Bacterial Kidney Disease (BKD)

Bacterial disease affecting salmonid species around the world.

- **External Signs:** lethargy, skin darkening, protruding eyes and blood-filled blisters on the flanks
- **Affects:** principally farmed and wild salmonids
- **Mortality:** chronic losses over an extended period
- **Treatment:** no treatment

Overview

Bacterial kidney disease (BKD) is a chronic disease caused by the bacterium *Renibacterium salmoninarum*. It occurs worldwide where salmonid fish are found. It is known to cause up to 80% mortality in Pacific salmon and 40% in Atlantic salmon.

Disease causing agent: *Renibacterium salmoninarum* of the Corynebacteriaceae family

BKD has no implications for human health.

Geographical Distribution

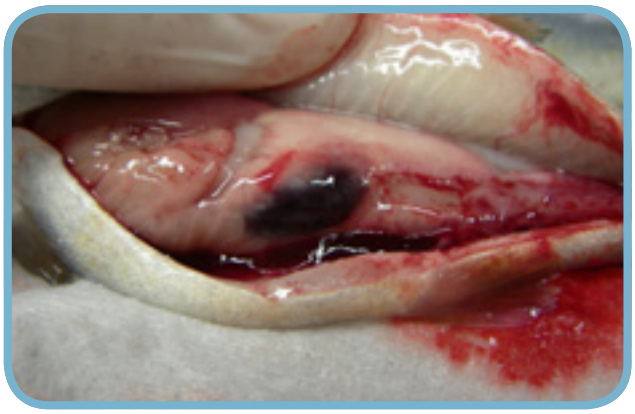
BKD was first described in 1933, following a mortality of wild salmon in the River Dee, Scotland. Since then, BKD has been reported worldwide where salmonid aquaculture takes place, including North America, South America, Europe, Japan and Iceland. Although the disease is still exotic to Australia.

In the UK, BKD has been found in both fresh and seawater locations in Scotland, England and Wales. Northern Ireland is free from BKD.

BKD is not restricted to cultivated fish. The condition has been found in naturally spawning salmonid populations.

Characteristics & clinical signs

BKD may be transmitted horizontally, through contact with infected fish or water, and vertically, from infected broodstock eggs. It is thought infected fish shed bacteria in faeces. Experimental evidence shows viable bacteria can persist for several weeks in fish faeces and in sediment.



BKD is a systemic infection in affected fish. Due to the slow-growing nature of the bacteria it may take several months before disease signs appear.

Infected fish display a wide range of clinical signs, however some are asymptomatic with no signs of disease. Clinical signs include; lethargy, skin darkening, protruding eyes (exophthalmia), anaemia, distended abdomens, blood-filled blisters on the flanks and bruising (haemorrhaging) around the vent.

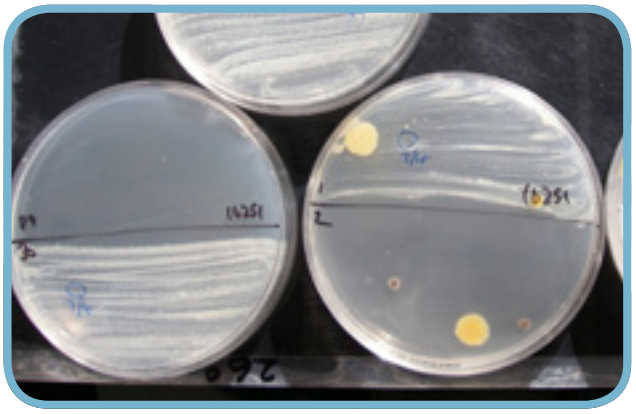
Internal signs include presence of fluid in the abdominal cavity, swollen kidneys sometimes with white/grey lesions and diffuse white membranes over the internal organs. Microscopically bacteria are often seen in granulomatus lesions in the kidney and also in the white blood cells (macrophages), within which they appear able to survive and multiply.

Susceptible species

BKD causes significant mortalities in wild Pacific salmon Other salmonids have varying degrees of susceptibility. In Great Britain the disease has been found in Atlantic salmon, brown trout, rainbow trout and grayling.

Diagnosis

In England and Wales salmonids are screened for BKD by ELISA (Enzyme Linked Immunosorbant Assay), and PCR (polymerase chain reaction) tests. Growth in culture is carried out by inoculating kidney swabs onto SKDM (selective kidney disease medium), with bacterial colonies confirmed by serological tests and PCR.



Treatment and control

Despite research, there is no effective treatment for BKD. Good biosecurity and avoidance of infection are the effective means of controlling the disease.

The chronic nature of BKD and the presence of asymptomatic fish in the early stages of infection can be problematic when adopting control measures. Effective health monitoring of farmed stocks and attention to biosecurity systems may help avoid BKD.

If BKD was found on a UK farm, movement restrictions would be applied. All contact sites are investigated for evidence of the disease source and spread.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of BKD in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of BKD.

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Epizootic Haematopoietic Necrosis (EHN)

Serious systemic viral disease infecting fish.

- **External Signs:** abdominal distension, darkening of the body, reddening fin bases and skin ulcers
- **Affects:** rainbow trout and perch are known to be susceptible
- **Mortality:** up to 100% mortality in perch, lower mortality in rainbow trout
- **Treatment:** no treatment and the virus is resistant to disinfection

Overview

Epizootic haematopoietic necrosis (EHN) is a systemic viral disease of rainbow trout, *Oncorhynchus mykiss*, and the European or red-fin perch, *Perca fluviatilis*. EHN related viruses infect wels catfish, ictalurid catfish, and turbot. Additionally similar viruses have been found in healthy and diseased amphibia and reptiles.

Disease causing agent: Epizootic haematopoietic necrosis virus of the genus Ranavirus of the family Iridoviridae.

EHN has no implication to human health.

Geographical Distribution

EHN is endemic in southeastern Australia. Infections in perch in the wild are thought to be widespread. Disease outbreaks occur on rainbow trout farms, often connected to waters holding perch.

Characteristics and clinical signs

The first signs of EHN infected perch are large-scale mortalities of the population. The most seriously affected are fingerling and juvenile fish. However the disease occurs in fish of all ages.

Internally an enlarged spleen and kidney often accompany pinpoint bruising (petechial haemorrhaging) in the internal organs.

The disease has more chronic presentation in rainbow trout. It mainly affects fingerlings. Clinical signs include; abdominal distension, darkening of the body surface, loss of appetite, flared gill covers (opercula) and reddening at the fin bases, loss of equilibrium and skin ulcers. Total mortality is usually low to moderate.

EHN outbreaks are associated with water temperatures between 11°C and 17°C and poor water quality. Outbreaks generally occur in summer, and result in dramatic population declines of wild perch.

Susceptible Species

In the wild EHN has been reported in European perch and rainbow trout. However under experimental conditions EHN was demonstrated to be pathogenic in a number of other species of fish including silver perch, mosquito fish, and Macquarie perch.

Diagnosis

The virus is isolated in cell culture from internal organs of affected fish and identification confirmed by PCR (Polymerase Chain Reaction).

Treatment and control

There is no treatment for EHN.

Due to viruses in the EHN group being resistant to inactivation, they are presumed to persist for months or even years on infected farms in water, pond sediment, plants and equipment. EHN is highly resistant to drying and disinfection.

If EHN was found in the UK, movement restrictions would be applied to affected farms and measures taken to eradicate the disease. All contact sites would be investigated for evidence of the virus source and spread.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of EHN in any farmed or wild fish to the Fish Health Inspectorate.

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Epizootic Ulcerative Syndrome (EUS)

Serious disease of farmed and wild fish in fresh and brackish waters.

- **External Signs:** red spots, blackish burn-like marks, deep ulcers at the base of fins and over the body
- **Affects:** a wide range of species are susceptible
- **Mortalities:** up to 80%
- **Treatment:** no treatment

Overview

Epizootic ulcerative syndrome (EUS) is a disease of wild and farmed, fresh and brackish water fish. For over three decades it has caused major fish losses in many countries. EUS has a complex aetiology characterised by the presence of the fungal infection, *Aphanomyces invadans,* and necrotising ulcerative lesions. Over 100 species of freshwater fish are reported as EUS susceptible.

Disease causing agent: The fungus *Aphanomyces invadans* is widely acknowledged to be the causative agent of EUS.

EUS has no impact on human health.

Geographical Distribution

EUS is endemic in south-east and south Asia, Australia and Japan. It has spread rapidly in recent years and recently been reported in Africa.

To date there have been no reported EUS cases in the UK or European Union.

Characteristics and Clinical Signs

The disease is transmitted horizontally through contact with infected fish or water. Epidemics can develop rapidly.

EUS is characterised by large bruises or necrotic (dead) ulcerative lesions on the base of fins and other body parts. Later these become larger inflamed areas with acute degeneration of skin (epidermal) tissues. This includes; red spots, blackish burn-like marks or deep ulcers with red centres and red rims, with some ulcers eroding to expose the spine, brain and internal organs. The fungus may invade internal organs such as the kidney and liver. Mortality of up to 80% of stock is associated with an EUS outbreak.

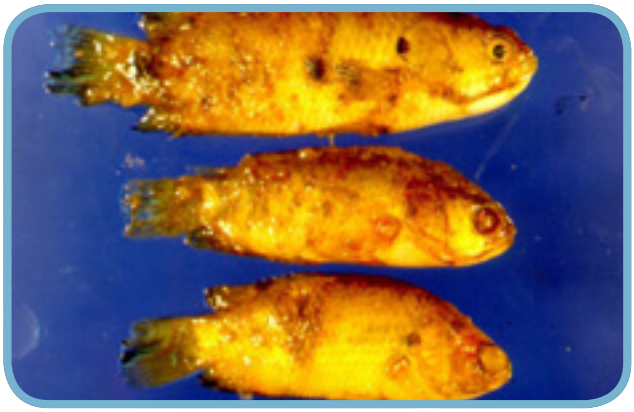
EUS is thought to be triggered by rapid changes to pH, extreme weather events, such as heavy rainfall resulting in flooding, and variations in salinity and water temperature.

Susceptible Species

EUS has been reported in over 100 species of fresh and brackish water fish including; crucian carp, rudd, barramundi, wels catfish, perch, eels, mullet and rainbow trout.

Diagnosis

EUS is grown in culture by inoculating swabs taken from affected fish onto agar medium. Identification is confirmed by PCR (Polymerase Chain Reaction) on culture isolate.



Treatment and control

There is no treatment for EUS.

If EUS was found in the UK, control measures would be implemented, including disinfection of farms and fisheries. Movement restrictions would be applied and measures taken to eradicate the disease. All contact sites would be investigated for evidence of disease in order to identify the source and spread of the infection.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of EUS in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of EUS.

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Gyrodactylus salaris (GS)

External parasite affecting Atlantic salmon.

- **External Signs:** fish have a white or grey appearance often with secondary fungal infections
- **Affects:** primarily affects Atlantic salmon in freshwater
- **Mortality:** high mortality levels - up to 95%
- **Treatment:** no treatment of wild fish, except eradication

Overview

Gyrodactylus salaris (GS) is an external parasite primarily affecting Atlantic salmon in freshwater. GS can occur in high densities resulting in heavy loss of salmon parr and smolts. Although not currently found in UK waters, an introduction of GS could rapidly spread across the country and dramatically reduce wild salmon stocks.

Disease causing agent: *Gyrodactylus salaris* is a monogenean parasite of the phylum Platyhelminthes.

GS has no implication on human health

Geographical Distribution

GS was first reported in Sweden and is believed to occur naturally in Russia and the Baltic regions of Finland, Latvia and Lithuania. The Baltic strain of salmon has a high level of tolerance to infection. However Atlantic salmon are highly susceptible to the parasite.

Experiments have demonstrated that salmon from UK waters are susceptible to infection. GS has also been reported in Norway, Denmark and Germany.

GS is not currently found in the UK or Ireland.

Characteristics and clinical signs

GS is a small worm like organism up to 2mm in length. It attaches to the fins, gills and skin of fish using specially modified hooks. Once attached the parasite feeds by releasing digestive enzymes on to the skin of the fish, digested material is consumed by the parasite.

Clinical signs of infection reflect the irritation that the parasite causes to fish. Behavioural changes such as flashing may be observed, with increased mucus production. Skin damage may include ulcers and small lesions, leaving the fish subject to secondary fungal infection.

The parasite can be seen under low magnification. Without magnification, heavily infected fish may have a white or grey appearance due to excess mucus production. Also fungal infections may occur.

Experience in Norway has shown when GS is introduced to a new host population, 95% of wild Atlantic salmon parr will be lost within a few years. It is possible a single GS specimen introduced into an unaffected water system would be capable of starting an epidemic. GS has the capability to reproduce and spread very rapidly.

GS can be transmitted on infected fish both alive or dead. Other fish species - such as eels, minnows and sticklebacks - can transport the parasite. It can also survive for several days in damp conditions. Therefore it can be accidentally transported on damp equipment - such as fishing tackle, nets and waders - used in infected waters.

Susceptible species

The main host of GS is the Atlantic salmon, although other salmonids are also susceptible including; rainbow trout, Arctic char, North American brook trout, grayling, North American lake trout and brown trout.

Clinical signs are generally not seen on species other than Atlantic salmon

Diagnosis

The fins of affected fish are examined microscopically. Individual parasites are removed from the fish and examined under high power magnification. Presumptive diagnosis is made by examination of the shape and size of the hooks of the attached organ, and comparing with morphological data held on other species. Identification is confirmed by PCR (Polymerase Chain Reaction) on isolated parasites.

Treatment and control

The UK is recognised as being GS free and therefore imposes controls on live fish movements from infected countries. Other potential avenues of introduction exist, such as through contaminated angling equipment.

Anglers returning from fishing excursions in countries with GS infection should thoroughly clean and disinfect all fishing equipment. This kills any parasites and prevents accidental introductions. Countries such as Iceland and Norway may require certification confirming angling equipment disinfection before permitting fishing.

Norway has tried to eradicate GS from infected river systems by chemical means. Chemical treatment is costly, environmentally damaging, only possible under favourable conditions, and has had varying degrees of success. GS cannot survive full strength sea-water, therefore migratory fish will not reintroduce the parasite.

The most effective control on GS is to prevent its introduction into the UK. If GS was found in the UK, control measures would be implemented, including disinfection of farms and fisheries. Movement restrictions would be applied and measures taken to eradicate the disease. All contact sites would be investigated for evidence of disease in order to identify the source and spread of the infection.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of GS in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of GS.

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Infectious Haematopoietic Necrosis (IHN)

Serious viral disease affecting salmon and trout.

- **External Signs:** lethargy with period of frenzy, darkening of skin, pale gills, distended abdomen and protruding eyes
- **Affects:** salmonid species
- **Mortality:** up to 100% mortality in juvenile rainbow trout
- **Treatment:** no known treatment

Overview

Infectious haematopoietic necrosis (IHN) is a viral disease affecting a range of salmonid fish species. IHN can occur on farms rearing fry or juvenile rainbow trout in freshwater, where acute outbreaks result in high mortality. Mortality rates can be acute or chronic and depend on factors such as species, water temperature and husbandry conditions. Mortality of up to 95% is common.

Disease causing agent: Infectious haematopoietic necrosis virus of the genus novirhabdovirus and family Rhabdoviridae.

IHN has no implications for human health.

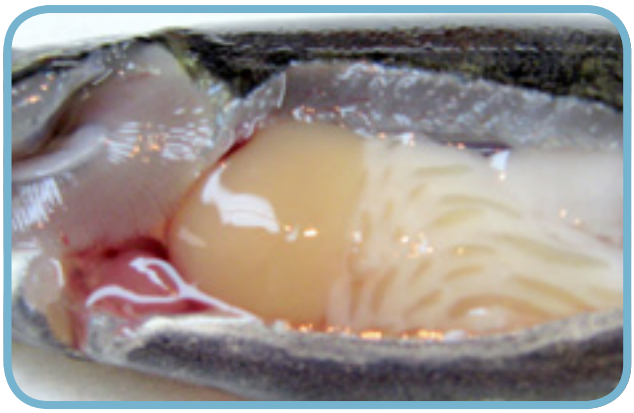
Geographical Distribution

Historically IHN was geographically limited to the western part of North America. However the disease has spread to continental Europe and Asia through movement of infected fish and eggs. Once IHN is introduced to farmed stock or wild fish, the disease may become persistent in carrier fish.

Characteristics and clinical signs

IHN is typically characterised by lethargy interspersed with periods of frenzied, abnormal activity. Clinical signs include darkening of the skin, pale gills, fluid in the abdomen (ascites), distended abdomen, protruding eyes (exophthalmia), and pinpoint bruising (petechial haemorrhaging). Fish appear anaemic and lack food in the gut. The liver, kidney and spleen are often pale.

The disease may be transmitted horizontally, through contact with infected fish or water, and vertically, from infected broodstock eggs.



Water temperature is the most important factor in IHN outbreak progression, typically causing highest mortality between temperatures of 8°C and 15°C.

Outbreaks are most severe in fingerling and juvenile fish. In aquaculture older rainbow trout are reported to be chronically affected. Wild fish act as an important reservoir of infection where the disease is endemic. Carrier fish are thought to release large amounts of virus resulting in widespread and persistent infection in affected catchments.

Susceptible Species

Species that have been shown to be susceptible to IHN include; rainbow or steelhead trout, Atlantic salmon. Pacific salmon including chinook, sockeye, chum, yamame, amago, and coho.

Diagnosis

The virus is isolated in cell culture from internal organs of affected fish and identification is confirmed by IFAT (Immunofluorescent Antibody Technique) and PCR (Polymerase Chain Reaction).

Treatment and control

There is no treatment for IHN.

If IHN was found on a UK farm, eradication and disinfection programmes would be implemented. Movement restrictions would be applied to contact sites and all farms on the same river catchment. Attempts would be made to eradicate IHN and all contact sites would be investigated to identify the virus source and spread. Any approved status would be suspended until disease eradication has been confirmed.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of IHN in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of IHN.

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Infectious Salmon Anaemia (ISA)

Serious viral disease affecting farmed salmon.

- **External Signs:** pale gills, distended abdomen, lethargy, lack of appetite, protruding eyes and gasping at water surface
- **Affects:** farmed Atlantic salmon
- **Mortality:** up to 90% mortality in cage sites
- **Treatment:** no known treatment

Overview

Infectious salmon anaemia (ISA) is a disease of farmed Atlantic salmon (*Salmo salar*) in seawater. Mortality rates can vary from 1% per day to 90%. The UK was historically free of ISA, until an outbreak in Scotland in 1998. A further outbreak occured in farmed salmon in the Shetland Isles in 2008 and 2009.

Disease causing agent: Infectious salmon anaemia virus (ISAV) is the only recorded member of the Orthomyxoviridae family found in fish.

ISA has no implication for human health.

Geographical Distribution

ISA was first reported in Norway in the 1980's and since then has been identified in Canada, Scotland, the Faroes and the USA. The virus has been isolated in Coho salmon and rainbow trout in freshwater. ISA is having a serious economic impact on salmon farming in Chile.

Characteristics and Clinical Signs

The disease may be transmitted horizontally through contact with infected fish, faeces, urine or water. The gills are thought to be the most likely infection point.

Prominent clinical signs on fish include pale gills, protruding eyes (exophthalmia) and distended abdomen. Internally a darkened liver and swollen kidney may indicate the presence of infection. Bruising (haemorrhaging) in the intestine may also be seen. In many cases, one of the above symptoms may be more prevalent than others.

Behaviourally, infected stock may display lethargy, lack of appetite, and gasping at the water surface .

Susceptible Species

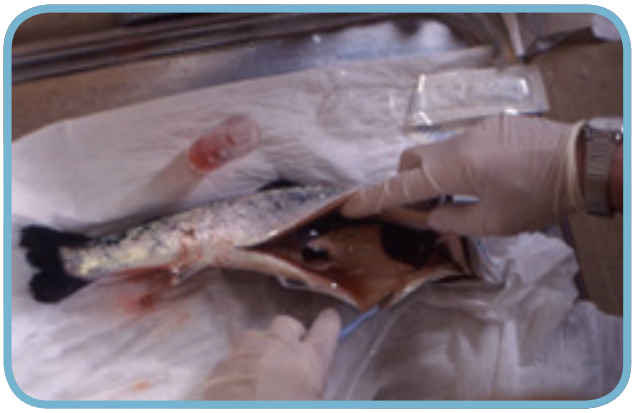
Outbreaks of ISA have mainly occurred in farmed Atlantic salmon. However the virus has been recovered from wild Atlantic salmon, brown and sea trout and also pollock and cod caught in the vicinity of cages holding farmed salmon. Recently the virus has been reported in freshwater farmed rainbow trout. Clinical disease is generally only seen in farmed fish

Diagnosis

The virus is isolated in cell culture from internal organs of affected fish and identification is confirmed by RT-PCR (Reverse Transcriptase Polymerase Chain Reaction).

Treatment and control

There are no effective treatment for ISA and no licensed vaccines in the EU. The occurrence and spread of ISA is reduced by good biosecurity measures and husbandry practices.



Like other notifiable diseases, suspicion of infection would result in movement restrictions being placed on all farms within the catchment, or zone, and contact sites. If confirmed, attempts to eradicate the disease and investigate all contact sites to establish the source and spread of the virus.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of ISA in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of ISA.

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Koi Herpesvirus Disease (KHV)

Serious viral disease affecting all varieties of common carp.

- **External Signs:** lethargy or erratic behaviour, loss of balance, sunken eyes and necrotic patches in the gills
- **Affects:** all varieties of common carp, including varieties such as mirror, leather, koi and ghost koi
- **Mortality:** up to 100% mortalities
- **Treatment:** no known treatment

Overview

Koi herpesvirus (KHV) disease is a viral disease of common carp, *Cyprinus carpio*, and all varieties of common carp including; koi, ghost koi, mirror, and leather carp. The virus is highly contagious and may cause up to 100% mortality. KHV has caused severe fish losses to ornamental fish trade and carp fishery owners, and continues to pose a significant threat to anyone dealing with or keeping common carp.

Disease causing agent: Cyprinid Herpesvirus III of the family Herpesviridae.

KHV has no implication to human health.

Geographical Distribution

KHV disease was first recorded in Israel in 1998, following large scale farmed fish mortalities. Further losses, attributed to the virus, were later found in Europe, the USA and have subsequently been reported in a number of countries worldwide.

In the UK KHV disease was first detected in imported koi in 2000. It has subsequently spread to angling waters, as well as occurring in ornamental dealers and hobbyists fish stocks.

KHV disease appears to be widespread in ornamental carp. It is possible the disease is widespread in more countries than have previously reported its presence.

Characteristics & clinical signs

The disease may be transmitted horizontally, through contact with infected fish or water, and the disease can possibly spread through contaminated nets and equipment. Vertical transmission cannot be ruled out from infected broodstock eggs. The virus is thought to be shed through faeces and urine, and also possibly via gills and skin.

There are a variety of associated clinical signs with KHV disease. Affected fish may display erratic behaviour, often gathering at water inlets or points of oxygenation, show loss of balance, loss of mucus resulting in dry, rough patches, sloughing of mucus and sunken eyes. The gills generally show the most dramatic clinical signs, characterised by patches of dead tissue. In addition to the direct clinical signs of KHV disease, the gills are often affected by secondary bacteria and fungi infections.

The disease occurs at water temperatures between 16ºC and 28ºC. Acute mortalities occur within this temperature range and are often highest mid-range. Stress may contribute as a 'trigger' before an outbreak of the disease.

Diagnosis

The virus is detected and its identification confirmed by PCR (Polymerase Chain Reaction) from gills and internal organs of affected fish.

Susceptible species

Currently KHV infections have been recorded in common carp, *Cyprinus carpio,* and its different varieties (mirror, leather, koi, ghost koi, etc). There is also evidence KHV disease may occur in common carp hybrids such as goldfish/common carp hybrids and crucian carp/ common carp hybrids.

Treatment and control

There is no treatment for KHV. Fish that recover from KHV disease may carry the virus and act as an infection source.

On confirmation of KHV disease a risk based approach is used to determine controls imposed. This is dependent upon the type of water infected. Other factors include whether it is a fishery or inland water, fish farm, fish dealer or retailer, aquaria or garden pond.

Controls may include the application of movement restrictions to the site, culling of stocks and disinfection of the facility. Investigations into the source of infection will be undertaken by the FHI on a case by case basis.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of KHV disease in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of KHV disease.

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Spring Viraemia of Carp (SVC)

Serious viral disease affecting all varieties of carp and other coarse fish species.

- **External Signs:** include darkening of the skin, protruding eyes, abdominal swelling, pale gills, trailing faecal casts and protusion of the anus
- **Affects:** common carp and other coarse fish species
- **Mortality:** large scale mortalities up to 100%
- **Treatment:** no known treatment or vaccine

Overview

Spring viraemia of carp (SVC) is a viral disease of carp and many other coarse fish species. The disease causes up to 100% mortality and affects fish of all ages. Outbreaks have resulted in significant economic losses in fisheries across England and Wales.

Disease causing agent: A viral disease caused by *Rhabdovirus carpio* of the family Rhabdoviridae.

SVC has no implications for human health.

Geographical Distribution

The disease is widespread in continental Europe and western Eurasia. The first cases seen in the USA occurred in 2002.

SVC was first found in Great Britain in 1976. Outbreaks within Great Britain are infrequent and often associated with illegal movements of fish.

Characteristics & clinical signs

SVC is usually spread horizontally through close contact between infected fish. There is no evidence to suggest the disease can be transmitted vertically through eggs. Some vectors, such as the fish louse *Argulus* spp. and the fish leech *Piscicola geometra,* also transfer the disease to healthy fish.

Outbreaks of SVC occur as water temperatures rise above 5°C in the spring. Maximum mortalities occur between 10°C and 15°C. Mortalities usually decrease at water temperatures over 17°C but may sometimes occur up to 23°C.

Clinical signs vary but include darkening of the skin, swollen eyes, abdominal swelling (dropsy), pale gills, trailing faecal casts and protrusion of the anus. Infected fish may be lethargic, show loss of balance and display areas of bleeding on the gills, skin and internal organs. It is important to note one or more signs may be absent at any stage of the outbreak, especially during the earliest and very late stages.

Susceptible species

SVC affects common carp (including all variants, such as mirror, leather and koi carp), grass carp, bighead carp, silver carp, crucian carp, goldfish, orfe, pike, tench and wells catfish. Fish of all ages are susceptible.

Diagnosis

The virus is isolated in cell culture from internal organs of affected fish and identification confirmed by PCR (Polymerase Chain Reaction).

Treatment and control

There is no known treatment for SVC.

In the event of an SVC outbreak in a UK site, fish movement restrictions are applied. All sites receiving fish from, or supplying fish to a confirmed positive site would be inspected and sampled to establish the infection source.

Movement controls will be lifted following the clearance and disinfection of the infected site under supervision of the Fish Health Inspectorate (FHI), or after a period of negative disease testing results.

Fishery owners and managers must be sure any fish introduced are disease free. Do not buy fish of unknown origin; one 'bargain' fish may cost the stock and reputation of a fishery. Fishery managers should ensure animals in their care are kept according to good husbandry practices.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of SVC in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of SVC.

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Viral Haemorrhagic Septicaemia (VHS)

Serious viral disease of rainbow trout in Europe.

- **External Signs:** haemorrhaging, protruding eyes, anaemia, spiral swimming behaviour
- **Affects:** a wide and increasing range of fish species
- **Mortality:** up to 80% mortalities
- **Treatment:** no known treatment

Introduction

Viral haemorrhagic septicaemia (VHS) is considered the most serious disease of rainbow trout in aquaculture. In affected stocks it can cause up to 80% mortality. The UK was historically free from this disease. However, in spring 2006, a VHS outbreak caused high levels of mortality in a Yorkshire trout farm. This outbreak, which was subsequently controlled and eradicated, resulted in serious economic losses to farms in the designated area.

Disease causing agent: Viral haemorrhagic septicaemia virus (VHSV) of the family Rhabdoviridae.

VHS has no implications for human health.

Geographical Distribution

VHS was first reported in a rainbow trout farm in Denmark. The disease has since caused significant losses in rainbow trout farms in continental Europe. It has also been reported in Japan and Russia. A highly virulent new strain of VHS has recently been reported in the USA, affecting a range of freshwater fish species.

The VHS virus has also been isolated from the marine environment in the Baltic and North seas, the Atlantic Ocean and off the Pacific coast of North America.

Characteristics & clinical signs

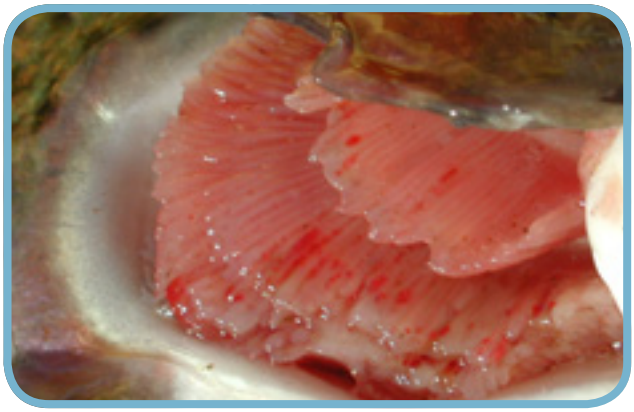
The disease is transmitted horizontally, through contact with infected fish or water. Large numbers of virus particles are shed in the faeces, urine and sexual fluids. There is thought to be no vertical transmission of the virus.

The disease normally progresses in three stages.

1. The acute stage sees a rapid onset of high mortalities, often exhibiting severe clinical signs such as darkening of body colour, protruding eyes (exophthalmia), bleeding around eyes and fin bases, pale gills and pinpoint bruising (petechial haemorrhaging) on the surfaces of the gills, internal organs and in the muscle.

2. During the second sub acute, or chronic, stage mortalities may decline but the body colour continues to darken, protruding eyes may become more pronounced, and bruising around the eyes and fin bases is often reduced. Fish are severely anaemic, and paleness is particularly evident in the abdomen. Fish may develop a spiralling swimming motion, corkscrewing around the body axis.
3. The final, nervous stage, sees reduced mortality. Clinical signs are usually absent other than the corkscrew swimming motion which may become more pronounced.

Outbreaks of VHS in rainbow trout typically occur between temperatures of 7°C and 14°C.



Susceptible species

VHS is principally a disease of farmed rainbow trout, but most salmonid fish are considered susceptible, as are whitefish, grayling and pike. The disease has also been reported in farmed turbot, wild Pacific herring and numerous other marine fish species. The list of host species is increasing substantially with the emergence of the North American strain of VHS.

Diagnosis

The virus is isolated in cell culture from internal organs of affected fish and identification confirmed by ELISA (Enzyme Linked Immunosorbent Assay) and PCR (Polymerase Chain Reaction).

Treatment and control

There is no treatment for VHS.

If VHS occurs on a UK farm the site would be subject to an eradication and disinfection programme. Movement restrictions would be applied to all farms on the river catchment and all contact sites. Attempts would then be made to eradicate the disease. All contact sites would be investigated for evidence of the virus source and spread.

The approved status of any infected area would be suspended until a testing programme has confirmed VHS eradication.

Legal obligation and who to notify

Under the Aquatic Animal Health (England and Wales) Regulations 2009 it is a legal obligation to report suspicion of VHS in any farmed or wild fish to the Fish Health Inspectorate.

It is an offence under the Regulation to fail to inform the FHI of suspicion of VHS.

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