New trends in important diseases affecting the culture of fish and molluscs in the ICES area 2002-2015

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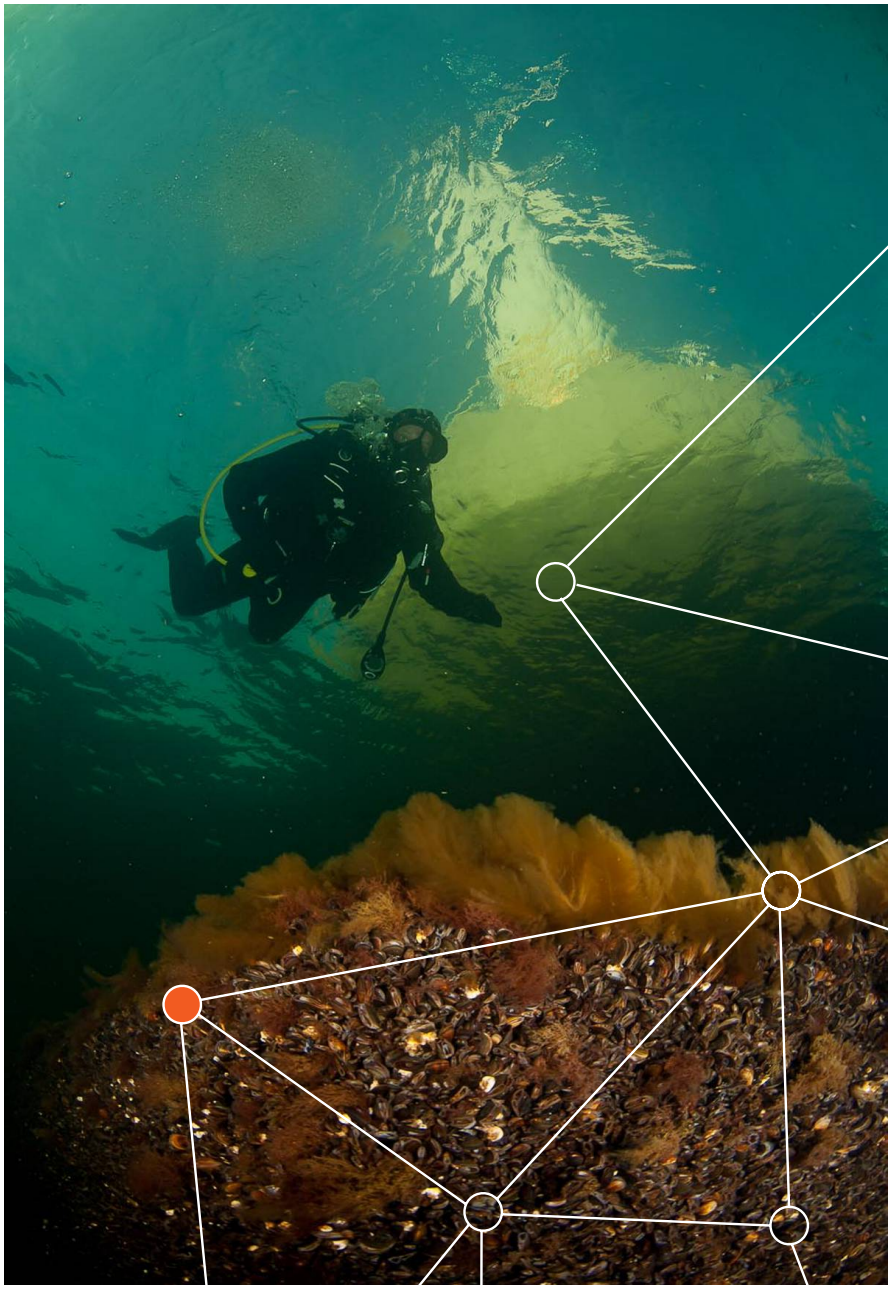

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Editors

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1 Background

The ICES Working Group on Pathology and Diseases of Marine Organisms (WGPDMO) provides annual reviews of national reports on the disease status of wild and farmed fish and molluscs in the ICES area. In 2004, the group published a first report collating this information from 1998-2002. This second report aims to provide an update on the status of the major diseases described in the original report and also to provide an overview of new diseases which have emerged since the previous report was published.

2 Viral Diseases of Farmed Fish

2.1 Infectious salmon anaemia

2.1.1 Description of Agent

Infectious salmon anaemia virus (ISAV), the causal agent of infectious salmon anaemia (ISA) is an enveloped virus consisting of eight single-stranded RNA segments. It is classified as the type species of the genus *Isavirus* within the family *Orthomyxoviridae*. Differentiation of ISAV isolates is based on sequencing a highly polymorphic region (HPR) of the haemagglutinin esterase (HE) gene (Kibenge *et al.*, 2007). All clinical isolates have deletions (HPRΔ) in this region while isolates which do not have deletions in the HPR, (HPR0), are avirulent. It has been shown that HPR0 ISAV is widespread in farmed Atlantic salmon, *Salmo* salar, (McBeath *et al.*, 2009; Christiansen *et al.*, 2011; Godoy *et al.*, 2013).

2.1.2 Geographical Distribution and Temporal Trends

Clinical ISA has now been reported in Norway, Scotland, Faroe Islands, eastern Canada, the eastern USA and Chile. ISA occurs each year in Norway; the number of cases varies annually with 15 cases reported in 2015 (Lyngstad *et al.*, 2008; Hjeltnes *et al.*, 2016). Between 2001 and 2004 an ISA epidemic occurred in the Faroe Islands almost wiping out the Atlantic salmon industry. An ISA contingency plan was introduced in 2005 with the result that no clinical outbreaks of ISA have occurred since then. As part of the plan, all Atlantic salmon going to sea are vaccinated against ISA and a large scale monitoring programme involving monthly samples from all sites is in place. In Scotland, an ISA outbreak occurred in the Shetland Isles in 2009 (Murray *et al.*, 2010). The disease was eradicated with the result that Scotland is once again declared free of ISA. ISA outbreaks occurred in New Brunswick, Canada and Maine, USA between 1998 and 2004 (Gustafson *et al.*, 2007), but there have been no outbreaks at these locations since 2007. However, outbreaks were reported in Nova Scotia and Newfoundland, Canada in 2012 (OIE, 2012). The disease has been reported in farmed Atlantic salmon in Chile since 2007 (Godoy *et al.*, 2008; 2013).

2.1.3 Short Description of Clinical Signs

Infected fish are lethargic, congregate in the upper water level, gasp at the surface, go off feed and hang motionless at the sides of the cage. Affected fish may exhibit exophthalmia, ocular haemorrhage, distended abdomen and/or skin haemorrhage. Internal pathology may include dark, pale or yellow liver, ascites, pale gill and heart, enlarged spleen, petechial haemorrhage in visceral fat and a darkened foregut. Low haematocrit values (< 10) are a typical finding. Histological findings include multifocal haemorrhagic hepatic necroses that may become confluent to give the changes a "zonal" appearance, leaving areas around large veins intact (late stage of disease development). Focal congestion and dilatation of hepatic sinusoids, sometimes with distribution as described for necroses (early stage), and rupture of the sinusoidal endothelium with the presence of erythrocytes within the space of Disse (early sign) are also observed

2.1.4 Control/Preventative Measures

According to the OIE International Aquatic Animal Health Code and EU Directive 2006/88/EC (detection of HPR0 ISAV is not reportable in the EU), ISA is a notifiable disease which means that specific eradication protocols have to be implemented in areas which have been declared disease-free. In Canada ISA is reportable under the Health of Animals Act. ISA control plans are based on active surveillance, vaccination

and eradication of infected cages. These measures are supplemented with increased biosecurity, disease free certification and a greater traceability. The virus is known to be susceptible to a range of common disinfectants (Smail *et al.*, 2004) and to ultraviolet irradiation (Øye and Rimstad, 2001).

2.1.5 Other Host Species

Clinical ISA has only been reported in farmed Atlantic salmon, however the virus has been found in marine farmed rainbow trout, *Oncorhynchus mykiss*, without visible signs of disease. Virus can be detected in wild salmonids only and to date there is no evidence that the virus is present in non-salmonid fish (Raynard *et al.*, 2001; Plarre *et al.*, 2005).

2.2 Viral Haemorrhagic Septicaemia

2.2.1 Description of Agent

The causative agent of viral haemorrhagic septicaemia (VHS) is a rhabdovirus of the genus *Novirhabdovirus* within the family *Rhabdoviridae*. It is a single stranded, enveloped, RNA virus. Sequencing of the VHSV genome has revealed four major genotypes (Snow *et al.*, 2004; Einer-Jensen *et al.*, 2005):

Genotype I: Danish freshwater isolates ($I_{(unclassified)}$); predominantly continental Europe (Ia); northern Europe marine (Ib); continental Europe (Ic); Scandinavia – Baltic Sea and freshwater (Id); Black Sea region (Ie) (Cieslak *et al.*, 2016).

Genotype II: Baltic Sea marine isolates.

Genotype III: North Atlantic marine isolates.

Genotype IV: North American Pacific coast (IVa), Great Lakes (IVb), North American Atlantic coast (IVc), (Cieslak *et al.*, 2016).

2.2.2 Geographic Distribution and Temporal Trends

VHSV has been isolated throughout the Northern Hemisphere from a wide range of marine and freshwater species (Studer and Janies, 2011). VHS (genotype Ia) was reported in a rainbow trout farm in England in 2006 (Stone *et al.*, 2008) and an outbreak of genotype III VHS occurred in wrasse species (ballan, *Labrus bergylta*, corkwing, *Symphodus melops*, cuckoo, *Labrus mixtus*, goldsinny, *Ctenolabrus rupestris* and rock cook, *Centrolabrus exoletus*) held in a marine hatchery in the Shetland Isles, Scotland (Munro *et al.*, 2015). In 2007, the virus (genotype III) was detected in three marine rainbow trout sites in Norway (Dale *et al.*, 2009). There were two sites positive in 2008, one in 2009 and the country has remained free since. In Finland, VHS (genotype Id) continued to spread after its first isolation in 2000, with 24 farms reported infected by 2004 (Raja-Halli *et al.*, 2006). Denmark has been declared free of VHS after a long term eradication programme, with no clinical outbreaks since 2009 (Bang Jensen *et al.*, 2014). In N. America the majority of isolates belong to genotype IVa (Garver *et al.*, 2013). In 2005, VHS (genotype IVb) occurred for the first time in the Great Lakes Basin, North America, affecting a number of different species in Lake St Claire and Lake Ontario (Elsayed *et al.*, 2006).

2.2.3 Short Description of Clinical Signs

The clinical signs of classical VHS in freshwater salmonids are dark skin and pale gills, petechiae in the gills and in the skin, and haemorrhages in the orbits and exopthalmia.

Widespread petechiae developing to haemorrhages are observed in the peritoneal surfaces, in the swimbladder, in the skeletal muscles and in the meninges. The liver is pale with haemorrhages, and the spleen is often enlarged and reddish. High mortality is observed in the acute phase of the disease.

The clinical signs vary among species of marine fish. In Pacific salmonids, VHSV was isolated from fish without clinical signs. In Pacific cod (*Gadus macrocephalus*) and herring (*Clupea harengus pallasi*), infection appeared to be associated with skin lesions, while other Pacific fish species from which VHSV was isolated did not display any gross clinical signs. In European waters, the clinical signs in farmed salmonids and turbot, *Scophthalmus maximus,* were almost identical: classical signs were observed in freshwater salmonids, whereas VHSV in most other fish species was isolated from specimens showing no clinical signs.

2.2.4 Control/Preventative Measures

According to the OIE Aquatic Animal Health Code and EU Directive 2006/88/EC, VHS is a notifiable disease which means that, should the disease occur, specific eradication protocols have to be implemented in areas which have been declared disease-free. Strict biosecurity protocols are essential for remaining disease free. Øye and Rimstad (2001) demonstrated that VHSV is sensitive to ultraviolet irradiation.

2.2.5 Other Host Species

VHS virus has been isolated from 48 different marine and freshwater fish species in the Northern hemisphere (Skall *et al.*, 2005). The updated annex IV of EU Directive 2006/88/EC lists the following species as susceptible to VHSV, herring (*Clupea* sp.), whitefish (*Coregonus* sp.), pike (*Esox lucius*), haddock (*Gadus aeglefinus*), Pacific cod, Atlantic cod (*G. morhua*), Pacific salmon (*Oncorhynchus* sp.), rainbow trout, rockling (*Onos mustelus*), brown trout (*Salmo trutta*), turbot, sprat (*Sprattus sprattus*) and grayling (*Thymallus thymallus*). It is likely that wrasse will be added to this list in the near future (Munro *et al.*, 2015).

2.3 Pancreas Disease

2.3.1 Description of Agent

Pancreas disease (PD) is caused by the salmonid alphavirus (SAV), a member of the genus *Alphavirus* of the family *Togaviridae* (McLoughlin and Graham, 2007). Based on sequence analysis, six subtypes of SAV have been reported. In general, SAV1, 4, 5 and 6 have been associated with PD in Ireland or Scotland and SAV3 causes PD in Norwegian farmed Atlantic salmon (Fringuelli *et al.*, 2008). SAV2 has been associated with sleeping disease in freshwater rainbow trout, however it has now been isolated from diseased Atlantic salmon in Scotland and Norway (Graham *et al.*, 2012).

2.3.2 Geographical Distribution and Temporal Trends

Pancreas disease has emerged as a significant disease of farmed Atlantic salmon in Ireland, Scotland and Norway (McLoughlin and Graham, 2007). The disease is endemic in Ireland (Rodger and Mitchell, 2007), and has increased significantly in Scotland as well (Lester *et al.*, 2011). PD is a listed disease in Norway and an endemic zone was established for SAV3 cases in 2007 (Jansen *et al.*, 2010). A second zone was established for the newly identified SAV2 in 2012 (Hjortaas *et al.*, 2013). In 2015, there were 137 cases of PD registered in Norway (Hjeltnes *et al.*, 2016). There have been no reports of PD in Chile or North America. Sleeping disease of freshwater reared rainbow trout has

been reported in Scotland, England, France, Spain, Italy (Graham *et al.*, 2007b), Germany (Bergmann *et al.*, 2008) and Croatia (Vardić Smrzlić *et al.*, 2013).

2.3.3 Short Description of Clinical Signs

Early clinical signs include a cessation of feeding, lethargy and the observation of yellow faecal casts in the water column. A small percentage of survivors typically fail to thrive and become runts. The changes most commonly found in clinically diseased fish were severe loss of exocrine pancreatic tissue, cardiomyocytic necrosis and heart inflammation, inflammation of the red skeletal muscle and degeneration of white skeletal muscle. Muscle lesions are typical of hyaline degeneration with swollen fragmented eosinophilic sarcoplasm, central migration of myocytic nuclei and subsequent invasion of the sarcoplasm by phagocytic macrophages (McLoughlin *et al.*, 2002; Taksdal *et al.*, 2007).

2.3.4 Control/preventative measures

Pancreas disease was listed by the OIE in 2014 and is a list 3 disease in Norway, but is not a notifiable disease under EU Directive 2006/88/EC. A commercial vaccine is available and is widely used by the industry (Bang Jensen *et al.*, 2012). Regular screening of fish is recommended using serological (Graham *et al.*, 2003) or molecular techniques (Hodneland and Endresen, 2006) as an early indicator of infection. Strict biosecurity protocols are essential and the salmonid alphavirus is known to be sensitive to a range of common disinfectants (Graham *et al.*, 2007a). There are also indications that selective breeding for resistance to SAV infection is possible although research is still ongoing (Norris *et al.*, 2008).

2.3.5 Other Host Species

SAV has only been isolated from farmed Atlantic salmon and rainbow trout. The virus has been detected by PCR in wild flatfish; (common dab *Limanda limanda*, long rough dab *Hippoglossoides platessoides*, and plaice *Pleuronectes platessa*) off the coasts of Scotland (Snow *et al.*, 2010) and in common dab and plaice in Ireland (McCleary *et al.*, 2014). Bruno *et al.* (2014) demonstrated that SAV5 isolated from common dab, off the coast of Scotland could be grown on salmonid cell lines.

2.4 Infectious Pancreatic Necrosis

2.4.1 Description of Agent

Infectious pancreatic necrosis (IPN) is caused by a double-stranded, non-enveloped, RNA virus of the genus *Aquabirnavirus*. Aquabirnaviruses infect a wide range of fish species, aquatic molluscs and crustaceans. Seven genogroups of the IPN virus have been described (Blake *et al.*, 2001; Nishizawa *et al.*, 2005).

2.4.2 Geographical Distribution and Temporal Trends

Historically IPN has been a major problem for freshwater salmonid aquaculture, however with the expansion of the industry it has emerged in the last twenty years as a significant disease of marine farmed Atlantic salmon. In farmed Atlantic salmon, the disease has been reported in Scotland (Bain *et al.*, 2008), Ireland (Ruane *et al.*, 2009; 2015) and Norway (Hjeltnes *et al.*, 2016) where genogroup 5 isolates are the dominant form. The number of cases in Norway is decreasing, with 30 cases reported in 2015 (Hjeltnes *et al.*, 2016) and in Ireland there have been no clinical cases of IPN since 2012 (ICES, 2016). In contrast, IPN has spread from the coastal region of Finland to inland areas since 2012. Both genogroups 2 and 5 are found in Finland, however only genogroup 2

isolates occur in the inland areas (Eriksson-Kallio *et al.*, 2016). IPN has been present in Chile for many years where most isolations are genogroup 5, but genogroup 1 has also been detected (Calleja *et al.*, 2012). Genogroup 1 isolates also dominate in Mexico (Barrera-Mejía *et al.*, 2011). Although clinical IPN has not been recorded in Australia or New Zealand, an aquatic birnavirus which clusters with genogroup 5 isolates was reported in Tasmania (Davies *et al.*, 2010).

2.4.3 Short Description of Clinical Signs

Diseased fish usually appear dark with abdominal distension, particularly in fry. Swimming behaviour is lethargic: a spinning movement (whirling around the longitudinal axis) is characteristic. At necropsy, petechiae in the perivisceral adipose tissue are the most consistent lesions. Fry often show pronounced ascites, whereas post-smolts usually have little or no ascitic fluid and a remarkably dry body muscle. A whitish liver is most commonly found in fry. In the acute stage, extensive necrosis of the exocrine pancreas is the most prominent lesion at histopathological examination. In these lesions, individual acinar cells are seen at different stages of degeneration and cell death. Also, foci with necrotic remnants of exocrine pancreatic cells appearing as an amorphous eosinophilic mass are often found. In liver tissue, similar foci of necrotic eosinophilic hepatocytes may be found. Usually there are small necrotic foci in the pyloric intestinal epithelium and cell debris in the lumen of the pyloric caeca. Some individuals develop a chronic disease with fibroplasias of pancreatic tissue and emaciation (Roberts and Pearson, 2005).

2.4.4 Control/Preventative Measures

The disease has now become so widespread that it is no longer listed by the OIE and very few countries perform screening for the virus. The inland area of Finland has an IPN-free status for genogroup 5 IPNV under EC Decision 2010/221/EU. It is common practice to immunize smolts against IPN before going to sea as a control measure in combination with selective breeding and stricter biosecurity measures. Compared with other fish pathogenic viruses, the IPN virus is more resistant to inactivation (Smail *et al.*, 2003., 1993; Øye and Rimstad, 2001). It is however sensitive to a number of commonly used chlorine and iodine based disinfectants which can be used to inactivate the virus on equipment and the surface of eggs. In recent years the number of cases in Norway have reduced which is believed to be due to the use of more resistant strains of Atlantic salmon (Moen *et al.*, 2009).

2.4.5 Other Host Species

Aquabirnaviruses have been isolated from a variety of wild and farmed teleost fish, molluscs and crustacea in freshwater, estuarine and marine environments (Hill and Way, 1995; Ahne *et al.*, 2003).

2.5 Viral Nervous Necrosis/Viral Encephalopathy and Retinopathy

2.5.1 Description of Agent

Viral nervous necrosis (VNN) is caused by a piscine nodavirus, of the genus *Betanodavirus* from the *Nodaviridae* family which are small, non-enveloped RNA viruses.

2.5.2 Geographical Distribution and Temporal Trends

The disease has been commonly found in the Mediterranean region. Over the last decade nodavirus infections have occurred in marine fish farming in Norway and Scotland, most notably in turbot (Johansen *et al.*, 2004) and Atlantic cod (Hellberg *et al.*, 2010). It also occurs on the Atlantic coast of North America (Johnson *et al.*, 2002).

2.5.3 Short Description of Clinical Signs

Clinical signs are associated with lesions in the brain and retina and include failure to control movement and swim bladder function. Sight and colouration are also affected, however the most significant outcome is mortality, particularly among larvae (Munday *et al.*, 2002). Mortality has also occurred among harvest size sea bass, groupers and Atlantic halibut and may be related to elevated water temperature.

2.5.4 Control/Preventative Measures

VNN is listed by the OIE and is a list 3 disease in Norway, but is not a notifiable disease under EU Directive 2006/88/EC. In the absence of vaccines or treatment, biosecurity is the most effective way to limit the introduction and spread of nodavirus within populations of cultured finfish.

2.5.5 Other Host Species

VNN is associated with mortalities in larvae and juveniles of several marine species worldwide, including haddock *Melanogrammus aeglefinus*, Atlantic halibut *Hippoglossus hippoglossus*, Atlantic cod, turbot, Gilthead sea bream *Sparus aurata* and sea bass *Dicentrarchus labrax*.

2.6 Heart and Skeletal Muscle Inflammation

2.6.1 Description of agent

Heart and skeletal muscle inflammation (HSMI) is believed to be a viral disease although the causative agent has not yet been conclusively identified. Studies have suggested that a novel reovirus, termed piscine reovirus (PRV) may be the causative agent (Palacios *et al.*, 2010). Direct localisation of the virus in heart tissues of HSMI affected fish has indicated an association of the virus with HSMI. The virus has also been detected in healthy farmed and wild fish sometimes at elevated tires similar to those measured in fish diagnosed with HSMI (Garseth *et al.*, 2013). The development of HSMI may involve environmental co-factors which influence the pathogenicity of an infectious agent (Løvoll *et al.*, 2012).

2.6.2 Geographical Distribution and Temporal Trends

HSMI was first reported in farmed Atlantic salmon in Norway in 1999 (Kongtorp *et al.*, 2004a). Outbreaks of HSMI have been officially recorded in farmed salmon in Norway since 2004 and the disease is now widespread in Norwegian salmon aquaculture with 135 cases recorded in 2015 (Hjeltnes *et al.*, 2016). The disease has also been reported in Scotland (Ferguson *et al.*, 2005) and in Ireland (ICES, 2016). The virus was detected, by qPCR, in wild Atlantic salmon broodstock and progeny for the first time in Denmark in 2014 (ICES, 2016). In 2014, a new disease showing HSMI-like clinical symptoms was reported in freshwater farmed rainbow trout in Norway (Olsen *et al.*, 2015). Outside Europe, HSMI has been described in farmed Atlantic salmon and coho salmon, *O. kisutch*, in Chile (Godoy *et al.*, 2016). The virus has been detected in farmed Atlantic salmon in the Pacific north-west of America and Canada (Siah *et al.*, 2015).

2.6.3 Short Description of Clinical Signs

The disease is most frequently reported in spring and early summer, approximately 6 months after sea transfer. Morbidity can be high and mortality up to 20%. Gross signs include pallor and loose texture of the heart, and pericardial haemorrhage with ascites. Haematocrit tends to be normal. Microscopic lesions include myocardial necrosis and a severe ventricular myocarditis of both compact and spongy layers with a predominantly monocytic infiltrate. Coincident epicarditis is common. Lesions occasionally observed include myocarditis of red skeletal muscle, focal necrosis of liver, and oedema and congestion in several organs (Kongtorp *et al.*, 2004b).

2.6.4 Control/preventative measures

None available. The disease is not listed by the OIE nor under EU Directive 2006/88/EC. In 2014 the disease was delisted in Norway (Hjeltnes, 2016).

2.6.5 Host Species

HSMI has only been diagnosed in farmed Atlantic salmon. The virus has been detected in wild Atlantic salmon and sea trout, *Salmo trutta*, (Garseth *et al.*, 2013) and in a range of marine fish (Wiik-Nielsen *et al.*, 2012a) by qPCR in Norway. The virus has also been detected in pacific salmon, namely coho salmon in Chile (Godoy *et al.*, 2016) and coho and chinook salmon, *O. tshawytscha*, on the Pacific coast of America and Canada (Siah *et al.*, 2015).

2.7 Cardiomyopathy Syndrome

2.7.1 Description of agent

A novel virus, the piscine myocarditis virus (PMCV) has been proposed as the causative agent of cardiomyopathy syndrome (CMS). The virus has been detected in fish with clinical CMS and has also been localised in the infected tissues (Haugland *et al.*, 2011). Infection trials have also supported the hypothesis that CMS has a viral aetiology (Bruno and Noguera, 2009; Fritsvold *et al.*, 2009). The virus may be present in farmed salmon for long periods without any signs of clinical disease (Wiik-Nielsen *et al.*, 2012b).

2.7.2 Geographical Distribution and Temporal Trends

CMS was first diagnosed in Norway in the 1980's and later in Scotland and the Faroe Islands (Brun *et al.*, 2003). A single case of CMS was reported in Ireland with very low mortalities in 2012 (Rodger *et al.*, 2014). The number of cases reported in Norway was 105 in 2015 (Hjeltnes *et al.*, 2016).

2.7.3 Short description of clinical signs

Gross signs include haemopericardium (cardiac tamponade) resulting from rupture of the atrium or sinus venosus. Microscopic lesions include myocardial degeneration and coagulative necrosis associated with the spongious layer of the ventricle and atrium. An infiltrate comprised of lymphocytes and macrophages can be observed.

2.7.4 Control/Preventative Measures

None available. The disease is not listed by the OIE nor under EU Directive 2006/88/EC.

2.7.5 Host Species

CMS has only been diagnosed in farmed Atlantic salmon although the virus has been detected by PCR in wild Norwegian Atlantic salmon (Garseth *et al.*, 2012). In Norway, a range of wild marine fish species have been screened for PMCV by PCR and only samples from the Atlantic argentine, *Argentina silus* were positive (Böckerman *et al.*, 2011).

3 Bacterial Diseases of Farmed Fish

3.1 Francisellosis

3.1.1 Description of Agent

Bacteria belonging to the genus *Francisella* are Gram-negative, non-motile, aerobic, facultative intracellular organisms belonging to the γ-proteobacteria. Most, if not all strains isolated from teleost fish belong to either *F. noatunensis* subsp. *orientalis* in warm water fish species or *F. noatunensis* subsp. *noatunensis* in coldwater fish species (Colquhoun and Duodu, 2011). Within the ICES area, francisellosis, caused by *F. noatunensis* subsp. *noatunensis*, has emerged as a major disease in farmed Atlantic cod (Ottem *et al.*, 2009).

3.1.2 Geographical Distribution and Temporal Trends

Francisellosis was diagnosed for the first time in farmed Atlantic cod in 2004 in Norway (Olsen *et al.*, 2006), however the bacteria (*F. noatunensis* subsp. *noatunensis*) have also been detected in archived samples of wild-caught cod in the North Sea (Zerihun *et al.*, 2011). The bacteria have also been detected by real-time PCR in wild cod along the Norwegian coast line (Ottem *et al.*, 2008). There was one reported case of francisellosis in Norway in 2014 and none in 2015, down from a peak of 14 in 2008 (Hjeltnes *et al.*, 2016). In 2009, the disease occurred in Ireland in a stock of wild-caught cod held in captivity as broodstock (Ruane *et al.*, 2015).

3.1.3 Short description of clinical signs

Francisellosis is principally a chronic systemic granulomatous inflammatory disease with varying degrees of mortality. The gross signs observed among most host species are similar and include disseminated white nodules of various sizes in liver, spleen and kidney although most tissues and organs may be affected. Splenomegaly and serosanguinous ascites has been observed in cod. Microscopic lesions include widespread chronic granulomatous inflammation in all organs, associated with variable numbers of Gram-negative bacteria (Birkbeck *et al.*, 2011).

3.1.4 Control/Preventative Measures

None available. The disease is not listed by the OIE nor under EU Directive 2006/88/EC, but is a list 3 disease in Norway.

3.1.5 Host species

F. noatunensis subsp. *noatunensis* has primarily been reported infecting Atlantic cod, however there is a single report of francisellosis in Atlantic salmon in Chile (Birkbeck *et al.*, 2007).

3.2 Rainbow Trout Fry Syndrome/Bacterial Coldwater Disease

3.2.1 Description of Agent

Flavobacterium psychrophilum is the causative agent of Rainbow Trout Fry Syndrome and Bacterial Coldwater Disease. *F. psychrophilum* is a Gram-negative, slender, flexible rod that displays gliding motility. The bacterium requires specific media for isolation and growth, e.g. tryptone yeast extract salts agar (Holt *et al.*, 1993). The name "psychrophilum" refers to the low growth optimum of this bacterium, which is around

15°C *in vitro* (Holt *et al.* 1993) with no growth above 25°C. Other features of this bacterium are its high proteolytic activity as well as its lack of growth in media with > 1% salt under laboratory conditions (Dalsgaard and Madsen, 2000).

3.2.2 Geographical Distribution and Temporal Trends

F. psychrophilum was originally isolated from diseased juvenile coho salmon in the USA in the 1940s (Borg, 1948, 1960), and was first found outside North America during the 1980s (see Nematollahi *et al.*, 2003), where it appeared in Europe, South America, Japan, Korea and Australia. It has for many years been considered an obligate freshwater pathogen due to its sensitivity to salt, but there have been reports of disease outbreaks with *F. psychrophilum* in rainbow trout reared in brackish water in Finland (ICES, 2001) and spawning bream, *Abramis brama*, on the Baltic coast of Sweden (ICES, 2013). In 2008, *F. psychrophilum* was isolated for the first time in farmed Atlantic salmon in Norway, causing septicaemia and mortalities (Nilsen *et al.*, 2011a). The disease has regularly caused high mortalities among rainbow trout farmed in the Scandinavian countries such as Norway (Nilsen *et al.*, 2011b). Baltic salmon brood fish have been shown to be carriers of *F. psychrophilum* during their spawning migration (Ekman *et al.* 1999), and laboratory experiments with water microcosms have shown that the bacterium is able to survive salinities up to 0.6‰ (Madetoja *et al.*, 2003). In all, this means that *F. psychrophilum* is able to adapt to more saline environments than originally expected (Nilsen *et al.*, 2011b).

3.2.3 Short description of clinical signs

Disease caused by *F. psychrophilum* results in septicaemia and/or skin lesions and fin rot (Nematollahi *et al.*, 2003). In salmonid fry, the primary sign will be high mortalities due to septicaemia, up to 90 % in an affected population, whereas larger fish display skin lesions with lower mortalities (Borg, 1960; Holt *et al.*, 1993; Nematollahi *et al.*, 2003).

3.2.4 Control/Preventative Measures

F. psychrophilum disease outbreaks can be treated with antibiotics such as oxytetracycline, amoxicillin, oxolinic acid and florfenicol (Barnes and Brown, 2011), however resistance to antibiotic treatments is becoming an issue (Bruun *et al.*, 2003). There are no registered vaccines against the disease at present, therefore prophylactic measures like good management procedures (including egg disinfection) as outlined by Madsen and Dalsgaard (2008) are recommended. Phage therapy as a potential method for controlling *F. psychrophilum* infections is also being investigated (Madsen *et al.*, 2013).

3.2.5 Host species

Juvenile rainbow trout and coho salmon are particularly susceptible (Nematollahi *et al.*, 2003). However, *F. psychrophilum* infections have been reported in a wide range of both anadromous and non-anadromous salmonids of various sizes. In addition, *F. psychrophilum* has either caused disease or been detected in Japanese eel *Anguilla japonica*, European eel *Anguilla anguilla*, common carp *Cyrpinus carpio*, crucian carp *Carassius carassius*, tench *Tinca tinca*, ayu *Plecoglossus altivelis*, pale chub *Zaco platypus*, perch *Perca fluviatilis* and roach *Rutilis rutilis* (Barnes and Brown, 2011).

3.3 Enteric Redmouth Disease

3.3.1 Description of Agent

The aetiological agent of enteric redmouth disease (ERM) is *Yersinia ruckeri*, a gram negative, slightly curved, rod shaped bacteria. Most of the bacteria are motile due to the presence of flagellae (Barnes, 2011). Strains of *Y. ruckeri* can be classified on the basis of biotype, serotype and outer-membrane protein (OMP) type (Tobback *et al.*, 2007) however a combination of these is useful for discriminating between strains (Davies, 1991; Wheeler *et al.*, 2009). The most common *Y. ruckeri* serovar is O1, with the most virulent being the O1a serotype Hagerman strain (Davies, 1991). Serovar O1 *Y.ruckeri* grow well on tryptone soya agar with or without 5% blood. After a 48 h incubation at 20-25°C, round, raised, shiny off-white colonies of 2-3 mm in diameter develop (Austin and Austin, 2007). Over the last decade, a new biotype 2 strain has emerged causing mortalities in Europe (Wheeler *et al.*, 2009). There were 34 confirmed sites affected by ERM in Norway in 2015 (Hjeltnes *et al.*, 2016).

3.3.2 Geographical Distribution and Temporal Trends

ERM was first described in the USA in 1955 and became widespread in farmed salmonid species throughout the USA and Canada (Furones *et al.*, 1993). The disease is now present throughout Europe, Australia, Chile and South Africa (Barnes, 2011). The most probable route of spread has been by horizontal transmission through the import of live fish and ova. In recent years, Biotype 2 strains have emerged causing significant losses in rainbow trout facilities in the UK (Austin *et al.*, 2003), Spain (Fouz *et al.*, 2006), USA (Arias *et al.*, 2007) and Finland (ICES, 2012).

3.3.3 Short description of clinical signs

ERM can affect fish of all ages, but is most acute in juveniles, while in larger fish the disease appears as a more chronic condition. The characteristic haemorrhages around the oral cavity led to the name 'redmouth' disease, although this is often not apparent in many cases of disease (Tobback *et al.*, 2007). The disease is mainly seen as a generalised haemorrhagic septicaemia with anorexia, darkening of the skin, haemorrhages in the skin, gills and at the base of the fins and the lateral line. Bleeding is also common in internal organs; swimbladder, liver, spleen, pancreas and visceral fat in connection with the pyloric caecae. Kidney and spleen are swollen and darkened in colour (Roberts, 2012; Barnes, 2011). McArdle (2014) recently reported that heart pathology was a consistent finding in outbreaks in Ireland over a number of years and may explain the behavioural changes of diseased fish.

3.3.4 Control/Preventative Measures

ERM has successfully been controlled by the use of a monovalent vaccine based on a Hagerman type strain, biotype 1. Following reports of vaccine failures, connected to new strains classified as biotype 2, new vaccines contain antigens from both the Hagerman type strain and Biotype 2 (Deshmuk *et al.*, 2012). This vaccine offers good protection when administered by intra-peritoneal injection supplemented with an additional booster by immersion (Chettri *et al.*, 2013). Antibiotics also play a role in controlling infection as *Y. ruckeri* is sensitive to a range of antibiotics (e.g. oxytetracycline, oxolinic acid and potentiated sulphonamides), however there are increasing reports of antibiotic resistance (Tobback *et al.*, 2007).

3.3.5 Host species

ERM is primarily a disease infecting salmonid fish species, particularly rainbow trout. The agent has also been isolated from a wide range of non-salmonid fish species, though not all exhibited pathology related to infection with *Y. ruckeri* (Furones *et al.*, 1993).

3.4 Red Spot Disease/Pseudomoniasis

3.4.1 Description of Agent

Pseudomona anguilliseptica, a Gram-negative motile rod, is an opportunistic pathogen for a variety of fish species cultured in marine and brackish waters worldwide. It is the aetiological agent of 'red spot disease' also known as 'Sekiten-byo' disease of eels. The bacteria can be isolated from blood, kidney, liver and spleen samples on a nutrient agar, supplemented with 10% blood. The bacteria are quite slow growing and incubation at 20-25°C should continue for at least 7 days, when round, raised, shiny, pale-grey colonies, less than 1 mm in diameter develop (Wakabashi and Egusa, 1972). Serologically, two major groups have been established on the basis of their O-antigens with serotype O2 including the majority of eel isolates and serotype O1 including isolates from all other fish species (Lopez-Romalde *et al.*, 2003).

3.4.2 Geographical Distribution and Temporal Trends

P. anguilliseptica has been reported from Japan and Taiwan (Wakabayashi and Egusa, 1972; Nakai *et al.*, 1985), Kuwait (Al-Marzouk, 1999) and in a number of European countries such as Scotland, Finland, France, Spain and The Netherlands (Stewart *et al.*, 1983; Wiklund and Bylund, 1990; Berthe *et al.*, 1995; Domenech *et al.*, 1999; Haenan and Davidse, 2001). In Finland, *P. anguilliseptica* has become a significant cause of disease outbreaks in rainbow trout reared in brackish water during extremely warm summers when water temperatures rise above 20°C and rearing conditions are poor (ICES, 2012).

3.4.3 Short description of clinical signs

Diseased fish have pethechial haemorrhage of the skin, peritoneum and liver. Kidney can have liquefactive necrosis (Wakabashi and Egusa, 1972; Ellis *et al.*, 1983; Wiklund and Bylund, 1990). Diseased rainbow trout in Finnish brackish water farms appear dark in colour, with a sluggish appearance and large hyperaemic and odematic areas are observed on the sides of the fish.

3.4.4 Control/Preventative Measures

In Finland *P. anguilliseptica* infections in rainbow trout have successfully been treated with Sulfa-Trimetoprim (30 mg/Kg for 7 days) in combination with improve husbandry on the farms. There is no comercial vaccine available in Europe.

3.4.5 Host species

P. anguilliseptica was first described as red spot disease of Japanese eels (Wakabashi and Egusa, 1972). Since then, the pathogen has been recorded in a range of fish species including European eel, (Stewart *et al.*, 1983), rainbow trout, sea trout, white fish *Coregonus sp.*, Baltic herring, *Clupea harengus membras* (Wiklund and Lönnström, 1994; Lönnström *et al.*, 1994), sea bass, *Dicentrarchus labrax* (Berthe *et al.*, 1995), Gilthead sea bream, *Sparus aurata* (Domenech *et al.*, 1999), orange-spotted grouper, *Epinephelus coioides* (Al-Marzouk, 1999), Atlantic cod, (Ferguson *et al.*, 2004) and turbot (Magi *et al.*,

2009). *P. anguilliseptica* has also been isolated from lumpsucker (*Cyclopterus lumpus*) used as cleaner fish on Atlantic salmon farms in Norway (Hjeltnes *et al.,* 2016).

4 Parasitic Diseases of Farmed Fish

4.1 Amoebic Gill Disease

4.1.1 Description of Agent

AGD was initially ascribed to *Neoparamoeba pemaquidensis*, based on morphological (Dyková *at al*., 2000) and molecular characterisation (Wong *et al*., 2004). The isolation of *N. branchiphila* from AGD-affected fish (Dyková *et al*., 2005) meant that the disease may have a mixed aetiology. However, issues still remained with the development of a reproducible experimental challenge model using both species (Morrison *et al*., 2004; Vincent *et al*., 2007). Using molecular techniques, Young *et al.* (2008) showed that *N. perurans* was the aetiological agent of AGD which was subsequently confirmed by laboratory trials and fulfilment of Koch's Postulates (Crosbie *et al*., 2012).

4.1.2 Short Description of Clinical Signs

AGD is characterised by multifocal lesions that appear as pale gill tissue, or white mucoid spots and plaques. The main histological feature of the disease is prominent epithelial hyperplasia resulting in a complete lamellar fusion. Large mucous cells are often situated on the surface of the hyperplastic epithelium and between the lamellae, with significant leucocyte infiltration (Mitchell and Rodger, 2011).

4.1.3 Geographical Distribution and Temporal Trends

The disease has long been associated with farmed Atlantic salmon in Tasmania (Munday *et al*., 1993) and has also been reported in farmed Atlantic salmon in Ireland (Palmer *et al*., 1997), Scotland, Norway (Steinum *et al*., 2008) and Chile (Bustos *et al*., 2011). The disease has also been reported in the Mediterranean (Dyková *et al*., 2000; Munday *et al*., 2001) and in South Africa (Mouton *et al*., 2014). The disease re-emerged in Ireland and Scotland in 2012 and in Norway and the Faroe Islands in 2014 (Rodger, 2014; Oldham *et al*., 2016). AGD has also been reported in Canada (ICES, 2016).

4.1.4 Control/Preventative Measures

The recommended treatment for AGD is a 2 – 3 h freshwater bath (Clark *et al*., 2003). Treatment with hydrogen peroxide has also shown to be effective (Adams *et al*., 2012). Regular screening of the gills through gill scoring (Taylor *et al*., 2009) and non-lethal sampling for molecular diagnostics (Downes *et al*., 2017) have proven useful for disease monitoring. AGD is not a notifiable disease.

4.1.5 Host Species

Amoebic Gill Disease (AGD) primarily affects salmonids and was first described in marine reared coho salmon (*S. kisutch*) in Washington and California, USA (Kent *et al*., 1988) and in Atlantic salmon (*Salmo salar*) and rainbow trout (*Oncorhynchus mykiss*) in Tasmania (Munday *et al*., 1993). However, the disease has also been reported in turbot, *Psetta maxima* and sea bass *Dicentrarchus labrax* (Munday *et al*., 2001) and has recently been described in ballan wrasse, *Labrus bergylta* (Karlsbakk *et al*., 2013) and lumpsucker (Hjeltnes *et al*., 2016).

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5 Viral Diseases in Farmed Molluscs

5.1 Ostreid herpesvirus 1 in bivalves

5.1.1 Description of Agent

Ostreid herpesvirus 1 (OsHV-1) is the aetiological agent of a contagious viral disease affecting bivalve species including the Pacific oyster, *Crassostrea gigas*. OsHV-1 particles have been purified from French *C. gigas* larvae (Le Deuff and Renault, 1999) and were observed by transmission electron microscopy to be enveloped icosahedral with electron dense cores and a diameter around 120 nm. The entire virus DNA was sequenced and OsHV-1 capsids appear structurally similar to those of other herpesviruses that have been studied (Davison *et al.*, 2005). The virus was classified under the name Ostreid herpesvirus 1 (OsHV-1) as the first known species in the family Malacoherpesviridae, order Herpesvirales.

Although the aetiological agent is represented by all specimens of OsHV-1 (Arzul *et al.*, 2001; Davison *et al.*, 2005; Moss *et al.*, 2007; Segarra *et al.*; 2010, Martenot *et al.*, 2011; Renault *et al.*, 2012), increased mortality outbreaks recently reported in Europe, Australia and New Zealand among *C. gigas* spat in association with all OsHV-1 µVar viral variants suggest differences in terms of virulence among OsHV-1 lineages. However, the detection of variants related to OsHV-1 µVar have also been reported in the absence of mortality events (Dundon *et al.*, 2011; Shimahara *et al.*, 2012) suggesting the involvement of several factors in disease expression.

5.1.2 Geographical Distribution and Temporal Trends

OsHV-1 representatives have been reported in Europe (France, Ireland, Italy, the Netherlands, Spain, Sweden, United Kingdom), Australia, Brazil, China (People's Rep. of), Korea, Japan, Morocco, Tunisia, Mexico, New Zealand and the United States of America.

Since 2008, widespread mortality was reported among *C. gigas* stocks in different Member States of the European Union in association with emergence of the hypervirulent OSHV-1 µVar variant (Segarra *et al.*, 2010; Peeler *et al.*, 2012; Roque *et al.*, 2012). The outbreaks are seasonal and highly temperature dependent with mortality rates of up to 100% (Clegg *et al.*, 2014). Mortality events attributed to OsHV-1 µVar variants were also reported beginning in 2010 in Australia and New Zealand (Jenkins *et al.*, 2013).

5.1.3 Short Description of Clinical Signs

The virus can be found in adult bivalves in the absence of mortality. Infection-associated lesions in juveniles are mainly observed in connective tissues of all organs in which fibroblastic-like cells exhibit enlarged nuclei with perinuclear chromatin (Renault *et al.*; 1994; Schikorski *et al.*, 2011).

5.1.4 Control/Preventative Measures

The herpes infection affecting bivalves is not listed by the World Organisation for Animal for Animal Health (WOAH) (OIE) and the EU (Directive 2006/88/EC). Pacific cupped oyster families less susceptible to OsHV-1 including the variant OsHV-1 µVar can be produced for aquaculture use (Sauvage *et al.*, 2009). Biosecurity may be successfully applied in confined and controlled facilities such as hatcheries and nurseries in order to protect the facility and the surrounding environment from the introduction of the virus.

5.1.5 Host Species

OsHV-1 infection causes mortality in larvae and juveniles of several bivalve species (Renault *et al.*, 1994; Garcia *et al.*, 2011) including the Pacific oyster, *C. gigas*, Portuguese oyster, *Crassostrea angulata*, suminoe oyster, *Crassostrea ariakensis*, European flat oyster, *Ostrea edulis*, Manila clam, *Ruditapes philippinarum*, carpet shell clam, *Ruditapes decussatus*, and great scallop, *Pecten maximus*. However, the variant µVar (Segarra *et al.*, 2010) primarily infects *C. gigas*.

6 Bacterial Diseases of Farmed Molluscs

6.1 *Vibrio* sp. infecting marine molluscs

6.1.1 *Vibrio splendidus* infecting Pacific oysters

6.1.1.1 Description of Agent

Gram-negative bacteria related to *Vibrio splendidus* are frequently found in coastal areas and can infect and induce mortalities in the Pacific oyster, *C. gigas* (Lacoste *et al.*, 2001; Samain *et al.*, 2004; Saulnier *et al.*, 2010). Through epidemiological studies a high genetic diversity was observed in this group suggesting that the *splendidus* clade is diverse and possibly polyspecific (Le Roux *et al.*, 2002).

6.1.1.2 Geographical Distribution and Temporal Trends

Since 2008, widespread mortality events have been reported among *C. gigas* in France. The outbreaks are seasonal and most frequently affect spat and juveniles (< 18 months). Mortality rates of 40-100% were experienced. While the mortality in young oysters has generally been attributed to emergence of the OsHV-1 µVar (EFSA, 2010; Segarra *et al.*, 2010), *V. splendidus* was frequently detected in affected, as well as unaffected, oysters (EFSA, 2010).

6.1.1.3 Short Description of Clinical Signs

The main sign of *V. splendidus* infections in the Pacific oyster remains reports of mortality events (Saulnier *et al.*, 2010). There are no reliable clinical indicators of *V. splendidus* infection in oysters.

6.1.1.4 Control/Preventative Measures

V. splendidus infection is not listed by the World Organisation for Animal Health (WOAH) and the EU (Directive 2006/88/EC).

6.1.1.5 Host species

V. splendidus-related species were reported in association with mortality outbreaks affecting different mollusc species (Sugumar *et al.*, 1998; Macian *et al.*, 2000; Lacoste *et al.*, 2001; Gay *et al.*, 2003; Gay *et al.*, 2004).

6.1.2 *Vibrio aestuarianus* infecting oysters

6.1.2.1 Description of Agent

The gram-negative bacterium *Vibrio aestuarianus* is frequently found in coastal areas and can infect and induce mortality outbreaks among the Pacific oyster, *C. gigas* (Samain *et al.*, 2004; Garnier *et al.*, 2008; Saulnier *et al.*, 2010).

6.1.2.2 Geographical Distribution and Temporal Trends

V. aestuarianus has been reported to be associated with mortality of oyster reared in open marine waters in France. Some bacterial isolates related to this species were demonstrated to be pathogenic to *C. gigas* under experimental conditions (Garnier *et al.*, 2007; Azandegbe *et al.*, 2010).

6.1.2.3 Short Description of Clinical Signs

V. aestuarianus has frequently been among the pathogens associated with massive mortality events occurring during summer in *C. gigas* oysters. These events often occur

when seawater temperatures reach 19°C on the French Atlantic coast (Garnier *et al.*, 2007; Labreuche *et al.*, 2010). Classic bacteriology studies revealed that moribund animals were predominantly infected with *V. aestuarianus*, found in the hemolymph but also in other oyster tissues (Azandegbe *et al.*, 2010).

6.1.2.4 Control/Preventative Measures

V. aestuarianus infection is not listed by the World Organisation for Animal Health (WOAH) or the EU (Directive 2006/88/EC).

6.1.2.5 Host species

The gram-negative bacterium *V. aestuarianus* is mainly reported infecting the *C. gigas.* However, *V. aestuarianus* has also been recently detected in the cockle *Cerastoderma edule* in France in association with mortality outbreaks (C. Garcia *et al.*, IFREMER, pers. comm.).

6.1.3 *Vibrio harveyi* in abalone

6.1.3.1 Description of Agent

The gram-negative bacterium *Vibrio harveyi* is known to be highly pathogenic for the European abalone *Haliotis tuberculata*. Since 1998, specific strains of *V. harveyi* have been implicated in mortality outbreaks in French farms and field stocks of abalone (Nicolas *et al.*, 2002). *V. harveyi* has been widely recognized as a common pathogen of many commercially cultured fish and shellfish species worldwide (Gomez *et al.*, 2004) including abalone in Australia and Japan (Nishimori *et al.*, 1998; Handlinger *et al.*, 2005; Sawabe *et al.*, 2007).

6.1.3.2 Geographical Distribution and Temporal Trends

The bacterium has been involved in recurrent mortality outbreaks occurring seasonally, at the end of warm season, since 1998 in farms and field stocks of *H. tuberculata* in France (Nicolas *et al.*, 2002).

6.1.3.3 Short Description of Clinical Signs

Although non-specific, clinical signs of *V. harveyi* infection include a loss of muscular strength occurring concomitantly with the appearance of white pustules on the foot. Subsequently, diseased animals develop a fatal septicemia leading to up to 80% mortality within a few days to 3 weeks. Vibriosis outbreaks in *H. tuberculata* cultivated in France were shown to be driven by seawater temperature exceeding a 17°C threshold (Huchette and Clavier, 2004) and host physiology such as gametogenesis and reduced immune defense capacities (Travers *et al.*, 2008).

6.1.3.4 Control/Preventative Measures

V. harveyi infection is not listed by the World Organisation for Animal Health (WOAH) (OIE, 2012) and the EU (Directive 2006/88/EC).

6.1.3.5 Host species

Vibrio harveyi infects members of the genus *Haliotis,* but has been also reported in bivalve molluscs (Saulnier *et al.*, 2010).

6.2 Nocardia crassostreae in oysters

6.2.1 Description of Agent

Nocardia crassostreae, a gram-positive actinomycete, is the causative agent of Pacific Oyster Nocardiosis (PON). The cycle of *N. crassostreae* in the environment between oysters is unknown. As most *Nocardia* species are soil bacteria this may suggest that *N. crassostreae* is acquired from the environment as an opportunistic invader of live oysters (Bower *et al.*, 2005). The soil substrate could be a natural source of *N. crassostreae*.

6.2.2 Geographical Distribution and Temporal Trends

The disease has been reported since the late 1940s in Japan and the west coast of North America in association with *C. gigas* field mortality outbreaks (Friedman *et al.*, 1991; Bower *et al.*, 2005). The extent of associated mortalities has not been accurately measured but estimated at about 35% in some localities. In British Columbia (Canada), European flat oysters *O. edulis* cultured alongside infected *C. gigas* have been found infected by *N. crassostreae* but mortality rate is unknown.

Over the last decade, the geographical distribution of *N. crassostreae* has extended outside the North Pacific with the report of *N. crassostreae* in *C. gigas* from the Netherlands (Engelsma *et al.*, 2008) and in *Mytilus galloprovincialis* and *O. edulis* from Italy (Carella *et al.*, 2013).

6.2.3 Short Description of Clinical Signs

The bacterium can be found all year as bacterial foci primarily in gonad follicles, vesicular connective tissue, gills, heart and adductor muscle, but they can invade every tissue. Bacteria are usually associated with mortalities during the late summer and fall. *C. gigas* oysters experimentally infected showed clinical signs and mortality (Friedman *et al.*, 1991; Friedman *et al.*, 1998).

6.2.4 Control/Preventative Measures

N. crassostreae infection is not listed by the World Organisation for Animal Health (OIE) or the EU (Directive 2006/88/EC). Bottom culture of oysters may possibly expose oysters more to *N. crassostreae* than other types of culture and so may be avoided to potentially mitigate infection pressure where it is intense.

6.2.5 Host species

N. crassostreae causes infection in the oysters *C. gigas* and *O. edulis* and was recently described in the mussel *M. galloprovincialis* (Carella *et al.*, 2013).

6.3 *Candidatus* xenohaliotis californiensis in abalone

6.3.1 Description of Agent

Candidatus xenohaliotis californiensis is an intracellular bacterium (332 × 1550 nm in the bacillus form and an average of 1405 nm in the spherical morphotype) in the family Anaplasmataceae and is closely related to members of the genera *Ehrlichia*, *Anaplasma* and *Cowdria* (Friedman *et al.*, 2000). Infection has been reported in wild and farmed abalone, *Haliotis* spp. (Archeogastropoda: Mollusca) (Gardner *et al.*, 1995; Friedman *et al.*, 2000). The disease caused by this bacterium is known as withering syndrome and may be more appropriately termed abalone rickettsiosis.

6.3.2 Geographical Distribution and Temporal Trends

Candidatus xenohaliotis californiensis accurs along the south-west coast of Noth America in California, USA and Baja California, Mexico. However, as infected abalone have been transported to Chile, Japan, Israel, Iceland and possibly other countries, a large geographical distribution of the bacterium is suspected. Infections have resulted in severe economic impacts to abalone culturists along the west coast of North America. Reduced profits have been associated with farm closures as well as contributing to the closure of commercial fisheries in California. Moreover, recurring disease outbreaks are also implicated in failure of wild abalone to repopulate historic habitats (Friedman and Finley, 2003). *Candidatus* xenohaliotis californiensis has now been detected in Europe including Ireland, Spain and France in the European abalone, *H. tuberculata* (Balseiro *et al*., 2006).

6.3.3 Short Description of Clinical Signs

Clinical disease is typically observed in abalone over 12 months of age. Clinical disease has only been observed in infected individuals exposed to elevated seawater temperatures (e.g. 18°C). Gross signs of the infection include pedal atrophy, mottled digestive gland, anorexia, weakness, and lethargy before death. Associated losses may reach 99% of the population and depending on seawater temperatures and host species.

6.3.4 Control/Preventative Measures

The most effective prevention is avoidance of the pathogen. Infection caused by *Candidatus* xenohaliotis californiensis in molluscs is not listed as a notifiable disease under EU legislation (Directive 2006/88/EC). Infection is however included in the Manual of Diagnostic Tests for Aquatic Animals (www.oie.int) and an OIE Reference Laboratory for this pathogen has been designated. Should infection occur, holding abalone at temperatures below 15°C may reduce pathogen transmission and subsequent disease development. Application of oxytetracycline reduces losses.

6.3.5 Host species

Candidatus xenohaliotis californiensis infects members of the genus *Haliotis* and natural infections have been observed in black abalone (*H. cracherodii*), white abalone (*H. sorenseni*), red abalone (*H. rufescens*), pink abalone (*H. corrugata*), green abalone (*H. fulgens*), the small abalone (*H. diversicolor supertexta)*, and the European abalone (*H. tuberculata*) in the wild or in culture facilities, as well as flat (*H. wallalensis*) and Japanese abalone (*H. discus-hannai*) in laboratory challenges. Other abalone species have not been tested.

7 Diseases of Mollusc: Parasitic Diseases

7.1 *Bonamia exitiosa*

7.1.1 Description of Agent

Bonamia exitiosa is a protozoan parasite in the Haplosporida, phylum Cercozoa (Cavalier-Smith and Chao, 2003), infecting haemocytes of several oyster species and inducing physiological disorders and eventually death of the animal (Dinamani *et al.*, 1987; Cranfield *et al.*, 2005). It is an intrahaemocytic protozoan, but it can be observed extracellularly (Dinamani *et al.*, 1987). This intrahaemocytic protozoan quickly becomes systemic and can be found in different organs, especially in connective tissues (Hine, 1991).

7.1.2 Geographical Distribution and Temporal Trends

Infection with *B. exitiosa* is found in oyster *Ostrea chilensis* in the Foveaux Strait and other locations around South Island, New Zealand (Dinamani *et al.*, 1987); and in *Ostrea angasi* and *Saccostrea glomerata* in New South Wales, Victoria, Tasmania, and Western Australia (Hine and Jones, 1994; Hine, 1996; Corbeil *et al.*, 2006; Carnegie *et al.*, 2014).

More recently, infection with *B. exitiosa* was reported in *O. edulis* in Galicia (Spain) (Abollo *et al.*, 2008), in the Adriatic Sea in Italy (Narcisi *et al.*, 2010), in the Mediterranean Sea in France and in Cornwall in the United Kingdom as well as in *Ostrea stentina* in Tunisia (Hill *et al.*, 2010). Molecular analyses have confirmed the presence of the parasite in the Americas as well, infecting *O. stentina* (= *Ostreola equestris*) along the southeastern Atlantic coast of the USA, *Ostrea lurida* in California, USA, and *Ostrea puelchana* in Argentina (Hill *et al.*, 2014).

7.1.3 Short Description of Clinical Signs

Infection can be lethal. In *O. chilensis*, death usually occurs as infections peak in intensity, particularly in association with high intensity apicomplexan infections (Hine and Wesney, 1994; Hine *et al.*, 2002). In one episode the disease killed more than 80% of *O. chilensis* as a wave of infection passed through an oyster bed over 2–3 years (Cranfield *et al.*, 2005). The impact of *B. exitiosa* in *O. edulis* or *O. stentina* has not yet been evaluated.

In *O. angasi*, the parasite is epitheliotropic, and apparently very light infections may cause a massive focal haemocyte infiltration with necrotic foci. In *O. edulis*, the parasite is associated with heavy haemocytic infiltration and appears in the connective tissue of different organs mostly within haemocytes, but sometimes outside host cells (Abollo *et al.*, 2008). In *O. stentina*, marked haemocytosis was not observed in animals found to be infected with the parasite (Hill *et al.*, 2010).

7.1.4 Control/Preventative Measures

Infection caused by *B. exitiosa* in molluscs is listed as a notifiable disease by the EU legislation (Directive 2006/88/EC). The infection is also listed by the OIE and included in the Manual of Diagnostic Tests for Aquatic Animals (www.oie.int).

It has been considered that in New Zealand development of lighter dredges and less damaging fishing strategies may reduce the chance of disease outbreaks by lowering disturbance (Cranfield *et al.*, 2005). Avoiding stressors such as exposure to extreme temperatures (below 7 or above 26°C) and salinity (40%), starvation, handling, or

heavy infection with other parasites, as well as decreasing density, should help to reduce the impact of the disease in that system (Cranfield *et al.*, 2005; Hine *et al.*, 2002). Control elsewhere should focus on preventing the introduction of *B. exitiosa* to areas in which it has not yet become established.

7.1.5 Host species

Oyster species *O. chilensis* (= *Tiostrea chilensis* = *T. lutaria*) (Dinamani *et al.*, 1987), *O. angasi* (Hine and Jones, 1994; Hine, 1996; Corbeil *et al.*, 2006), *O. edulis* (Abollo *et al.*, 2008; Narcisi *et al.*, 2010), *O. stentina* (Hill *et al.*, 2010) and *O. puelchana*, *O. lurida*, and *S. glomerata* (Hill *et al.*, 2014).

7.2 *Bonamia ostreae*

7.2.1 Description of Agent

Bonamia ostreae is a protozoan parasite in the Haplosporida (Pichot *et al.*, 1979; Comps *et al.*, 1980; Carnegie *et al.*, 2000), phylum Cercozoa (Cavalier-Smith and Chao, 2003), infecting haemocytes of flat oysters, *O. edulis*, and inducing physiological disorders and eventually death of the animal (Grizel, 1985). It is an intrahaemocytic protozoan, but it can be observed extracellularly.

7.2.2 Geographical Distribution and Temporal Trends

Infection with *B. ostreae* has been found in Europe (France, Ireland, Italy, The Netherlands, Portugal, Spain and the United Kingdom), Canada (British Columbia), the United States of America (California, Maine and Washington states), and New Zealand (Lane *et al.*, 2016). The parasite was also reported for the first time in flat oysters in Denmark in 2014 (ICES, 2015).

7.2.3 Short Description of Clinical Signs

B. ostreae is an intrahaemocytic protozoan but it can be observed extracellularly between epithelial or interstitial cells in the gills and stomach or in necrotic connective tissue areas. Intraepithelial localisation has also been reported in gills (Montes *et al.*, 1994) and the parasite has been reported in ovarian tissue (Van Banning, 1990). Advanced infections can become systemic. *O. edulis* of a year or less in age can develop a high prevalence and intensity of infection with associated mortality within six months of exposure to *B. ostreae* (Lynch *et al.*, 2005). However, individuals older than two years appear to be more susceptible to the disease (Culloty and Mulcahy, 1996; Grizel, 1985; Engelsma *et al.*, 2010). Seed from natural settlements appear to be significantly more parasitised than oyster seed from hatcheries (Conchas *et al.*, 2003). Infection of wild and cultured flat oysters is often lethal, and death usually occurs concurrently with the highest intensity infection level.

7.2.4 Control/Preventative Measures

Infection caused by *B. ostreae* in molluscs is listed as a notifiable disease under EU legislation (Directive 2006/88/EC) and also by the OIE (www.oie.int). Mortalities caused by bonamiosis can be reduced using suspension culture, lower stocking densities or by culturing *O. edulis* with *C. gigas*, which is far less susceptible to infection. Oyster seed from hatcheries are preferred for aquaculture use over seed from natural settlements as the latter appear to be significantly more parasitised (Conchas *et al.*, 2003). Resistant strains of *O. edulis* developed through selective breeding may offer an alternative in

infected areas. Selective breeding has been shown to be effective in reducing susceptibility and mortality caused by *B. ostreae* (Naciri-Graven *et al.*, 1998).

7.2.5 Host species

O. edulis is the only known species that is significantly naturally susceptible to *B. ostreae*, *C. gigas* being only scarcely susceptible at most (Lynch *et al.*, 2010). Infection intensity increases concurrently to mortality with age and/or size of the oysters (Culloty and Mulcahy, 1996; Grizel, 1985).

7.3 Marteiliosis

7.3.1 Description of Agent

Marteiliosis is a disease of marine bivalve molluscs caused by protozoan parasites in the genus *Marteilia* (order Paramyxida, phylum Cercozoa; Cavalier-Smith and Chao, 2003), including *M. refringens*, *M. sydneyi*, *M. chungmuensis*, *M. cochillia* among other described species. *Marteilia* species display a cell-within-a-cell structure in which tricellular spores are produced; the pattern of secondary cells produced within primary cells and tricellular spores within secondary cells distinguishes individual *Marteilia* species (Feist *et al.,* 2009). Sporulation occurs extracellularly within digestive tubule epithelia in *M. refringens* (Herrbach, 1971), *M. sydneyi* (Wolf, 1972), and *M. cochillia* (Carrasco *et al.,* 2011), but within oocytes in Asian *M. chungmuensis* (Comps *et al.,* 1986).

7.3.2 Geographical Distribution and Temporal Trends

M. refringens occurs from France southward along the Atlantic coast of Europe as well as in the Mediterranean Sea at least as far east as Greece, and in Tunisia (Virvilis and Angelidis, 2003; Carrasco *et al.,* 2007; Elgharsalli *et al.*, 2013; Arzul *et al.,* 2014). It contributed to the decline of *O. edulis* oyster populations and remains an economically significant pathogen of flat oysters and mussels. Reports over the last decade have noted the occurrence of *M. refringens* in Sweden (ICES 2010, 2011) and southern England (ICES 2012). *M. cochillia* was discovered in the context of significant cockle (*C. edule*) mortality in the Ebro Delta of Mediterranean Spain in 2008 (Carrasco *et al.*, 2011), and was associated with cockle mortality that approached 100% in the Ría de Arousa of the Atlantic coast of Spain in 2012 (Villalba *et al.,* 2014). It represents a notable new threat to an important European fishery species. *M. sydneyi* and *M. chungmuensis* are economically significant pathogens but remain absent from the ICES area, *M. sydneyi* occurring in eastern Australia (Wolf, 1979) and *M. chungmuensis* in Korea and Japan (Comps *et al.*, 1986; Itoh *et al.,* 2002).

7.3.3 Short Description of Clinical Signs

Marteilia parasites sporulating in host digestive tubule epithelia migrate to that tissue from portals of entry elsewere, primarily the epithelia of palps or stomach in the case of *M. refringens* (Grizel, 1974; Berthe *et al.,* 2004). Infections can be difficult to detect before colonization of the digestive tubules, but parasite proliferation afterward produces sharp increases in infection intensity and marked disruption to digestive tubule structure and function, with emaciation of the host and significant mortality a common result (Figueras and Montes, 1988; Villalba *et al.,* 1993). Gonadal infection by *M. chungmuensis* produces gross nodular lesions in affected Pacific oysters (Bower *et al.,* 2011).

7.3.4 Control/Preventative Measures

Infection with *M. refringens* is a disease listed by both the World Organisation for Animal Health (OIE) and the EU (Directive 2006/88/EC). It is on the US National List of Reportable Animal Diseases and on Canada's list of Federally Reportable Aquatic Animal Diseases - Molluscs. Infection with *M. chungmuensis* is also on the Canadian list, and infection with *M. chungmuensis* and infection with *M. sydneyi* are both under review for US listing. Infection with *M. cochillia* is not listed. As *M. refringens* primarily is a pathogen of warmer estuarine systems, parasite impacts may be minimized by culturing susceptible hosts in higher salinity outer coastal waters and in cooler areas, the parasite requiring temperatures of 17°C or greater to infect oyster hosts (Berthe *et al.*, 2004). *M. refringens* is known to infect copepods *Paracartia grani* (Audemard *et al.*, 2001, 2002) and *P. latisetosa* (Arzul *et al.,* 2014) in addition to its molluscan hosts, but limited understanding of its presumably indirect life cycle otherwise limits options for its control.

7.3.5 Host Species

M. refringens primarily infects oyster *O. edulis* and mussels *M. edulis* and *M. galloprovincialis*, though numerous other molluscs have been proven to be hosts, and there is molecular evidence suggestive of infection in others (www.oie.int). Vigorous debate continues about whether *M. refringens* infecting mussels represents a distinct species, *M. maurini* (Comps *et al.,* 1982). Two distinct genetic lineages of *M. refringens* exist, with parasites on one more likely to infect oysters and parasites on the other to infect mussels (LeRoux *et al.,* 2001; López-Flores *et al.,* 2004, Novoa *et al.,* 2005). While the divergence of these parasite lineages may reasonably be interpreted as a speciation event, imperfect fidelity to host type among the parasites on the two lineages would argue that they should conservatively be regarded as a single species, *M. refringens*, for most effective health management.

M. cochillia infects cockle *C. edule* (Carrasco *et al.,* 2013), *M. sydneyi* infects the Sydney rock oyster *S. glomerata* (Perkins and Wolf, 1976), and *M. chungmuensis* infects the Pacific oyster *C. gigas* (Comps *et al.,* 1986), and possibly suminoe oyster *C. ariakensis* and Manila clam *R. philippinarum* (Yanin *et al.*, 2013).

7.4 Haplosporidiosis

7.4.1 Description of Agent

Haplosporidiosis is caused by protozoan parasites in the Haplosporida, phylum Cercozoa (Cavalier-Smith and Chao, 2003). Members of two genera, *Haplosporidium* and *Minchinia*, are responsible for haplosporidiosis in various molluscs. The parasites present uni- and binucleate cells and plasmodia of varying nuclear counts, in addition to sporogonic forms, with spore structure being used to assign parasites to genus (Burreson and Ford, 2004; Burreson and Reece, 2006). All are believed to be indirectly transmissible through intermediate hosts, none of which have ever been identified. Mollusc pathogens causing haplosporidiosis in the region include *H. nelsoni*, *H. costale, H. armoricanum, H. edule,* and *M. tapetis.*

7.4.2 Geographical Distribution and Temporal Trends

H. nelsoni is the most significant agent of haplosporidiosis. It is native to Pacific oyster *C. gigas* populations in Asia (Burreson *et al.,* 2000), and is associated with established *C. gigas* populations in other parts of the world, including Europe (Renault *et al.* 2000, Lynch *et al.,* 2013) and western Canada (ICES, 2008), though without causing much

disease. It has been far more pathogenic in eastern oyster *C. virginica* along the Atlantic coast of North America, however, where it ranges from Florida to Maine, USA (Ford, 1996) and occurs also in Nova Scotia, Canada. The most significant *H. nelsoni* impacts in recent years have been in the northern part of its distribution, with major epizootics in Nova Scotia begining in 2002 (ICES, 2003) and in Maine in 2010. In the Chesapeake and Delaware Bays of the Mid-Atlantic coast of the USA, however, *H. nelsoni* impacts have been declining with increasing disease resistance in the oyster host (Carnegie and Burreson, 2011; Ford and Bushek, 2012). The increased activity of *H. nelsoni* in the north probably represents the influence of climate change, with the parasite (and possibly its unidentified intermediate host) expanding its distribution northward and encountering relatively naive host populations. In the Mid-Atlantic, the parasite has been long established, providing the host more time to adapt to its presence (Ford and Bushek, 2012).

H. costale occurs from the Mid-Atlantic coast of the USA to the Atlantic Provinces of Canada (ICES, 2003) and reportedly has also been observed more frequently in northern waters, but this could reflect increased surveillance more than differences in pathogen dynamics in those areas. There is no evidence that *H. costale* contributes more than occasionally to substantial oyster mortality in eastern North America. *H. costale* was observed in 2008 in *C. gigas* in British Columbia at 4-10% prevalence (ICES, 2009).

The distribution of *H. armoricanum* includes the Netherlands and both Atlantic and Mediterranean coasts of France (Hine *et al.*, 2007) as well as Spain (Azevedo *et al.*, 1999), with a likely recent observation in Ireland (Lynch *et al.*, 2013). *H. edule* occurs in northwest Spain (Azevedo *et al.*, 2003) and *M. tapetis* occurs in northwest Spain and Portugal (Vilela, 1951; Azevedo, 2001), but both of these pathogens were detected in in 2009 in Wales (ICES, 2010). An undescribed haplosporidian basal to the described genera was observed in 2002 in *R. decussatus* from Spain at high prevalence (to 71%) but without associated mortality (Novoa *et al.*, 2004); this pathogen has not been reported since.

7.4.3 Short Description of Clinical Signs

Haplosporidiosis generally involves invasion of hemal spaces in host connective tissues by uninucleate or plasmodial forms, followed by the production of masses of operculate spores in those tissues. One species of the derived haplosporidian genus *Bonamia*, *B. perspora*, presents a typical haplosporidiosis in its oyster host (Carnegie *et al.*, 2006), but none of the other *Bonamia* species do. *H. nelsoni*, sporulating in host digestive tubule epithelia, is a notable exception with regard to tissue tropism (Couch *et al.*, 1966).

7.4.4 Control/Preventative Measures

No haplosporidiosis is listed as notifiable by either the World Organisation for Animal Health (OIE) or the EU (Directive 2006/88/EC). Infection with *H. nelsoni* is on the list of Federally Reportable Aquatic Animal Diseases – Molluscs for Canada. Limited understanding of the presumably indirect life cycles of these pathogens limits options for their control. Breeding for resistance to *H. nelsoni* in the eastern USA has been highly successful (Haskin and Ford, 1979; Ragone Calvo *et al.*, 2003).

7.4.5 Host Species

H. nelsoni infects oysters *C. virginica* and *C. gigas* (Haskin *et al.* 1966; Burreson *et al.* 2000). *H. costale* infects *C. virginica* (Wood and Andrews, 1962) and has been reported recently from *C. gigas* in British Columbia, Canada. *H. armoricanum* infects oyster *O.*

edulis (Van Banning, 1977). *H. edule* infects cockle *C. edule* (Azevedo *et al.*, 2003). *M. tapetis* infects clam *R. decussatus* (Vilela, 1951).

7.5 Perkinsosis

7.5.1 Description of Agent

Perkinsosis is caused by protozoan parasites in the genus *Perkinsus*, order Perkinsida (Levine, 1978). Infections by *Perkinsus* parasites have been noted in a number of marine molluscs, primarily of the classes Bivalvia and Gastropoda. Seven species are presently accepted, *P. marinus*, *P. olseni*, *P. chesapeaki*, *P. mediterraneus*, *P. beihaiensis*, *P. honshuensis*, and *P. qugwadi*. Of these, *P. marinus* and *P. olseni* have been the most impactful on host populations and associated industries. *Perkinsus* parasites typically occur extracellularly in host connective tissues, with both uninucleate trophozoites and multinucleate schizonts observed, but *P. marinus* displays a distinct tropism for digestive epithelia (Carnegie and Burreson, 2012), a habit that it may share with *P. beihaiensis* (Moss *et al.*, 2008). *P. qugwadi* is unique in expressing zoospore forms within host tissues (Blackbourn *et al.*, 1998).

7.5.2 Geographical Distribution and Temporal Trends

Of the two major *Perkinsus* pathogens, *P. marinus* occurs from the northeastern USA to Mexico in the western Atlantic Ocean and Gulf of Mexico, with colonization of the Pacific coast of Mexico presumed to be a recent event (Caceres-Martinez *et al.*, 2008). It was recently detected as well in Brazil (Da Silva *et al.*, 2013). *P. olseni* was described from Australia (Lester and Davis, 1981) but occurs widely in Asia and southern Europe as well as in New Zealand (Dungan *et al.*, 2007), and more recent work has found it in Uruguay and Brazil along the Atlantic coast of South America (Cremonte *et al.*, 2005; Da Silva *et al.*, 2014). Anthropogenic contributions to the distribution of *P. olseni* are not known.

The recent observations of *P. chesapeaki*, described from the eastern USA, in Europe (Arzul *et al.*, 2012; Carrasco *et al.*, 2014) and Australia (Dang *et al.*, 2015), and of *P. beihaiensis*, described from China (Moss *et al.*, 2008), in Brazil (Sabry *et al.*, 2009) and India (Sanil *et al.*, 2012) are noteworthy. As for *P. olseni*, however, it is not clear to what extent these observations represent emergence of established pathogens in new locations; the discoveries may be a product of increased surveillance. *P. qugwadi* reemerged at one location in British Columbia, Canada, in 2011 after not having been observed anywhere since 1997 (Itoh *et al.*, 2013).

7.5.3 Short Description of Clinical Signs

While very small (2-3 μm) *P. marinus* can be phagocytosed by host hemocytes and distributed generally through host hemal spaces, larger *Perkinsus* species are frequently observed as clusters or masses of parasite cells surrounded and sometimes encapsulated by host hemocytes (Blackbourn *et al.*, 1998; McLaughlin and Faisal, 1998; Burreson *et al.*, 2005; Park *et al.*, 2006). Infections reaching high intensities can be lethal, and infection by *P. marinus* has caused mortality exceeding 70% (Burreson and Ragone Calvo, 1996). Emaciation has long been considered a hallmark of perkinsosis caused by *P. marinus* and continues to be observed in association with heavy infections, although this clinical presentation can have numerous other causes (Carnegie and Burreson, 2012).

7.5.4 Control/Preventative Measures

Both *P. marinus* and *P. olseni* are notifiable to the World Organisation for Animal Health (OIE). Infection with *P. marinus* is listed by the EU (Directive 2006/88/EC) and on the list of Federally Reportable Aquatic Animal Diseases – Molluscs for Canada. Infection with *P. olseni* is on the Canadian list and the US National List of Reportable Animal Diseases as well. As *Perkinsus* parasites are directly transmissible and easily spread with transfer of infected stocks, avoiding introduction to new areas with movement of infected shellfish is critical. In *P. marinus*-enzootic areas of the USA, control of *P. marinus* in aquaculture populations through selective breeding for disease-resistant oysters has been successful (Ragone Calvo *et al.,* 2003).

7.5.5 Host Species

P. marinus infects the eastern oyster *C. virginica* (Mackin *et al.*, 1950) but also oysters *C. corteziensis* (Caceres-Martinez *et al.*, 2008), *S. palmula* (Caceres-Martinez *et al.*, 2012), and *C. rhizophorae* (Da Silva *et al.*, 2013). *P. olseni* (= *P. atlanticus*) was described from the abalone *H. ruber* (Lester and Davis, 1981) but infects a wide range of bivalve and gastropod species, notably including Manila clam *R. philippinarum* in Asia (Choi and Park, 1997) and Europe (Navas *et al.* 1992). *P. chesapeaki* (= *P. andrewsi*) infects clams *Macoma balthica* (Coss *et al.*, 2001), *Mya arenaria* and *Tagelus plebeius* (Dungan *et al.*, 2002), *Cyrtopleura costata* (Reece *et al.* 2008), and *R. decussatus and R. philippinarum* (Arzul *et al.*, 2012), cockle *C. edule* (Carrasco *et al.,* 2014), and the ark *Anadara trapezia* (Dang *et al.,* 2015). *P. beihaiensis* infects oysters *C. hongkongensis* and *C. ariakensis* (Moss *et al.* 2008), *C. rhizophorae* (Sabry *et al.,* 2009), and *C. madrasensis* (Sanil *et al.,* 2012), and clam *Anomalocardia brasiliana* (Ferreira *et al.,* 2015). *P. mediterraneus* infects oyster *O. edulis* (Casas *et al.*, 2004) and ark *Arca noae* and scallop *Chlamys varia* (Ramilo *et al.,* 2015). *P. honshuensis* infects the clam *R. philippinarum* (Dungan and Reece, 2006). *P. qugwadi* infects the scallop *Patinopecten yessoensis* (Blackbourn *et al.*, 1998).

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