 nm in diameter.

### 1.6. Authority (first scientific description, reference)

Nelson R.T., McLoughlin M.F., Rowley H.M., Platten M.A. & McCormick J.I. (1995). Isolation of a toga-like virus from farmed Atlantic salmon *Salmo salar* with pancreas disease. *Dis. Aquat. Org.*, **22**:25–32.

### 1.7. Pathogen environment (fresh, brackish, marine waters)

Fresh, brackish and marine water

# 2. MODES OF TRANSMISSION

### 2.1. Routes of transmission (horizontal, vertical, direct, indirect)

SAV is horizontally transmitted, via water & water currents, transport of infected fish, contaminated equipment, well boats etc. The virus can survive for extended periods in seawater. (McLoughlin & Graham, 2007, Viljugrein *et al.,* 2009),

Vertical transmission via eggs is unlikely (Kongtorp *et al.,* 2010).

### 2.2. Reservoir

Infected farmed salmonid fish is the main reservoir.

The natural reservoir of SAV is not known but might be wild fish as SAV has been detected in some wild flatfish species in Scotland (Snow *et al.*, 2010, Bruno *et al.,* 2012).

### 2.3. Associated factors (temperature salinity, etc.)

Water temperature may affect infection dynamics with regard to the duration and amount of mortality.

Subtypes 1, 2, 3, 4, 5 and 6 may have different virulence in different hosts (Graham *et al.,* 2011).

Different strains of Atlantic salmon may have different susceptibility to SAV (McLoughlin *et al.,* 2006).

Management and environmental factors that induce stress reactions in the fish probably affect mortality significantly.

### 2.4. Additional comments

None.

# 3. HOST RANGE

### 3.1. Host type

Salmonid fish species

### 3.2. Host scientific names

Atlantic salmon (*Salmo salar* L.), rainbow trout (*Oncorhynchus mykiss* [Walbaum]) and brown trout (*Salmo trutta* L.) (McLoughlin & Graham, 2007, Boucher *et al.,* 1995).

### 3.3. Other known or suspected hosts

Other salmonid fish species may be susceptible but this has not been investigated. A wild marine and/or freshwater host for SAV is suspected as reservoir.

### 3.4. Affected life stage

Probably all life stages, depending on the local reservoir of SAV.

### 3.5. Additional comments

None

# 4. GEOGRAPHICAL DISTRIBUTION

### 4.1. Region

Europe

### 4.2. Countries

Known presence in United Kingdom (England, Scotland & Northern Ireland), Ireland, Norway, France, Germany, Italy, Spain, and Croatia.

### 4.3. Additional comments

None

## DISEASE INFORMATION

# 5. Clinical Signs and Case Description

## 5.1. Host tissues and infected organs

All organs of susceptible fish species should be regarded as potentially infected because SAV infection is a systemic disease with a viraemic phase. SAV has also been detected in mucus and faeces.

## 5.2. Gross observations and macroscopic lesions

Often a sudden drop in appetite has been observed a week or two before a disease outbreak. During the outbreak, clinically diseased fish may be observed close to the surface, swimming against the water current or close to the corners of the cage. Fish may also be found resting at the bottom of the tank or cage. One to several months after the onset of mortality, a portion of the survivors usually fail to grow and become thin and slender ("runts").

Macroscopic changes will vary, but yellow mucoid gut contents are commonly found, similar to what is found in other fish that lack appetite. Some fish may have petechiae in the pyloric fat, pale hearths and/or haemopericardium due to heart rupture. Signs of circulatory disturbance as scale pocket oedema, exophthalmos and/or ascites may be found.

## 5.3. Microscopic lesions and tissue abnormality

Exocrine pancreas, heart and skeletal muscle are the main organs with histopathological changes. Necrosis of exocrine pancreas develops shortly before cardiomyocytic necrosis. The most prominent changes appear later as the disease progresses. Then, severe or total loss of exocrine pancreas, myocarditis and skeletal muscle degeneration and myositis are the main findings.

## 5.4. OIE status

Under consideration for listing.

# 6. Social and Economic Significance

Infection and/or outbreaks usually result in significant economic losses due to mortalities, extra management costs, treatment, prevention and reduced slaughter quality (Aunsmo *et al.,* 2010).

# 7. Zoonotic Importance

None

# 8. Diagnostic Methods

## 8.1. Surveillance methods

**8.1.1.** Surveillance through general disease diagnostics is efficient in order to identify the disease, and is especially useful in regions yet unaffected by infection with SAV (Hjortaas *et al.,* 2013). Such examinations should include autopsy of clinical diseased fish, histopathology and methods for the detection of SAV and/or antibodies against SAV.

**8.1.2.** Surveillance through examination of tissue samples for virus by real-time RT-PCR (Hodneland & Endresen 2006, Graham *et al.,* 2006) is a sensitive and specific method, especially if samples from clinical diseased fish or thin, slender fish ("runts") are included (Jansen *et al.,* 2010).

**8.1.3.** Surveillance through examination of blood samples for specific antibodies against SAV can also be applied (Graham *et al.,* 2003).

## 8.2. Presumptive test methods

Real-time RT-PCR detects virus RNA (Hodneland & Endresen 2006, Graham *et al.,* 2006). The samples should include heart tissue (Andersen *et al.,* 2007).

A serum neutralisation assay as described by Graham *et al.,* 2003 identifies either antibodies against SAV or SAV (viraemia) in serum samples.

## 8.3. Confirmatory test methods

Histopatological examination with the detection of characteristic pathological changes (Boucher *et al.,* 1995, McLoughlin *et al.,* 2002, Taksdal et al 2007) in combination with the detection of SAV in the same individual fishes is confirmatory for infection with SAV and is a combination with high specificity for identification of the disease.

Isolation of SAV by cell culture (McLoughlin & Graham, 2007) and/or sequencing of selected parts of SAV genome are methods with high specificity for the identification and subtyping of SAV.

# 9. Control Methods

All measures that may prevent horizontal transmission from infected to non-infected fish populations, like avoidance of moving of live fish, avoidance of sharing fish farming equipment, personnel etc. between fish farms, are important.

Disinfection of offal and water from slaughtered fish may be crucial to prevent further spread of SAV.

Disinfection of well boats and aquaculture equipment will contribute in prevention of virus transmission.

Vaccines against SAV have a positive effect in decreasing cumulative mortality and the number of fish discarded at slaughter (Bang Jensen *et al.,* 2012).

In infected populations most measures that reduce stress will probably reduce the impact of infection with SAV.

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| **OIE Reference Experts and Laboratories in 2013: none** | |
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