A QUALITATIVE ASSESSMENT OF THE RISK OF INTRODUCTION OF
VIRAL HAEMORRHAGIC SEPTICAEMIA VIRUS INTO THE RAINBOW TROUT
INDUSTRY SCOTLAND

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A QUALITATIVE ASSESSMENT OF THE RISK OF INTRODUCTION OF VIRAL HAEMORRHAGIC SEPTICAEMIA VIRUS INTO THE RAINBOW TROUT INDUSTRY SCOTLAND

Alison Gregory

Fisheries Research Services, Marine Laboratory
375 Victoria Road, Aberdeen, AB11 9DB

SUMMARY

A qualitative risk assessment has been conducted to identify the key risk pathways for the introduction of Viral Haemorrhagic Septicaemia Virus (VHSV) into the rainbow trout industry in Scotland. The risk of each pathway was estimated based on available evidence. There is a lack of data for all pathways and these data gaps present difficulties in quantifying risk. All data gaps are detailed in the assessment. The most significant pathways for Scotland were introduction via dead fish and introduction of VHSV into the marine trout industry from wild marine fish. These pathways have been identified as low risk, based on available data, which means they have a less than 50% chance but more than a rare chance of occurring over a time scale of interest. For carcass import (dead fish) processing plants in close proximity or at rainbow trout farms are of particular concern. This route could introduce genogroup 1a or the marine genogroups (1b and 3) depending on the type and quantity of fish processed. For wild marine fish, VHSV could be introduced directly to marine trout or indirectly to marine trout via cod farms. There is a potential for strains to infect marine trout asymptptomatically and evolve to a more virulent form.

INTRODUCTION

Viral haemorrhagic septicaemia (VHS), historically known as Elgved disease (Jensen 1965), was first observed in farmed freshwater rainbow trout in Germany in the 1930s (Schäperclaus 1938) and subsequently in Southern Poland, (Pliska 1946) Denmark (Schäperclaus 1954; Rasmussen 1965) and France (Besse 1955). The disease is characterised by external signs that include general darkening of the skin, exophthalmia and haemorrhaging in the oribits and fin bases (Wolf 1988). Internal signs include petechial haemorrhaging in peritoneum and muscle tissue and ascites (Wolf 1988). Mortality is higher in fry (up to 100 %) than in adults (30 – 70 %) and disease outbreaks occur when temperature fluctuates and rises (Wolf 1988). A viral aetiology was established with isolation of viral haemorrhagic septicaemia virus (VHSV) using trout cells (Jensen 1963).

VHS is a list 2 disease (EEC91/57), present in Europe but exotic to the UK. Following the outbreak in England in May 2006, there is a non-approved zone within the UK. There is a surveillance programme in Scotland to maintain this disease-free status, all farms holding susceptible species of fish are inspected at least once a year. At least once every two years samples of internal organs (and ovarian fluid in the case of broodstock) are taken from 30 fish from each farm and tested for the presence of the virus.
The application of risk analysis in aquatic animal health management is a relatively recent development (MacDiarmid 2001). Consequently, there are few published studies; those published are comprehensively reviewed by Peeler et al (2007). Risk analysis consists of four components, hazard identification, risk assessment, risk communication and risk management (OIE). The risk assessment is the scientific part of the process and itself consists of a release assessment (describing the pathways for introduction); exposure assessment (describing the pathways necessary to expose the susceptible species to the imported pathogen) and consequence assessment (identifying direct consequences such as disease outbreak and indirect consequences such as surveillance costs (OIE). The process of risk analysis can be qualitative or quantitative and is a formal, transparent process where the likelihood of an unwanted event occurring is assessed. The majority of risk assessments published on aquatic animal health to date have been qualitative (Peeler et al 2007). This is primarily because there is a lack of data to conduct quantitative assessments. However, qualitative assessments are often sufficient for decision making and are useful in identifying data gaps which should be used to prioritise future research.

In this study, a qualitative risk assessment is presented for the introduction of Viral Haemorrhagic Septicaemia Virus (VHSV) to Scotland. A risk assessment specifically examining the pathways by which VHSV was introduced to a farm in England and Wales, which experienced an outbreak in May 2006, has been published in the report on the outbreak (www.efishbusiness.co.uk/news/071219_1.pdf). Although some of the pathways for introduction of VHSV are applicable to both countries, there are significant differences in the aquaculture industry in Scotland and England/Wales, for example, there is no mariculture of rainbow trout, cod, haddock or Atlantic salmon in England and Wales. Consequently, some risk pathways have different levels of risk in the respective countries and these will be presented in this study. The terms and definitions used in this paper are based on those developed by (Kahn et al 1999) and used by Peeler and Thrush (2004) in a qualitative assessment on the risk of introduction of Gyrodactylus salaris to the United Kingdom (Table 1).

<table>
<thead>
<tr>
<th>Risk</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very high</td>
<td>Almost certain to occur</td>
</tr>
<tr>
<td>High</td>
<td>Expected to occur</td>
</tr>
<tr>
<td>Moderate</td>
<td>Less than 50:50 probability</td>
</tr>
<tr>
<td>Low</td>
<td>Unlikely to occur</td>
</tr>
<tr>
<td>Very low</td>
<td>Rarely occur</td>
</tr>
<tr>
<td>Extremely low</td>
<td>Very rarely occur</td>
</tr>
<tr>
<td>Negligible</td>
<td>Probability of occurrence so small it can be ignored</td>
</tr>
</tbody>
</table>

Table 1. Description of terms used to describe risk in this study, after Kahn et al (1999).
HAZARD IDENTIFICATION

Virus Characteristics

VHSV is a member of the family Rhabdoviridae and genus norhabdoviridae (Walker et al 2000). It has an enveloped rod-shaped virion containing a negative-stranded RNA which codes for a non-structural protein with unknown function (Nv); five structural proteins: nucleocapsid (N), phospho (P), matrix (M), glyco (G) and the RNA polymerase (L) (Shutze et al 1999). In terms of viral characterisation there are two approaches, serological and genetic. All VHSV isolates to date share antigenic properties that are recognised in IF and ELISA by polyclonal antibodies raised against DK-F1 reference strain (Olsen et al 1993). Monoclonal Antibodies have been developed that can distinguish between the American/Japanese isolates and the European isolates (Ito et al 2005). To date, there are no monoclonal antibodies that can distinguish between the European isolates. A difference between North American and European isolates was demonstrated by DNA hybridisation (Betts et al 1993) other genetic methods that have been used to distinguish isolates are a PCR method (Einer-Jensen et al 1995) and a Ribonuclease protection assay (Snow et al 1999). DNA sequencing has proven the most informative method of distinguishing between isolates. Four main genotypes have been identified based on sequence analysis of the N-gene (Snow et al 1999, 2004) and the G gene (Einer-Jensen et al 2004; 2005). Genotype 1 includes the European freshwater isolates and, to date five sublineages (a – e) have been identified. The genotype 1b has been found in wild fish in the Baltic and North Sea and was the type isolated from a VHS outbreak in marine rainbow trout in Sweden that is thought to be of marine origin (Nordbolm 2000). Genotype 1d was identified as the isolate in a VHS outbreak in marine trout in Finland (Raja-Halli et al 2006) and genotype 1e from farmed turbot in Turkey (Nishizawa et al 2006). Genotype 2 has been found mainly in the Baltic Sea (Skall et al 2005) but also an isolate from eel in an estuary in Northern France (Castric et al 1992; Thiéry et al 2002). Genotype 3 has been found in the North and Baltic seas (King et al 2001, Skall et al 2005), the Atlantic Ocean, Flemish cap (Lopez Vasquez et al 2007) exclusively in marine species. It has recently (2007) been found in cultured marine rainbow trout in Norway (www.collabcen.net/toweb/aq2.asp). Genotype 4 has been found in wild marine fish from the west coast of North America (reviewed by Meyers and Winton 1995), in Japan in wild Japanese flounder, Paralichthys olivaceus (Takano et al 2000) and cultured Japanese flounder (Isshiki et al. 2001). Recently further sublineages of genotype 4 have been found in wild freshwater fish on the great lakes and eastern regions on North America (Gange et al 2006, Elsayed et al 2006, Lumsden et al 2007, Goocock et al 2007).

Susceptible Species

In terms of susceptible species, VHSV is a significant problem for cultured rainbow trout where it can produce 100 % mortality in fry and 30 – 70 % in adults and can cause mortality in freshwater or seawater fish. It has been implicated in wild epizootics in marine fish species in North American waters (Reviewed by Meyers & Winton 1995) but not conclusively in European waters. At present, VHSV has been reported (not necessarily currently present in) 40 countries and is listed as occurring naturally in 58 species worldwide (OIE international database http://www.collabcen.net/toWeb/aq2.asp). Experimental demonstration of VHSV infection is also reported in a further 6 species (OIE international database http://www.collabcen.net/toWeb/aq2.asp).
**Survival in the environment**

There are very few published studies on survival and decay rates of VHSV in the aquatic environment. VHSV (isolate F1 from freshwater) was reported to survive for 49 days at 10°C in tap water and for 10 days when suspended in mud at 4°C (Ahne 1982a). There is unpublished data that suggests that the time required to produce a 3 log reduction in titre was several years at -20°C, several months at 4°C, approximately 4 weeks at 20°C and 1 min at 70°C (a personal communication by N.J. Olsen published in EFSA 2005 [http://www.efsa.europa.eu/EFSA/Scientific_Opinion/ahaw_op_ej584_fishdiseasevectors_en.pdf](http://www.efsa.europa.eu/EFSA/Scientific_Opinion/ahaw_op_ej584_fishdiseasevectors_en.pdf)). It has been reported that both a North American marine isolate and a European freshwater isolate (F1) are more stable in seawater than freshwater and that the North American isolate is more stable than the freshwater isolate. These results were based on titre reduction over a one hour period at 12°C. (Winton et al (1991) conference proceedings cited in Meyers & Winton (1995). A study investigating the stability of nine VHSV isolates reported that survival of all isolates tested was longer at lower temperatures (Parry and Dixon 1997). There was variability in the survival time of isolates, for example one freshwater isolate survived for the greatest period of time, 28 -35 days at 4°C whereas another freshwater isolate survived for the least time, 3 – 7 days at 4°C (Parry & Dixon 1997). The stability of marine isolates from North America and Europe were similar to each other (Parry & Dixon 1997). More recently, Kocan et al (2001) reported the North American strain of viral hemorrhagic septicaemia virus (NA-VHSV) could be recovered for up to 40 h in natural filtered seawater (27 ppt) with a 50 % loss of infectivity after approximately 10 h at 15 °C. Ovarian fluid in seawater significantly prolonged virus survival; virus could be recovered after 72 h at 0.01% ovarian fluid and after 96 h at 1.0% (Kocan et al 2001).

**Viral Shedding**

Viral shedding via urine has been demonstrated from rainbow trout following immersion challenge (Neukirch & Glass 1984). VHSV could be detected in urine 3 days post immersion challenge at a titre of 10^2CCID_{50}ml^{-1} (Neukirch & Glass 1984). Relatively high yields of up to 10^5CCID_{50}ml^{-1}, of infectious VHSV could be demonstrated during acute VHS one day before death, but not by faeces (Neukirch & Glass 1984). It is also important to note that survivors of the challenge may excrete infectious virus for more than 30 days in the absence of clinical signs following a secondary challenge, demonstrating a carrier state in rainbow trout (Neukirch & Glass 1984). Virus shedding has not been observed in faeces (Neukirch & Glass 1984). Viral shedding has also been experimentally demonstrated by the onset of VHS in rainbow trout kept in outflow water from aquaria containing infected salmon fry (De Kinkelin & Castric 1982).

Virus shedding has also been demonstrated experimentally following immersion challenge of Pacific herring with a North American VHS isolate Kocan et al (1997). In this study a maximum shedding rate of more than 10^{6.5} PFU/h/fish was reported, at a time close to the first death (within one day) (Kocan et al 1997).

Virus shedding has also been reported from ovarian fluids (Wolf 1988). Two isolations were obtained from ovarian fluids of adult returning coho salmon in Washington state, 1 pool of 14 (90 fish tested) and 1 pool of five fish (Eaton & Hulett 1990). Also an isolation was obtained from 1 pool of 5 of Milt (Eaton & Hulett 1990). In 1991, a further isolation from a 5 fish pool (31 fish tested) here titre data indicated 5 x 10^4 pfu on EPC cells (unpublished data by Stewart in Meyers & Winton 1995).
TRANSMISSION ROUTES

Horizontal Transmission

Virus transmission has been demonstrated experimentally to occur by contact with other infected fish or contaminated freshwater or utensils at temperatures ranging from 1 – 12°C up to 15°C (Vestergaard Jorgensen 1973). VHSV can also be transmitted in seawater (Castric & DeKinkelin 1980) and there is a report that the virus can be transmitted up to 2km in seawater (pers comm by Vestergaard Jorgensen cited in Meyers & Winton 1995). A number of experimental studies have demonstrated horizontal transmission in rainbow trout and other species by immersion, injection or cohabitation (Table 2). An oral route of transmission has been demonstrated experimentally in pike (Ahne 1980; Ahne 1985) however, it has not been established in other species. A faecal oral route is unlikely given the lack of evidence of VHSV in faeces (Neukirch & Glass 1984). Wild birds have the potential to transmit VHSV. A variety of species of wild birds will scavenge salmonid carcasses: Heron Ardea cinerea, great black-backed gull Larus marinus and crow Corvus corone have been demonstrated to scavenge wild salmon carcasses post spawning along the Dee (Hewson 1995). Heron are known to prey on farmed trout and have been observed to take, a high proportion of the fish attacked by herons were blind and/or in poor condition (Carss 1993). Heron have been shown to feed on brown trout in Loch Leven, Scotland (Marquiss & Leitch 1990). VHSV has been re-isolated from samples of food regurgitated by herons up to 120 minutes after feeding contaminated fish. Therefore herons are able to act as mechanical vectors for VHSV (Peters & Neukirch 1986).
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<table>
<thead>
<tr>
<th>Host Species (challenged fish)</th>
<th>Fish size and Marine or FW</th>
<th>VHSV isolate (genogroup)</th>
<th>Challenge method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atlantic cod</td>
<td>14g SW</td>
<td>VI6775/3 (3)</td>
<td>10^5 TCID&lt;sub&gt;50&lt;/sub&gt; /ml</td>
<td>Snow et al 2000</td>
</tr>
<tr>
<td>Atlantic cod</td>
<td>12g SW</td>
<td>2 x (1a), 5 x (1b), and 1 x (4)</td>
<td>10^6 TCID&lt;sub&gt;50&lt;/sub&gt;/ml</td>
<td>Snow et al 2005</td>
</tr>
<tr>
<td>Atlantic halibut</td>
<td>11g SW</td>
<td>2 x (1a), 5 x (1b), and 1 x (4)</td>
<td>10^6 TCID&lt;sub&gt;50&lt;/sub&gt;/ml</td>
<td>Snow et al 2005</td>
</tr>
<tr>
<td>Atlantic salmon</td>
<td>6g FW</td>
<td>11 groups of marine isolates</td>
<td>x</td>
<td>King et al 2001</td>
</tr>
<tr>
<td>Pacific Herring</td>
<td>2 - 5g</td>
<td>Wild Pacific hering 1993 (4)</td>
<td>low 10&lt;sup&gt;5&lt;/sup&gt;</td>
<td>Kocan et al 2001</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>25-30cm FW</td>
<td>F1 (1)</td>
<td>10&lt;sup&gt;6&lt;/sup&gt; (F+H)</td>
<td>Neukirch &amp; Glass 1984</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>160g FW</td>
<td>07.71INRA (1a)</td>
<td>ip 10^6 PFU/ml</td>
<td>Castric &amp; DeKinkelin 1984</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>160g SW</td>
<td>07.71INRA (1a)</td>
<td>ip 10&lt;sup&gt;4&lt;/sup&gt; PFU/ml</td>
<td>Castric &amp; DeKinkelin 1984</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>15-20g FW</td>
<td>F1 (1)</td>
<td>x</td>
<td>Neukirch 1984</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>250-28bg FW</td>
<td>F1 (1)</td>
<td>x</td>
<td>Neukirch 1984</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>2-4g FW</td>
<td>866/94 turbot (3)</td>
<td>ip 10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Snow &amp; Cunningham 2000</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>2-6g FW</td>
<td>866/94 turbot (3)</td>
<td>ip 10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Skall et al 2004</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>3 months FW</td>
<td>115 isolates pooled</td>
<td>10&lt;sup&gt;6&lt;/sup&gt; CCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Castric et al 1992</td>
</tr>
<tr>
<td>Sea Bass</td>
<td>2g SW</td>
<td>VHSV 07/71 (1a)</td>
<td>4 x 10&lt;sup&gt;5&lt;/sup&gt; pfuml&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>Castric &amp; DeKinkelin 1984</td>
</tr>
<tr>
<td>Sea Bass</td>
<td>12g SW</td>
<td>237/75 (1a)</td>
<td>2 x 10&lt;sup&gt;5&lt;/sup&gt; pfuml&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>Castric &amp; DeKinkelin 1984</td>
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<tr>
<td>Turbot</td>
<td>8g SW</td>
<td>866/94 turbot (3)</td>
<td>4 x 10&lt;sup&gt;5&lt;/sup&gt; pfuml&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>Castric &amp; DeKinkelin 1984</td>
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<tr>
<td>Turbot</td>
<td>4g SW</td>
<td>11 groups of marine isolates</td>
<td>4 x 10&lt;sup&gt;5&lt;/sup&gt; pfuml&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>Castric &amp; DeKinkelin 1984</td>
</tr>
<tr>
<td>Turbot</td>
<td>6.5g SW</td>
<td>VHSV 07/71 (1a)</td>
<td>4 x 10&lt;sup&gt;5&lt;/sup&gt; pfuml&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>Castric &amp; DeKinkelin 1984</td>
</tr>
<tr>
<td>Turbot</td>
<td>11 - 50g mean = 28g SW</td>
<td>VHSV 07/71 (1a)</td>
<td>6 x 10&lt;sup&gt;5&lt;/sup&gt; pfuml&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>Castric &amp; DeKinkelin 1984</td>
</tr>
<tr>
<td>Turbot</td>
<td>25g</td>
<td>GH40 (halibut)</td>
<td>10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Lopez Vazquez et al 2007</td>
</tr>
<tr>
<td>Turbot</td>
<td>25g</td>
<td>UK860/94 (farmed turbot Scotland)</td>
<td>10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Lopez Vazquez et al 2007</td>
</tr>
<tr>
<td>Turbot</td>
<td>25g</td>
<td>SM2897 farmed Turbot spain</td>
<td>10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Lopez Vazquez et al 2007</td>
</tr>
<tr>
<td>Whitefish</td>
<td>1.5g FW</td>
<td>1p40 (marine 1b)</td>
<td>10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Skall et al 2004</td>
</tr>
<tr>
<td>Whitefish</td>
<td>1.5g FW</td>
<td>DH392 (FW1a)</td>
<td>10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Skall et al 2004</td>
</tr>
<tr>
<td>Whitefish</td>
<td>1.5g FW</td>
<td>SVA14 (5b marine trout)</td>
<td>10&lt;sup&gt;5&lt;/sup&gt; TCID&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Skall et al 2004</td>
</tr>
</tbody>
</table>
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Vertical Transmission

There has only been one study on vertical (true intra-ovum transmission of VHSV) to date which involved an immersion challenge of fertilised eggs from a VHS positive farm with no clinical symptoms at the time of spawning. It concluded vertical transmission was unlikely to occur because VHSV does not apparently survive the egg incubation period, since no virus was isolated from fry of either experimentally infected eggs (immersion challenge) or from eggs of latently infected rainbow trout (Vestergaard Jørgensen 1970). It is not clear whether true intra ovum transmission occurs however, the virus causing viral hemorrhagic septicaemia (VHSV) was found in ovarian fluid and milt pools from wild coho salmon obtained from the Soleduck and Bogachiel rivers and held at the Soleduck Hatchery (Washington state, USA) (Eaton & Hulett 1990). The virus was identified as VHSV by neutralization and immunoblot assays. (Eaton & Hulett 1990).

RELEASE PATHWAYS (introduction into Scotland)

Live Fish Import

The import of live fish has been implicated in a number of disease outbreaks, such as infectious salmon anaemia (ISA) by well boats (Murray et al 2002), and Gyrodactylus salaris into Norway (Mo 1994). The likelihood of VHSV being imported with live fish will depend upon a number of factors. In Scotland, there have been no live imports of rainbow trout from out with the UK in recent years (FRS Fish health inspectors (FHI) import data). Live fish are imported into the UK (approximately 200 tonnes in 2006 –Her Majesty’s Revenue and Customs (HMRC)) however; these are predominantly from Asia for the UK pet trade. It is important to consider in the import of live fish that VHSV can occur in a latent carrier state, without clinical signs in affected fish (Neukirch 1986). Current legislation would prevent the import of live fish from non-approved zones in the EU. However, the carrier state of VHSV poses a risk that VHSV could remain undetected in the exporting country or region prior to export. Current legislation for import states that imports of species susceptible to the notifiable diseases must come from a farm or zone recognised as free from that disease and the diseases must also be notifiable in the country of origin. All imports must be accompanied by a health certificate. EU imports can enter via any airport or port with no checks, but imports from third countries must enter via a border inspection post and won't be released by the state vets unless there is a health certificate (Warwick pers com). It should be noted that as the official service in the importing country can not confirm the nature and extent of testing of the exporting country, only that a health certificate is attached to the import. It does not appear that VHSV has been imported via this route to date but any change in legislation and import regulations could increase the risk.

It is very important to consider the risk of VHSV entering England and Wales and the potential for spread to Scotland from these countries. The legislation that controls fish imports is consistently applied across the UK because it originates at EU level. The risk of importation of VHSV into England and Wales was evaluated following the outbreak in England in May 2006. It was considered an unlikely route of introduction (www.efishbusiness.co.uk/news/071219_1.pdf).

There is potential for other freshwater species to carry VHSV, since the virus has been reported in at least 23 fish species that spend all or part of their life cycles in freshwater (OIE database...
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http://www.collabcen.net/toWeb/ag2.asp). Again, at present this route is extremely low because no live fish imports have occurred.

Important factors that affect the risk of introduction via live fish imports are the size of trade, legislation, mechanism of import and prevalence in other countries. Given the lack of imports of live fish into Scotland to date the likelihood of introduction of VHSV via this route is extremely low. This likelihood would alter significantly if current importation practices change in the future.

**Dead Fish Import**

The release assessment for introduction of VHSV with dead fish is shown in Figure 1. An outbreak of VHSV via this route of import firstly requires infection in the imported fish and that the virus survives transport, the evidence suggests that it is possible that VHSV could be imported in this way. It was considered one of the most likely routes of introduction of VHSV in the outbreak in England in May 2006 ([www.efishbusiness.co.uk/news/071219_1.pdf](http://www.efishbusiness.co.uk/news/071219_1.pdf)).

**VHSV infection**

The susceptible species have been detailed in this assessment. The important data in terms of importation of VHSV with carcasses is the quantity and distribution of virus in infected fish and the prevalence of infection in stocks to be imported. Although evisceration decreases the risk of pathogen transfer, studies on the pathogenesis of VHSV revealed the virus in brain, diencéphalon and optic lobe (Neukirch 1986). Some fish with virus in the brain also exhibited nervous symptoms. Neukirch (1986) made an isolation of VHSV was obtained from brain of an apparently healthy animal 400 days after experimental infection without detecting the virus in blood or other internal organs (Neukirch 1986) and has demonstrated the multiplication of VHSV in brain of experimentally infected (Neukirch 1986). VHSV was isolated from brain of fish from 8/39 fish infected via immersion between 35 and 379 days post infection. High titres of VHSV have been reported in the brain following experimental immersion challenge. Titre in the brain 13 days after water borne infection with an initial dose of $10^5$ CCID$_{50}$/ml reached $10^4$ CCID/ml (Neukirch 1986 Fish and shellfish pathology). The findings of Neukirch (1986) are significant however they represent results from a few fish.

For risk analysis, more data is required on the proportion of a population infected with VHSV and where and in what quantities VHSV is within tissues, particularly from asymptomatic carrier fish (Hervé-Claude et al. 2008).
Figure 1. Release assessment for the introduction of VHSV via fish carcasses.

Survival data

There is no data specifically relating to survival of VHSV in fresh fish carcasses. However, conditions for transporting carcasses are likely to be conducive for VHSV survival, since conditions are cold. VHSV has been demonstrated to survive longer (up to 35 days) at cold temperatures (4 °C) in water (Parry & Dixon 1987). Factors that affect whether VHSV will survive transport in fresh tissues may include, virus strain, location in carcass, temperature, water type and transport time.

In terms of frozen tissue, there is unpublished data that suggests that the time required to produce a 3 log reduction in titre was several years at -20°C, several months at 4°C, approximately 4 weeks at 20°C and 1 min at 70°C (a personal communication by N.J. Olsen published in EFSA 2005 http://www.efsa.europa.eu/EFSA/Scientific_Opinion/ahaw_op_ej584_fishdiseasevectors_en.pdf). Although commercial freezing can be expected to reduce VHS titre (Arkush et al 2006) high concentrations (10⁷ pfu ml⁻¹) of VHSV genotype 4 have been recovered from thawed fish from wild and experimentally infected marine fish from the west coast of North America (Meyers and Winton 1995; Kocan et al 1997).

Import data

There is a significant import of dead fish to the UK annually from many regions of the world (HMRC). In 2006 approximately 400 000 tonnes of dead fish were imported into the UK with a value of around £ 1 billion pounds (HMRC). The fish carcass trade represents 99.9 % of the import of live and dead fish. All the groups (defined by SITC - standard international trade code) of fish that are imported as carcasses into the UK contain species that are susceptible to VHSV and therefore pose a risk in terms of introduction (Table 3). In terms of salmonids, over 28 000 tonnes of chilled and a further 2800 tonnes of frozen fish were imported into the UK at a total value of around £92 million pounds in 2006 (Table 3 HMRC). The fresh salmonid carcasses (whole eviscerated) are mainly imported from European countries with a small proportion, 0.06 % from North America. Import regulation for fish carcasses is different to that of live fish and
imports could occur from regions or countries that have VHS. Frozen salmonid carcasses (whole eviscerated) are imported from both Europe (1359 tonnes) and North America (1409 tonnes) with small quantities also imported from other American countries (18.4 tonnes) and Asia (39.4 tonnes) (Table 3 HMRC). The import of carcasses from North America (USA and Canada) is of concern because it offers a mechanism for the introduction of genotype 4 VHSV, currently exotic to the European Union, into Scotland. VHSV can survive freezing albeit at reduced titres (Arkush et al 2006) and could be imported; however, exposure would depend on which species of salmonids were imported and where and how they are processed on arrival in the UK.

The import of aquatic animals to Scotland represented 9.5% of the total UK imports in the SITC commodity code 03 (fish, crustaceans, molluscs and aquatic invertebrates and preparations thereof). It is not possible to resolve the data further at present (as it is for the whole UK) for Scotland because it is not available at the regional level (confidential). However, the majority of imports of fish and shellfish in Scotland will be fish carcasses rather than live fish, based on the trend for the whole of the UK. The details of SITC 03 imports into Scotland are shown in Table 4.

Significant factors that affect introduction via fish carcasses are VHS status of exported stock, distribution and quantity of VHSV within infected fish and VHSV survival during transport. This pathway is considered low risk.
A Qualitative Assessment Of The Risk Of Introduction Of Viral Haemorrhagic Septicaemia Virus

<table>
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<tr>
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<th>West Europe (excluding EU)</th>
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<th>North America</th>
<th>Other America</th>
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**Table 3.** UK fish carcass import data (Source: HMRC [www.uktrade.com](http://www.uktrade.com))
Table 4. SITC 03 (fish, crustaceans, molluscs and aquatic invertebrates and preparations thereof) imports to Scotland. These include live and dead fish imports.

### Egg Import

The risk pathway for introduction of VHSV via the importation of eggs is shown in Figure 2. The first two steps of the pathway, infection of broodstock and infection of eggs have a number of stages that have previously been suggested in a general model for the vertical transmission of fish pathogen (Peeler 2005).

**Figure 2.** Risk pathway for introduction of VHSV via egg importation. Blue lines indicate release assessment and red lines exposure assessment.
There is a large import of Ova of both rainbow trout and Atlantic salmon into Scotland. Import data from 2007 indicates approximately 16 million rainbow trout eggs were imported. The majority (approximately 8 million) were imported from Denmark (Fig 3). Current legislation for import states that imports of species susceptible to the notifiable diseases must come from a farm or zone recognised as free from that disease and the diseases must also be notifiable in the country of origin. All imports must be accompanied by a health certificate. EU imports can enter via any airport or port with no checks, but imports from third countries must enter via a border inspection post and won't be released by the state vets unless there is a health certificate (Warwick pers com). It should be noted that as the official service in the importing country can not confirm the nature and extent of testing of the importing country, only that a health certificate is attached to the import. It does not appear that VHSV has been imported via this route to date but any change in legislation and import regulations could increase the risk.

![Graph showing egg imports to Scotland for 2006](source: FHI egg import records)

**Figure 3.** Annual egg imports to Scotland for 2006 (Source: FHI egg import records)

VHSV can be transmitted in association with eggs (extra-ovum) and has been isolated from ovarian fluids and milt (Wolf 1988; Eaton et al 1991). Virus transmission via eyed eggs has been recorded (Ghittino 1965). However, the virus is inactivated by disinfection of ova at importing farm. A number of disinfectants are effective against VHSV including chlorine, formalin and sodium hydroxide (Ahne 1982) and iodophors (Amend & Pietsch 1972; Ahne & Helde 1980). It as not been fully established if VHSV could be imported via true (intra-ovum) transmission of VHSV, however the only study on vertical transmission of VHSV reported it unlikely to occur (Vestergaard Jorgensen 1970).
For egg import, there are data gaps in the sensitivity, specificity and scale of testing on eggs that are imported. It should also be established using modern diagnostic techniques if VHSV can be internalised within eggs and can survive through to hatching. An outbreak of VHSV has not occurred via this mechanism in the UK and risk is considered very low.

**Introduction from the Marine Environment**

In Scotland, there are 12 registered marine trout farms (Fig 4). The scale of this industry is very small compared to the freshwater farms of which there are currently 65 registered sites (Aquadat). The other marine species commercially farmed in Scotland are Halibut and Cod. There has been growth, particularly in cod production, in recent years (Fig. 5). The locations of all marine trout and other marine species farms in Scotland is shown in Figure 4. All cod farms are in the Northern isles. The majority of the marine trout farms are located on the west coast with a small number in the Northern Isles (Fig 4).
Figure 4. Locations of Marine trout (red triangles) and cod farms (blue triangles) in Scotland.

VHSV (genotypes 1b and 3) are endemic in marine waters around the UK at very low prevalence (King et al 2001, Skall et al 2005). The routes by which VHSV from wild marine fish could enter the rainbow trout industry in Scotland are summarised in Figure 6 and evidence for each route is outlined in the individual sections below.
A Qualitative Assessment Of The Risk Of Introduction Of Viral Haemorrhagic Septicaemia Virus

**Figure 6.** Summary of risk pathways for introduction of VHSV from the marine environment. Pathways with blue arrows are extremely low risk. Pathways in red are low risk and detailed pathways for these routes are shown in Figure 7.

**Raw fish as a feed**

The use of raw or minced fish to feed freshwater rainbow trout was largely practised as the industry developed in the 1940s and 1950s. It is thought this was the mechanism by which VHSV emerged in the freshwater industry as far back as the 1950s when it was suggested intensive feeding of fresh herring produced clinical signs of the disease (Schäperclaus 1954; reviewed by Dixon 1999). This theory is supported by genetic evidence which suggested the emergence of genogroup 1a occurred around 50 years ago (Einer Jensen et al 2004). The use of raw fish or treated fresh fish was identified as a potential (although not confirmed) source of VHSV the Finnish outbreak in marine trout (Raja- Halli et al 2006) and the introduction of a genogroup 3 isolate to a turbot farm in Gigha Scotland (Munro 1996). It was also identified as exposing wild fish from risk analysis from Chilean salmon farms (Hervé-Claude et al. 2008). This evidence also highlights the importance of oral transmission in VHSV. Consequently, the practice of feeding farmed fish on raw fish has ceased throughout Europe because of the high risk of introduction. This pathway can be considered extremely low, due to lack of feeding on raw marine fish in the industry today.

**Anadromous fish**

The introduction of VHSV via anadromous fish is a potential risk. VHSV has been isolated from wild elvers in coastal rivers in Northern France (Castric et al 1992). This isolate has been identified as genogroup 2 (Thiery et al 2002) and has been demonstrated to induce mortality by intraperitoneal injection (Castric et al 1992). In Denmark, whitefish, *Coregonus lavaretus*, are anadromous and a total of 148 fish tested from rivers containing infected VHS farm in 2000, were negative for VHSV (Skall et al 2004a). In North America there have been isolations from wild salmonids returning to rivers to spawn (Meyers and Winters 1995). However, there have been no isolations from wild sea trout or wild Atlantic salmon. Also marine isolates have been demonstrated experimentally to have low or no virulence in freshwater rainbow trout (Skall et al
Given this evidence the risk of direct transfer from anadromous fish to freshwater rainbow trout is considered extremely low risk.

**Atlantic salmon farms**

There is potential risk of introduction to either marine or freshwater trout farms via Atlantic salmon farms. VHSV has been isolated from one freshwater farmed Atlantic salmon fry in Spain (Jimenez de la Fuente et al. 1988) and there is anecdotal evidence of isolation from Atlantic salmon farmed on the west coast of North America (Traxler et al. 1995). In Scotland Atlantic salmon are regularly tested (30 fish sample every two years) for VHS as part of EU List 2 surveillance. VHSV has not been isolated from Atlantic salmon in Scotland. This pathway is therefore considered extremely low risk.

**Wild Marine fish**

There is a risk of introduction to marine trout farms from wild marine fish. Outbreaks of VHSV in marine trout farms have been reported in Denmark (Horlyck et al. 1984; Jorgensen 1992), France (Castric & De Kinklin 1980), Finland (Raja-Halli et al. 2006), Sweden (Nordbom 2000) and Norway (2007). In some cases it was thought the disease was introduced by movement of fish from freshwater sites (Castric & De Kinklin 1980; Horlyck et al. 1984; Jorgensen 1992) whereas other cases suspect a marine origin (Nordbom 2000; Raja-Halli et al. 2006) however, Dixon in a review of the origins of VHSV reported that a direct marine route has not been ruled out in most outbreaks in marine trout (1999). The potential for an outbreak via freshwater trout is not considered here because it would have to have been imported into Scotland’s freshwater industry and then spread to marine fish; these risks have been fully considered elsewhere in this assessment (release pathways for live or dead fish and eggs).

The introduction of VHSV from wild marine fish to marine trout from the marine environment could occur via a number of risk pathways (Figure 7). VHSV could be transmitted from infected wild fish to farmed marine rainbow trout through waterborne or oral routes. It could also be transmitted indirectly by transfer from wild infected fish to marine rainbow trout via farmed marine species such as haddock or cod. In this case transmission from cod farms to marine trout farms would have to occur by waterborne transmission, due to the physical separation of cages, unless cod escaped and came into direct contact with the farmed marine trout. In 2006 there was one reported escape from ‘other species’ (i.e. Not salmon or trout) of 12 230 fish (source: FRS annual production survey). The pathways in Figure 7 provide a model in which different scenarios can be tested. The evidence available for the key stages in the pathway is outlined below.
Figure 7. Risk Pathways for introduction of VHSV to marine rainbow trout via wild marine fish.
VHSV has been isolated from 6 of 23 species of marine fish in the north east Atlantic, North Sea and Irish Sea with prevalence ranging from 0.03 – 0.7 % (King et al 2001). Of those species from which VHSV was isolated, the lowest prevalence was in herring and whiting and the highest prevalence was in Norway pout. The genogroups 1b and 3 have both been isolated from wild marine fish around Scotland (Einer Jensen et al 2004; snow et al 2004). A similar survey of marine fish in the Baltic Sea area isolated VHSV from 8 of 40 species with prevalence ranging from 0.2 – 16.7 % in these species (Skall et al 2005). It is interesting to note that the highest prevalence in both studies occurred in shoaling planktivorous fish, Norway pout (King et al 2001) and herring (Skall et al 2005). These studies suggest VHSV is endemic in some marine fish and that prevalence is generally low. It is important to consider that factors that may change the prevalence in wild fish could have an impact on the likelihood of VHSV being introduced in farmed marine trout. It has been reported that the epizootiology of North American VHSV in Pacific herring is that of an opportunistic pathogen that is triggered by stress (Meyers et al 1994). Stressors are thought to include spawning, harassment by predators, colder than normal winter seawater temperatures, strong year classes of fish, infection with other diseases (erythrocytic necrosis virus, ENV, in the case of Pacific herring), exposure to pollutants and nutritional deprivation (Meyers et al 1994). A more recent study used a modification of an age-structured assessment model, using a disease index that combines the prevalence of viral hemorrhagic septicemia virus (VHSV) with the prevalence of ulcers, to identify risk factors for an epidemic (Marty et al 2006). These included poor body condition and abundant recruitment before spawning in the spring (Marty et al 2006).

Outbreaks have occurred in farmed marine rainbow trout that may have had a marine origin (Nordbolm 2000) so it is likely there are isolates in the marine environment that are virulent for marine rainbow trout. There is no experimental data on the susceptibility of marine rainbow trout isolates to marine isolates of VHSV. There is some data on the virulence of marine isolates on rainbow trout fry. The isolate 96-43 from Atlantic herring in the English Channel did not induce mortalities in rainbow trout fry via immersion challenge at $10^4$ pgfuml$^{-1}$ (Dixon et al 1997). Skall et al 2004b did immersion challenges using pools of marine isolates from a number of species and genogroups. Generally the marine isolates (of genogroups 1b and 3) exhibited low virulence for rainbow trout fry by immersion. There was a group of sprat isolates in which there was a statistical difference in cumulative mortality. For all groups of isolates tested mortality ranged from 0 – 14 %. Isolations were made from at least one sample per group except for Scottish Norway Pout. The highest cumulative mortality was 14 %, from a group of genogroup 1b sprat isolates. The Swedish 1b isolates from maricultured rainbow trout produced about 20% mortality in rainbow trout fry (Skall, un unpublished data in Skall et al 2004).

There is some experimental evidence on the virulence of marine isolates for marine species of fish (Table 2). Pathogenicity of a number of 1b isolates from herring and sprat have been shown to have variable mortality for turbot via immersion challenge. Mortality with herring isolates ranged from 16.5 % to 39 % and mortality from sprat isolates ranged from 0 to 13.5 % (King et al 2001). Although VHSV of genogroup 1b has been isolated from cod, they have very low susceptibility by immersion challenge to the isolate tested (originally from marine cod) (Snow et al 2000). An immersion challenge using a 1b sprat isolate form the Baltic did result in one isolate from 90 fish being obtained (Snow et al 2005).

In the wild, all the species from which VHSV has been isolated from in UK waters are linked biologically in terms of a food web (Fig 8). Biological and behavioural data highlight the potential for transmission through the food chain. For example, during the spawning of herring, cod are often caught with stomachs full of herring roe (Muus & Nielsen 1999). Cod eat far less in the winter months and during their own spawning period. In March they show strong diet
shifts and abandoned their usual prey in favour of herring or herring eggs. (Hoeines & Bergstad 1994). Migratory Atlantic cod follow shoals of herring and capelin (Muus & Nielsen 1999). This evidence and that from experimental studies which demonstrate no virus associated mortality of cod following immersion challenge with a cod isolate (Snow et al 2000) strongly suggest an oral route of infection for cod.

Figure 8. Food web showing relationships between fish species from which VHSV has been isolated from in the wild.

In waters around the UK, it is interesting to observe that both VHSV isolates from Atlantic herring have been isolated in proximity to spawning areas at or around the time of spawning. The isolate MLA98/HE1 from Shetland (ICES 50E9) is relatively close to the Shetland spawning ground (49E9) and was obtained in August. The Shetland spawning ground is an autumn spawning stock (Hatfield & Simmonds 2002). The isolate 96-43 from the English Channel was obtained in rye bay in January/February (Dixon et al 1997). This is close to the Downs herring spawning ground which is a winter spawning stock (Hatfield & Simmonds 2002). The Swedish rainbow trout isolations fall in ICES 45G1 in which there are herring spawning grounds (spring spawners). Herring spawning occurs throughout the Gulf of Finland from April onwards, all along the coast. Again, the timing fits with the first Finnish outbreaks on rainbow trout farms. The location of herring spawning grounds and marine farms is shown in Figure 9. Should it be confirmed that spawning herring pose risk in transmitting VHS, it is important to determine whether any marine farms are in proximity to spawning grounds. Those farms situated in the Northern Isles are particularly vulnerable to this pathway (Fig 9).
Figure 9. Location of herring spawning grounds (Hatfield & Simmonds 2002) in relation to marine tour and cod farms in Scotland.
For introduction via marine fish, there are data gaps in a number of key areas. There is no data available on the susceptibility of marine trout to marine isolates of VHSV. There is a need to better understand mechanisms of uptake, particularly in cod. Also shedding rates and minimum infectious dose data is very limited for VHSV, especially in marine fish, including marine trout. There is no understanding of the dynamics of VHSV infection in wild fish. These data are essential to quantify the risk of introduction to marine trout via wild marine fish. Based on the qualitative evidence available the route is considered low risk.

**EXPOSURE PATHWAYS (contact with susceptible species)**

**Live Fish Import**

The risk of exposure to VHSV via the importation of live fish is very high. This is because if infected fish were imported it is very likely they would come into direct contact with susceptible species because they would enter trout farms. Infection could be established via direct contact with fish stocks or waterborne transmission. Measures to reduce the exposure risk, if live fish are imported, would involve quarantine of new stocks.

**Dead Fish Import**

The exposure assessment indicates how the virus could come into contact with susceptible species through fish processing. Processing plants in Scotland receive both fish from UK farms (whole fish) and eviscerated carcasses from all over the world. A schematic diagram showing the operation of a typical processing plant is shown in Figure 10. Exposure could occur through three main routes, the first is mechanical transmission via transport of fish and movement of vehicles between farms, processors and haulage depots (Fig 11). The second is through the production of liquid waste from the processing of carcasses (Fig 12) and the third is via the production of solid waste (Fig 13). Each of the risk pathways for transport, liquid waste and solid waste present a mechanism for the exposure of susceptible species to VHSV and may be considered separately. Important factors that will affect the risk include the source of fish to be processed, the quantity of fish processed, the location of processing plant and distances from trout farms with which they have contact. The processing of marine fish carcasses at freshwater processing plants, especially those attached to freshwater trout farms poses a risk for the introduction of marine strains directly into the freshwater environment. The processing of marine carcasses and freshwater carcasses in proximity to marine trout farms poses a direct risk to marine trout farms. The locations of processing plants in Scotland in relation to the locations of marine and freshwater trout farms is shown in Figure 14. There are clearly a number of marine trout sites in close proximity to processing plants, it is essential to obtain data on the quantities and origins of fish processed at these plants to quantify the risk posed by exposure to dead fish. The risk of exposure will be high for some plants and low for others, depending on proximity to farms.
Figure 10. Schematic diagram of a typical processing plant.
**Figure 11.** Exposure pathway for transport following processing of fish carcasses.

**Figure 12.** Exposure pathway for liquid waste following processing of fish carcasses.
Figure 13. Exposure pathway for solid waste following the processing of fish carcasses.
Figure 14. Location of processing plants (blue circles) in relation to freshwater trout (red triangles) and marine trout (white triangles) farms.
Egg Import

If VHSV was imported with eggs the risk of exposure would depend on whether the virus was internally or externally transmitted. The introduction of VHS externally would occur through a failure in disinfection (see release assessment) and the exposure risk would be very high because virus could be released directly into a hatchery water supply. If VHSV was internally transmitted there would be a moderate risk of exposure because virus would be within newly hatched fry and it is more likely VHS infection would be observed in fry given their high susceptibility to VHSV (Wolf 1988).

Introduction from the Marine Environment

Raw fish food

The cessation of use of raw fish as a food source in aquaculture in the UK creates a negligible risk of exposure via this route. This risk would increase substantially if practices changed, for example, if the culture of new aquaculture species required the use of live or raw fish feed.

Anadromous fish

In Scotland, many rivers have large populations of anadromous fish. Therefore in terms of exposure, if anadromous fish had VHSV the exposure risk is high because of the possibility of direct contact with freshwater or marine rainbow trout sites.

Atlantic salmon farms

In some regions of Scotland there is proximity between Atlantic salmon farms and marine and freshwater trout farms. There is a moderate risk of exposure from Atlantic salmon farms. Although this assessment is largely based on the introduction of VHSV to Atlantic salmon from wild fish in European waters, it is important to consider that Atlantic salmon may introduce the exotic genogroup 4 from North America. There is no available data as yet on the susceptibility of the recent (Gange and Elsayed) freshwater genogroup 4 isolates from eastern North America for either rainbow trout or Atlantic salmon.

Wild marine fish

If VHSV was introduced to a marine trout farm the exposure to the rainbow trout industry could be very high. It is possible that marine isolates infecting marine rainbow trout farms may not induce high or significant levels of mortality and therefore have the potential to remain undetected. In this scenario, there is potential for VHSV to adapt into a more virulent strain in marine trout. RNA viruses have a relatively high mutation rate, for VHSV substitution rates have been calculated as $7.06 \times 10^{-4}$ site$^{-1}$ year$^{-1}$ for seawater isolates and $1.74 \times 10^{-3}$ site$^{-1}$ year$^{-1}$ for freshwater isolates (Einer Jensen et al 2004) and $1.3 \times 10^{-4}$ to $2.6 \times 10^{-4}$ site$^{-1}$ year$^{-1}$ for isolates from marine rainbow trout outbreaks in Finland (Raja-Halli et al 2006). Snow and Cunningham (2000) reported an increase in virulence of a turbot isolate (860/94), following passage through rainbow trout. In this study, there was no difference in the G gene sequence between the original isolate and that from which increased virulence was observed (Snow & Cunningham...
2000). It is vital to obtain data on virulence markers to indentify marine isolates that pose a risk to the rainbow trout industry. The risk of exposure is high.

**RISK ESTIMATION**

A qualitative summary of risk estimation based on available evidence to date is shown in Table 5.

<table>
<thead>
<tr>
<th>Risk Pathway</th>
<th>Release Assessment</th>
<th>Exposure Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Live fish import</td>
<td>Extremely low</td>
<td>very high</td>
</tr>
<tr>
<td>Dead fish import</td>
<td>low</td>
<td>low to high</td>
</tr>
<tr>
<td>Egg import</td>
<td>very low</td>
<td>moderate to high</td>
</tr>
<tr>
<td>Marine:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raw fish feed</td>
<td>extremely low</td>
<td>very high</td>
</tr>
<tr>
<td>Anadromous fish</td>
<td>extremely low</td>
<td>high</td>
</tr>
<tr>
<td>Atlantic salmon fish</td>
<td>extremely low</td>
<td>moderate</td>
</tr>
<tr>
<td>Wild marine fish</td>
<td>low</td>
<td>high</td>
</tr>
</tbody>
</table>

Table 5. Risk estimation. 1. Exposure for dead fish is low for processing plants distant for farms but importing susceptible species. 2. Exposure assessment is high for processing plants on at in close proximity to farms and importing susceptible species.

**CONSEQUENCE ASSESSMENT**

If VHSV was introduced into the Scottish rainbow trout industry the consequences would be severe. The direct consequences of introduction of VHSV to Scotland’s rainbow trout industry would be in terms of infection, disease and mortality at the farm. Restrictions would be placed which affects movement of stocks and can be economically damaging to the industry. There would be increased costs in terms of the increased efforts in surveillance within control and buffer zones. The use of economic analysis methods would be required to conduct a thorough consequence assessment.

Further consequences would be dependent on how far VHSV could spread to both other farms and wild fish before detection. Analysis of these consequences will become possible with the application of new approaches to aquatic animal health risk analysis such as network analysis and risk mapping.
ACKNOWLEDGEMENTS

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