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Characterization of Infectious Hematopoietic Necrosis Virus (IHNV)

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Foreword

This series documents the scientific basis for the evaluation of aquatic resources and ecosystems in Canada. As such, it addresses the issues of the day in the time frames required and the documents it contains are not intended as definitive statements on the subjects addressed but rather as progress reports on ongoing investigations.

Research documents are produced in the official language in which they are provided to the Secretariat.

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ABSTRACT

Infectious Hematopoietic Necrosis Virus (IHNV) is a rhabdovirus that can result in the acute systemic disease infectious hematopoietic necrosis (IHN). The virus is endemic to British Columbia where it has been detected in freshwater and marine life stages of wild Sockeye Salmon as well as in marine cultured Atlantic Salmon. Through controlled laboratory exposure studies, Atlantic Salmon post smolts were shown to be nearly 100 times more susceptible to IHN disease than the native Sockeye Salmon at a similar life stage. However, this differential susceptibility is progressively enhanced with increasing age, whereas adult Atlantic Salmon remain highly susceptible to IHNV while Sockeye Salmon become resistant to IHN disease. Atlantic Salmon with acute IHN disease can shed enormous quantities of virus with levels peaking one to two days prior to the death of the animal. Once shed into the marine environment, the infectiousness of IHNV is rendered inactive by exposure to sunlight and natural biota present in the seawater; consequently, IHNV has an abbreviated lifespan whereby it can infect another host. Laboratory studies exposing fish to IHNV via immersion in virus contaminated water or through cohabitation with IHNV infected fish, have demonstrated that IHNV is transmitted and spread through waterborne exposure. IHNV dispersion from infected ocean-based net-pen Atlantic Salmon farms is dependent upon the number of diseased fish in the farm population (virus shedding fish), the decay rate of IHNV, and the water movement (currents) in the area of the infected farm. Epidemiological analyses of farm to farm spread of IHN disease during historical outbreaks in farmed Atlantic Salmon when industry-wide disease management practices were not implemented revealed that farming practices, such as boat movements and the use of shared personnel and contractors were leading causes of the dissemination of the disease amongst farms. Nevertheless, waterborne transmission of IHNV during these historical unmanaged outbreaks cannot be discounted as model simulations of IHNV dispersion performed using numerical particle releases in accordance with laboratory derived virus shedding and inactivation rates, demonstrate that neighboring naïve farms can become exposed to IHNV via waterborne transport from an unmanaged IHN diseased farm. However, significant advancement in the control of IHNV have been achieved through industry-wide implementation of a viral management plan and universal use of vaccination against IHNV. Under modern management practices that include movement controls and eradication, no farm to farm spread was detected in 2012 when IHNV was last reported in farmed Atlantic Salmon. Additionally, the IHNV vaccine has proven highly efficacious. In laboratory studies, APEX-IHN® prevented an outbreak of IHN disease in a population of Atlantic Salmon exposed to a lethal dose of IHNV. Furthermore, the vaccinated fish were incapable of transmitting IHN disease to cohabitating Sockeye Salmon. Since its licensure, over 60 million doses of APEX-IHN® have been administered to Atlantic Salmon in BC and to date there has been no detection of IHNV in an APEX-IHN® vaccinated farmed Atlantic Salmon.

Caractérisation du virus de la nécrose hématopoïétique infectieuse

RÉSUMÉ

Le virus de la nécrose hématopoïétique infectieuse (VNHI) est un rhabdovirus qui peut causer la maladie aiguë systémique de la nécrose hématopoïétique infectieuse (NHI). Le virus est endémique en Colombie-Britannique, où il a été détecté chez les stades biologiques en eau douce et marine de saumons rouges sauvages ainsi que chez des saumons atlantiques d'élevage en milieu marin. Dans le cadre d'études d'exposition contrôlée en laboratoire, des post-saumoneaux de l'Atlantique se sont avérés près de 100 fois plus sensibles à la NHI que le saumon rouge indigène au même stade biologique. Toutefois, cette différence de susceptibilité s'accroît avec l'âge, c'est-à-dire que les saumons atlantiques adultes demeurent très vulnérables au VNHI, tandis que le saumon rouge devient résistant à la maladie de la NHI. Un saumon atlantique atteint de la maladie aiguë de la NHI peut excréter une énorme quantité de virus, avec un pic un ou deux jours avant la mort de l'animal. Une fois le virus libéré dans le milieu marin, son infectiosité est désactivée par l'exposition à la lumière du soleil et au biote naturel présent dans l'eau de mer; par conséquent, la durée de vie du VNHI pendant laquelle il peut infecter un autre hôte s'en trouve abrégée. Des études en laboratoire exposant les poissons au VNHI par immersion dans de l'eau contaminée ou par cohabitation avec des poissons infectés par le VNHI ont démontré que le VNHI est transmis et propagé par l'exposition en milieu aqueux. La dispersion du VNHI à partir des élevages de saumon atlantique en parcs en filet en milieu océanique infecté dépend du nombre de poissons malades sur le site (poisson excrétant le virus), du taux de décomposition du VNHI et du mouvement de l'eau (courants) dans le secteur de l'exploitation infectée. Les analyses épidémiologiques de la propagation de la maladie causée par le VNHI entre les exploitations chez des saumons atlantiques d'élevage au cours d'épidémies historiques, alors qu'aucune pratique de gestion des maladies de l'industrie n'avait encore été mise en œuvre, ont révélé que les pratiques d'élevage, comme les déplacements en bateau et le partage du personnel et des entrepreneurs, constituaient les principales causes de la diffusion de la maladie entre les exploitations. Néanmoins, la transmission du VNHI par les courants au cours de ces épidémies historiques non gérées ne peut être écartée, car les simulations des modèles de dispersion du VNHI effectuées à l'aide de particules numériques libérées conformément aux taux dérivés d'excrétion et d'inactivation du virus démontrent que les poissons novices des exploitations voisines peuvent être exposés au VNHI par la voie aquatique à partir d'une exploitation dont l'infection à la NHI n'est pas gérée. Toutefois, des progrès importants ont été réalisés dans le contrôle du VNHI grâce à la mise en œuvre à l'échelle de l'industrie d'un plan de gestion virale ainsi que de l'utilisation généralisée du vaccin contre le VNHI. Selon les pratiques de gestion modernes, qui comprennent les contrôles des déplacements et l'éradication, aucune propagation entre exploitations n'a été détectée en 2012, année du dernier signalement du VNHI chez des saumons atlantiques d'élevage. De plus, le vaccin contre le VNHI s'est révélé très efficace. Dans les études en laboratoire, APEX-IHNMD^{MD} a permis d'éviter une épidémie de maladie de la NHI dans une population de saumon atlantique exposée à une dose létale du VNHI. En outre, les poissons vaccinés étaient incapables de transmettre la NHI aux saumons rouges avec qui ils cohabitaient. Depuis la certification du vaccin APEX-IHNMD^{MD}, plus de 60 millions de doses ont été administrées à des saumons atlantiques en Colombie-Britannique et, à ce jour, le VNHI n'a été détecté chez aucun poisson d'élevage ayant reçu le vaccin APEX-IHNMD^{MD}.

INTRODUCTION

Fisheries and Oceans Canada (DFO) has a regulatory role to ensure the protection of the environment while creating the conditions for the development of an economically, socially and environmentally sustainable aquaculture sector. Restoring funding to support federal ocean science programs to protect the health of fish stocks, to monitor contaminants and pollution in the oceans, and to support responsible and sustainable aquaculture industries in Canada has been identified as a top priority of the Minister of Fisheries, Oceans and the Canadian Coast Guard.

It is recognized that there are interactions between aquaculture operations and the environment (Grant and Jones, 2010; Foreman et al., 2015b). One interaction is the risk to wild salmon populations resulting from the potential spread of infectious diseases from Atlantic Salmon (*Salmo salar)* farms in British Columbia (BC) (Cohen, 2012). While several Atlantic Salmon farms are located within the migratory routes of Pacific salmon species, no risk assessment has been conducted to specifically determine the risk to wild fish populations associated with pathogens released from Atlantic Salmon farms.

DFO Aquaculture Management Division requested formal science advice on the risks of pathogen transfer from Atlantic Salmon farms to wild fish populations in BC. Given the complexity of interactions between pathogens, hosts and the environment, DFO will deliver the science advice through a series of pathogen-specific risk assessments followed by a synthesis.

PURPOSE OF THIS DOCUMENT

The information summarized in this document will assist in the environmental assessment of the risk to Fraser River Sockeye Salmon (*Oncorhynchus nerka*) due to the transfer of Infectious Hematopoietic Necrosis Virus (IHNV) from Atlantic Salmon farms located in the Discovery Islands area of British Columbia. This document is not designed to be an exhaustive review of IHNV but rather focuses on the natural distribution of IHNV and the characteristics that affect its transmissibility, pathogenicity and virulence to susceptible wild species occurring in the Discovery Islands area.

PATHOGEN AND DISEASE

GENERAL DESCRIPTION

Infectious Hematopoietic Necrosis Virus (IHNV) is a rhabdovirus that can cause an acute systemic disease called infectious hematopoietic necrosis (IHN) that can lead to the death of the infected host. The IHNV genome is negative sense, single stranded RNA and contains six genes in the order 3'-N-P-M-G-NV-L-5', representing the nucleocapsid, phosphoprotein, matrix protein, glycoprotein, non-virion protein and polymerase protein genes, respectively (Kurath and Leong, 1985; Morzunov et al., 1995). The disease, IHN, has led to significant mortality in both wild and cultured salmon and trout populations (Bootland and Leong, 1999). Due to the contagious nature and potential to cause large losses of fish, IHN is listed as reportable to the World Organization for Animal Health (OIE) (Dixon et al., 2016).

GEOGRAPHIC DISTRIBUTION AND GENETIC TYPES

The virus was first identified in Sockeye Salmon in north western North America in the 1950s (Rucker et al., 1953; Watson et al., 1954). In North America, its native range is from Alaska to California and in-land to Idaho and northern Montana. The virus was spread to other areas of

North America through the historical movement of infected live fish and/or eggs (Plumb, 1972; Wolf, 1988; Plumb and Hanson, 2011), although these introductions were eradicated and the present range of IHNV is limited to the west coast of North America where it is endemic. Outside of North America, IHNV has been reported to be present in Europe and Asia (OIE, 2012; Dixon et al., 2016).

Different variants of IHNV have been characterized primarily by nucleotide sequence diversity within the virus glycoprotein gene (Emmenegger et al., 2000; Garver et al., 2003; Kurath et al., 2003). There are five recognized genogroups of IHNV worldwide (Nishizawa et al., 2006; He et al., 2013) and three of these genogroups (U, M, and L) are endemic to North America. The U, M, and L genogroups correlate with geography (Figure 1) such that the U genogroup viruses are found in the northern (or 'upper') regions of North America (ranging from Alaska to Mid-Oregon), the M genogroup viruses are found in the 'middle' regions (Southern Idaho aquaculture region, Columbia River Basin and Washington Coast) and the L genogroup viruses are found in the southern (or 'lower') regions (ranging from Southern Oregon to California) (Emmenegger et al., 2000; Garver et al., 2003; Kurath et al., 2003; Kelley et al., 2007; Breyta et al., 2013).

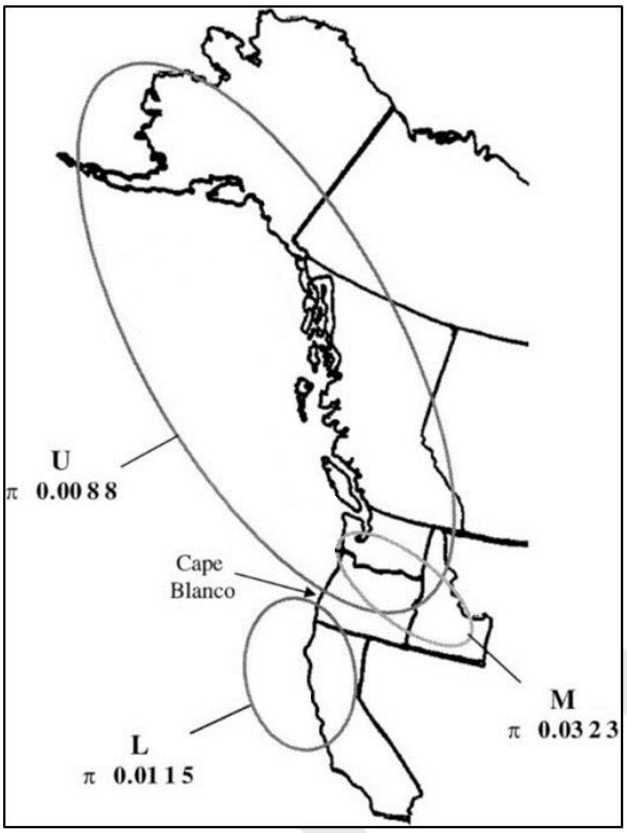

Figure 1. Map depicting IHNV genogroups (U, L, M) in the north eastern Pacific Ocean and west coast of North America (modified from: Molecular Epidemiology of Aquatic Pathogens Infectious Hematopoietic Necrosis Virus website) Infectious Hematopoietic Necrosis Virus *Intrapopulational diversity values are from data presented in Kurath et al. (2003).*

All isolates from British Columbia belong to the U genogroup (Kurath et al., 2003). U genogroup viruses are the most broadly distributed, encompassing isolates from Oregon, Idaho, Montana, Washington, British Columbia, Alaska, Russia and Japan (Rucker et al., 1953; Guenther et al., 1959; Wingfield et al., 1969; Grischkowsky and Amend, 1976; Busch, 1983; Kurath et al., 2003; Nishizawa et al., 2006). Despite its wide geographic distribution, the U genogroup is the least

genetically diverse of the three genogroups. Within the U genogroup, two broad subgroups, designated UC and UP, have been recognized. The UC viruses are predominately located in the Columbia River Basin while the UP viruses represent all northern portions of the virus range, including British Columbia (Black et al., 2016).

Virus typing of IHNV isolates obtained outside of North America highlights geographic differentiation yet reveals a common ancestor with North American strains supporting the hypothesis that these isolates were introduced through movement of contaminated products. In Europe, IHNV was first detected in 1987 in Italy and France, and has since been spread by trade of infected fish to become a widespread threat to the European Rainbow Trout aquaculture industry (Enzmann et al., 2010). The European isolates are designated into the E genogroup (Kurath, 2012) and are most closely related to the North American M group (Enzmann et al., 2005; Kolodziejek et al., 2008). In Asia (Japan, Korea, Taiwan, China), IHNV isolates group into the J genogroup that phylogenetically is derived from the U genogroup (Nishizawa et al., 2006).

HOST RANGE AND SUSCEPTIBILITY

Natural infections and controlled laboratory exposure studies indicate IHNV predominately infects salmonid fishes (Table 1, Table 2). The occurrence of IHNV in salmon, non-salmon, and invertebrate species is summarized in the following sections.

Salmonids

The natural host range of IHNV includes several salmonid species of the genera *Oncorhynchus*, *Salmo*, *Salvelinus*, and *Thymallus*, with each varying in susceptibility as reported by numerous researchers (Table 1, Table 2) (reviewed in Bootland and Leong (1999)). Of the Pacific salmon species (*Oncorhynchus* spp.), Coho Salmon (*O. kisutch*) and Pink Salmon (*O. gorbuscha*) are considered least susceptible to IHN disease (Table 2) as there are no reports of outbreaks in wild or free-ranging populations and experimental exposures showed low to no mortality (Wingfield et al., 1970; Chen et al., 1990). Despite the absence of disease, these species can be infected as IHNV has been isolated from fry and adults in natural and laboratory infections (Hedrick et al., 1987; LaPatra et al., 1989; Eaton et al., 1991; Kurath et al., 2003).

Conversely, Sockeye Salmon and kokanee, Chinook Salmon (*O. tshawytscha*), and steelhead/Rainbow Trout (*O. mykiss*) are considered highly susceptible to IHN disease (Table 1). Among the *Salmo* species, the Atlantic Salmon (*S. salar*) and Brown Trout (*S. trutta*) are susceptible to IHNV infection and disease.

Table 1. Salmonid species in which IHNV detections have occurred (reproduced from Bootland and Leong (1999)).

Common name	Scientific name	Reference
Sockeye Salmon	*O. nerka*	Rucker et al. (1953)
Chinook Salmon	*O. tshawytscha*	Sano et al. (1977)
Chum Salmon	*O. keta*	Ross et al. (1960)
Masou (Cherry) Salmon	*O. masou*	Sano et al. (1977)
Biwa Salmon	*O. masou rhodurus*	Sano et al. (1977)
Amago	*O. rhodurus*	Sano et al. (1977)
Rainbow Trout	*O. mykiss*	Amend et al. (1969)
Steelhead trout	*O. mykiss*	Amend et al. (1969)
Cutthroat Trout	*O. clarki*	Parisot and Pelnar (1962)
Atlantic Salmon	*S. salar*	Mulcahy and Wood (1986)
Brown Trout	*S. trutta*	Yamazaki and Motonishi (1992)
Brook Trout	*S. fontinalis*	Yamazaki and Motonishi (1992)

Common name	Scientific name	Reference
Japanese Charr	*S. leucomaenis*	Kimura and Awakura (1977)

Table 2. Species of low susceptibility to IHNV (reproduced from Bootland and Leong (1999)).

Common name	Scientific name	Reference
Coho Salmon	*O. kisutch*	Wingfield and Chan (1970)
Pink Salmon	*O. gorbuscha*	Follett et al. (1997)
Lake Trout	*S. namycush*	Yamamoto and Clermont (1990)
Arctic Charr	*S. alpinus*	Follett et al. (1997)
Arctic Grayling	*Thymallus arcticus*	Follett et al. (1997)
Mountain Whitefish	*Prosopium williamsoni*	Bootland et al. (unpublished)

In western North America, numerous instances of IHNV infection and disease have occurred among Atlantic Salmon (Mulcahy and Wood, 1986; St-Hilaire et al., 2002; Saksida, 2006). Among the field outbreaks, disease and mortality in Atlantic Salmon was highest in young fish within one year of salt water entry (Garver et al., 2013). Nonetheless, older adult life stages have exhibited disease under field and laboratory environments (Saksida, 2006; Garver et al., 2013), suggesting that Atlantic Salmon do not become refractory to IHN disease with increasing age as observed in Pacific salmon species (as described in section "Biological factors involved in modulating host susceptibility to IHNV). Laboratory trials have further demonstrated the extreme susceptibility of Atlantic Salmon by transmitting IHN disease through immersion, injection, or through cohabitation with infected Sockeye Salmon (Traxler et al., 1991, 1993) with disease occurring after a minimum lethal dose of 10 plaque forming units (pfu) /mL (Garver et al., 2013).

Non-salmonid fish

Kent et al. (1998) tested wild fish captured in and around marine salmon cages as well as "open" ocean locations of British Columbia for several pathogens including IHNV. A summary of the findings specific to IHNV has been provided (Table 3). In many cases, the sample sizes of individual species were small, nonetheless, of the 56 different species tested, Pacific Herring (*Clupea pallasii*) (n=1), Shiner Perch (*Cymatogaster aggregata*) (n=1) and Tubesnout (*Aulorhynchus flavidus*) (n=2) tested positive for IHNV. However, these fish did not show clinical signs of disease. Interestingly, the virus positive Tubesnout and Shiner Perch were collected at an Atlantic Salmon farm during an outbreak of IHNV, yet resampling of these species in the same location proved negative six weeks after the farm was fallowed (Kent et al., 1998), suggesting these are spill-over host infections resulting from the elevated IHNV exposure due to their close proximity to highly infected farmed fish. Similarly, Pacific Herring are likely only short-term hosts as laboratory trials failed to establish IHNV infection and disease in Pacific Herring (Hart et al., 2011). Based on the scientific literature, IHNV can infect several non-salmonid species; however, the infection appears to be transient and whether such infections could result in an alternative or reservoir hosts for IHNV remains unknown.

Table 3. Summary of wild marine fish captured in and around marine Atlantic Salmon cages (near) and other coastal areas (offshore) of BC that were screened for IHNV using cell culture (from Kent et al. (1998)). Species in bold are those in which IHNV was identified.

Species	Scientific name	Location of Sample (number +ve IHNV/ total tested	
		Near	Offshore
Pacific Herring	*Clupea pallasii*	**0/127**	**1/162**
American Shad	*Alosa sapidissima*	-	0/3
Pile Perch	*Rhacochilus vacca*	0/2	-

Species	Scientific name	Location of Sample (number +ve IHNV/ total tested	
		Near	Offshore
Shiner Perch	*Cymatogaster aggregata*	**1/307**	**0/11**
Tubesnout	*Aulorhynchus flavidus*	**2/72**	**-**
Bay Pipefish	*Syngnathus leptochyncus*	0/1	-
Threespined Stickleback	*Gasterosteus aculeatus*	0/42	0/4
Pacific Cod	*Gadus macrocephalus*	-	0/34
Walleye Pollock	*Gadus chalcogrammus*	-	0/23
Whitespotted Greenling	*Hexagrammos stelleri*	0/1	0/7
Lingcod	*Ophiodon elongatus*	0/7	0/24
Sablefish	*Anoplopoma fimbria*	0/3	0/15
Rockfish sp.	*Sebastes* sp.	0/34	-
Black Rockfish	*S. melanops*	0/3	-
Boccaccio	*S. paucispinis*	-	0/1
Canary Rockfish	*S. pinniger*	-	0/14
Copper Rockfish	*S. caurinus*	-	0/14
Pacific Ocean Perch	*S. alutus*	-	0/6
Quillback Rockfish	*S. maliger*	0/13	0/1
Redbanded Rockfish	*S. babcocki*	-	0/9
Redstripe Rockfish	*S. proriger*	-	0/37
Rougheye Rockfish	*S. aleutianus*	-	0/7
Sharpchin Rockfish	*S. zacentrus*	-	0/10
Silvergray Rockfish	*S. brevispinis*	-	0/11
Yelloweye Rockfish	*S. ruberrimus*	-	0/3
Pacific Spiny Dogfish	*Squalus suckleyi*	0/2	0/10
Spotted Ratfish	*Hydrolagus colliei*	0/1	0/6
Skate	*Raja* sp.	-	0/2
Sculpin	*Cottus* sp.	0/7	0/4
Threadfin Sculpin	*Icelinus filamentosus*	-	0/3
Spinyhead Sculpin	*Dasycottus setiger*	-	0/1
Soft Sculpin	*Malacocottus zonurus*	-	0/4
Right eyed flounder	Pleuronectidae	0/4	-
Arrowtooth Flounder	*Atheresthes stomias*	-	0/9
Flathead Sole	*Hippoglossoides elassodon*	-	0/1
Pacific Halibut	*Hippoglossus stenolepis*	-	0/23
Petrale Sole	*Eopsetta jordani*	-	0/5
Rex Sole	*Glyptocephalus zachirus*	-	0/10
Rock Sole	*Lepidopsetta bilineata*	0/6	0/2
Slender Sole	*Lyopsetta exilis*	-	0/6
Starry Flounder	*Platichthys stellatus*	0/2	0/1
Pacific Sanddab	*Citharichthys sordidus*	0/3	0/8
Eulachon	*Thaleichthys pacificus*	-	0/4
California Smoothtongue	*Leuroglossus stilbius*	-	0/1
Chub Mackerel	*Scomber japonicus*	0/25	-
Wattled Eelpout	*Lycodes paamiuti*	-	0/3
Shortfin Eelpout	*L. brevipes*	-	0/1
Prickleback	*Lumpenus* sp.	-	0/1
Snake Prickleback	*L. sagitta*	-	0/2
Pacific Sand Lance	*Ammodytes hexapterus*	0/1	0/10
Saddleback Gunnel	*Pholis ornata*	0/1	-
Giant Wrymouth	*Cryptacanthodes giganteus*	-	0/1
Pygmy Poacher	*Odontopyxis trispinosa*	-	0/1
Warty Poacher	*Chesnonia verrucosa*	-	0/1
Plainfin Midshipman	*Porichthys notatus*	-	0/1
Northern Lampfish	*Stenobrachius leucopsarus*	-	0/8

In freshwater, White Sturgeon (*Acipenser transmontanus)* has been shown to be of low susceptibility to IHNV (LaPatra et al., 1995) while Pacific Lamprey (*Entosphenus tridentatus*), appear resistant to infection (Kurath et al., 2013). In contrast, juvenile Northern Pike succumbed to IHN disease (Dorson et al., 1987).

Invertebrate hosts

Several invertebrate species have tested positive for IHNV; however, virus replication has not been confirmed. While it is possible that these invertebrates may serve as biological vectors, the available data suggest that they harbour the virus for only brief periods of time, during which they may serve as mechanical vectors. These species include mayflies (*Callibaetis* spp.) (Shors and Winston, 1989), leeches (*Piscicola salmositica*) (Mulcahy et al., 1990), and ectoparasitic copepods (*Salmincola* sp. and *Lepeophtheirus salmonis*) (Mulcahy et al., 1990). These invertebrates become contaminated with the virus from the water or directly from infected fish. In a laboratory study, the salmon louse (*L. salmonis*) was reported to acquire IHNV either after a one hour water bath exposure or after parasitizing IHNV infected Atlantic Salmon. In either instance, the acquisition of IHNV in sea lice was ephemeral with sea lice remaining IHNV positive for a maximum of 24 hours. Attachment of IHNV positive *L. salmonis* to naïve Atlantic Salmon resulted in IHNV transmission and disease development in the parasitized fish (Jakob et al., 2011).

Biological factors involved in modulating host susceptibility to IHNV

IHNV infection and the development of disease depends on factors such as host species, genetic stock, age, size, previous exposure status, and the virus strain (Plumb and Hanson, 2011).

As shown in Table 1 and Table 2, salmon and trout species vary in susceptibility to IHN disease and although many of these species exist in British Columbia (Table 4), the disease has predominately been reported in Atlantic and Sockeye Salmon. Laboratory studies evaluating susceptibility of these two species, have demonstrated that when exposed to IHNV from BC, Atlantic Salmon are more susceptible than Sockeye Salmon to clinical IHN disease. For instance Traxler et al. (1993), revealed that of Atlantic and Sockeye Salmon smolts receiving an equal bath exposure, only Atlantic Salmon developed IHN disease and mortality. Moreover, virus exposure studies aimed at determining the lowest dose of IHNV required to cause disease and mortality in Atlantic Salmon smolts was 10 pfu/mL (Garver et al., 2013), while a dose of at least 100 pfu/mL was required for Sockeye Salmon smolts (Long et al., 2017) (Table 5). Hence Atlantic Salmon are at least ten times more susceptible to IHNV disease than Sockeye Salmon.

Table 4. IHNV susceptible salmon and trout species present in BC as determined through virus detections in BC or elsewhere. IHNV detections may or may not have been associated with clinical signs of disease or mortality (ND = no data; NA = not applicable; NS = not specified; smolt/post smolt = freshwater or <1 year in salt water; adults = >1 year old).

Species	Life History Stage	Freshwater	Saltwater	References
Atlantic Salmon (*S. salar*)	Egg/milt	ND	NA	
	Fry/parr	YES	NA	Mulcahy and Wood (1986) Kurath et al. (2016)
	Smolt/post-smolt	YES	YES	SW: Saksida (2006) SW <1 yr + >1 yr Traxler et al. (1993); St-Hilaire et al. (2002)
	Adult	NA	YES	Saksida (2006) (SW >1 yr)
Chinook Salmon (*O. tshawytscha*)	Egg/Milt	ND	NA	
	Fry/Parr	YES	NA	Follett et al. (1987); Bendorf et al. (2007)
	Smolt/Post-smolt	ND	NO	SW: Traxler et al. (1993)

Species	Life History Stage	Freshwater	Saltwater	References
	Adult	YES	ND	FW: Anderson et al. (2000) returning fish Follett et al. (1987)
Chum Salmon (*O. keta*)	Egg/Milt	YES/NO	NA	Yoshimizu et al. (1989) (Injected eyed eggs yes, before no)
	Fry/Parr	YES	NA	Follett et al. (1987) British Columbia Ministry of Fisheries (1998)
	Smolt/Post-smolt	ND	ND	
	Adult	YES	ND	Follett et al. (1987) returning adult
Coho Salmon (*O. kisutch*)	Egg/Milt	ND	NA	
	Fry/Parr	NO	NA	FW: Follett et al. (1987) fry LaPatra et al. (1989) alevin
	Smolt/Post-smolt	ND	ND	
	Adult	YES	ND	FW: LaPatra et al. (1989) spawning
Pink Salmon (*O. gorbuscha*)	Egg/Milt	ND	NA	
	Fry/Parr	YES*	NA	Follett et al. (1997)*****refractory
	Smolt/Post-smolt	ND	ND	
	Adult	ND	ND	
Sockeye Salmon (*O. nerka*)	Egg/Milt	YES	NA	Mulcahy et al. (1987) (milt)
	Fry/Parr	YES	NA	Amend et al. (1969) Mulcahy and Bauersfeld (1983) Mulcahy et al. (1983) Williams and Amend (1976) Follett et al. (1997) FW: Garver et al. (2006)
	Smolt/Post-smolt	ND	YES	SW: Traxler et al. (1993)
	Adult	YES	YES	FW: Mulcahy et al. (1987) (spawning males) Mulcahy et al. (1982) (spawning and non-spawning) SW: Traxler et al. (1997)
Kokanee (*O. nerka*)	Egg/Milt	ND	NA	
	Fry/Parr	YES	NA	FW: Garver et al. (2006)
	Smolt/Post-smolt	ND	NA	
	Adult	YES	NA	Follett et al. (1987) Traxler (1986)
Steelhead trout (*O. mykiss*)	Egg/Milt	YES	NA	Mulcahy et al. (1987) (milt)
	Fry/Parr	YES	NA	FW: Anderson et al. (2000) FW: Garver et al. (2006)
	Smolt/Post-smolt	YES	ND	Breyta et al. (2013)
	Adult	YES	YES	FW: Mulcahy et al. (1987) (spawning male) Anderson et al. (2000) SW: Breyta et al. (2013)
Rainbow Trout (*O. mykiss*)	Egg/Milt	YES	NA	Amend (1975)
	Fry/Parr	YES	NA	Bendorf et al. (2007) Amend et al. (1969) Overturf et al. (2010) Park et al. (1993) Purcell et al. (2010) Yamamoto and Clermont (1990) Garver et al. (2006)
	Smolt/Post-smolt	ND	NA	
	Adult	YES	NA	Amend (1975) (spawning and pre-spawning)
Brook Trout (*S. fontinalis*)	Egg/Milt	ND	NA	
	Fry/Parr	YES	NA	Bootland et al. (1994) LaPatra et al. (1993a)

Species	Life History Stage	Freshwater	Saltwater	References
	Smolt/Post-smolt	ND	NA	
	Adult	ND	NA	
Brown Trout (*S. trutta*)	Egg/Milt	ND	NA	
	Fry/Parr	YES	NA	LaPatra and Fryer (1990)
	Smolt/Post-smolt	ND	NA	
	Adult	ND	NA	
Cutthroat Trout (*O. clarki*)	Egg/Milt	ND	NA	
	Fry/Parr	NS	NA	Parisot and Pelnar (1962) (laboratory note which stated injection of Cutthroat Trout with IHNV demonstrated host range extension, no further details were provided)
	Smolt/Post-smolt	NS	NA	
	Adult	NS	NA	
Lake Trout (*S. namycush*)	Egg/Milt	ND	NA	
	Fry/Parr	YES	NA	Follett et al. (1997)
	Smolt/Post-smolt	NA	NA	
	Adult	ND	NA	

Table 5. Cumulative percent mortality measured in Atlantic and Sockeye Salmon bath exposed to IHNV at doses ranging from 10 to 10,000 plaque forming units (pfu) per milliliter. Data for Atlantic and Sockeye Salmon as reported in Garver et al. (2013) and Long et al. (2017), respectively. Numbers in parentheses are the average cumulative percent mortality of the replicate tanks. NA = not applicable.

Virus dose (pfu/mL)	Cumulative percent mortality		
	Atlantic Salmon Fish size = 71 g	Sockeye Salmon	
		Fish size = 5.5 g	Fish size = 28 g
10	0, 20 (10)	0, 0, 0 (0)	NA
100	18, 40, 43 (34)	0, 0, 0 (0)	0, 1, 3 (1.3)
1000	23, 30, 50 (34)	8, 12, 16 (12)	5, 12 (8.5)
10000	23, 33, 38 (31)	8, 16, 36 (20)	NA

Differences in susceptibility may also vary within a species. For example, Wertheimer and Winton (1982) demonstrated that Chinook Salmon from a genetically-defined stock from Washington were more susceptible to IHNV than Chinook Salmon stocks from Alaska. Additionally, different stocks of steelhead trout from rivers in Washington State showed significant differences in mortality to IHNV (Breyta et al., 2014). Furthermore, it was demonstrated through controlled laboratory challenge studies conducted at the Pacific Biological Station, that one stock of Fraser River Sockeye Salmon exhibited higher susceptibility to IHNV than a Sockeye Salmon stock from the Upper Columbia River (Figure 2). After being exposed for one hour in a static bath of IHNV at 2×10^5 plaque forming units per milliliter, the Fraser River stock (Pitt River) had 30% higher mortality after 50 days than observed in the Upper Columbia River stock (Okanagan River) (Figure 2).

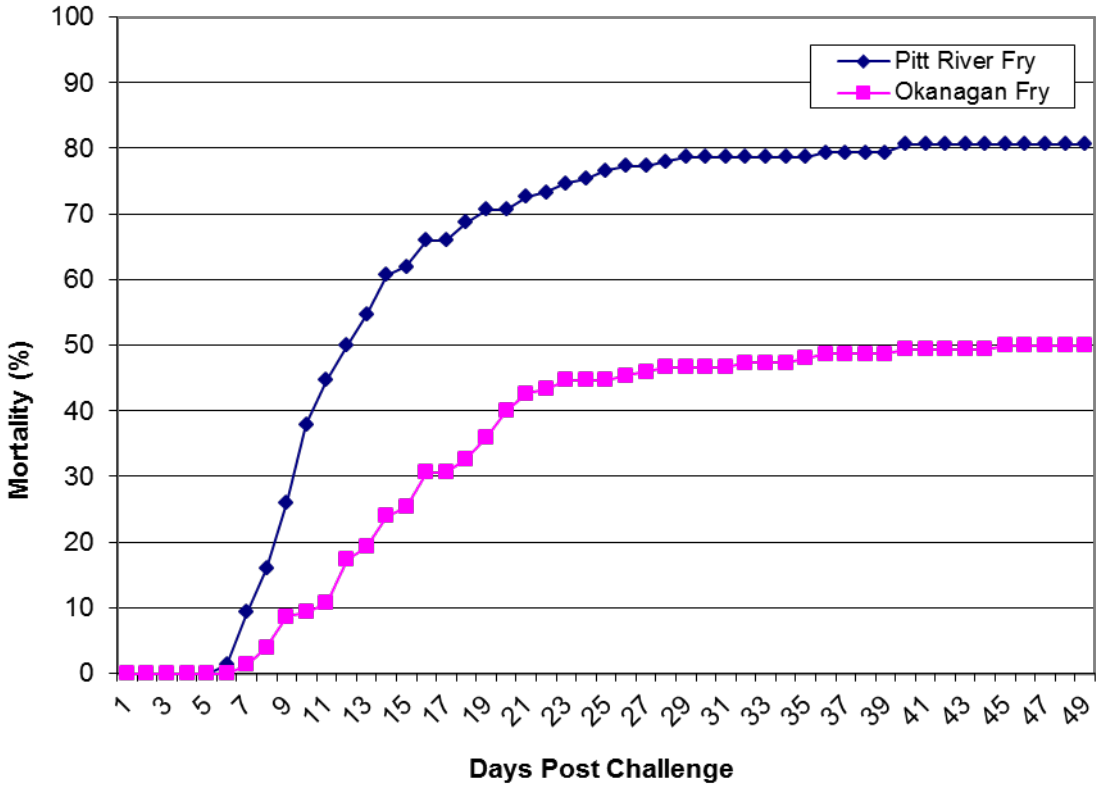

Figure 2. Cumulative percent mortality of Pitt River (Fraser River stock) and Okanagan River (upper Columbia River stock) Sockeye Salmon fry post waterborne IHNV (U genogroup) challenge.

Susceptibility to IHN disease is also highly dependent upon the life stage, age, and/or size of the host upon exposure to virus (LaPatra et al., 1990). Natural IHNV associated mortality has been documented in fry and juvenile Pacific salmon whereas neither disease nor epizootics have been reported in returning adult Pacific salmon that have tested positive for IHNV. Furthermore, results of laboratory challenge studies have corroborated field observation in that virus exposed Rainbow Trout become increasingly resistant to IHN disease with increased age and weight (Amend and Nelson, 1976; LaPatra et al., 1994; Troyer et al., 2000; Bergmann et al., 2003). This phenomenon of decreasing disease susceptibility with increasing age has also been documented in Sockeye Salmon. Laboratory studies exposing fry to waterborne IHNV yielded upwards of 80% mortality, while in a similar virus exposure of sockeye smolts, mortality rarely exceeded 16% (Long et al. (2017) and Garver unpublished data). Additionally smolt and yearling Sockeye Salmon in comparison to fry, show less severe histopathological changes with minor necrosis in kidney, spleen, pancreas and liver and little sloughing of the intestinal mucosa and no faecal casts (Yasutake, 1978; Burke and Grischkowsky, 1984; Traxler, 1986). This contrast in IHNV pathology between fry and older life stage Sockeye Salmon is further substantiated when comparing fry with spawning adult Sockeye Salmon. Yamamoto et al. (1989) found that spleen and kidney of adult sockeye infected with IHNV did not show the massive necrosis as observed in fry. Nevertheless adult fish are capable of being infected with IHNV despite the lack of disease at this lifestage. Infection of adult Sockeye Salmon with IHNV is predominately detected just before and after the fish spawn (Table 6) (Mulcahy et al., 1982).

Another determinate of whether IHNV infections lead to disease is whether the virus strain is virulent towards its host. Host specific virulence has been correlated with IHNV genogroup in field surveillance and laboratory studies. The U, M and L genogroups exhibit the highest virulence to *O. nerka* (Sockeye Salmon and kokanee), *O. mykiss* (steelhead and Rainbow Trout), and *O. tshawytscha* (Chinook Salmon), respectively (Garver et al., 2006; Bendorf et al., 2007; Kelley et al., 2007; Peñaranda et al., 2009; Purcell et al., 2009; Breyta et al., 2013). However, these genogroup specific virulence patterns are not definitive and wide ranging virulence can occur even among isolates within a single genogroup (Mochizuki et al., 2009; Wargo et al., 2010; Breyta et al., 2014). For instance, among Pitt River Sockeye Salmon immersion exposed for one hour to IHNV (2 x 10^5 pfu/mL), mortality varied from 31% to 80% dependent upon which U-genogroup virus isolate fish were exposed to (Figure 3). Thus, in the absence of a specific molecular marker for IHNV virulence, forecasting the virulence of an isolate based on genogroup is not completely accurate. To fully understand the virulence of an isolate, each IHNV requires strain typing and a virulence test in different fish species and stocks.

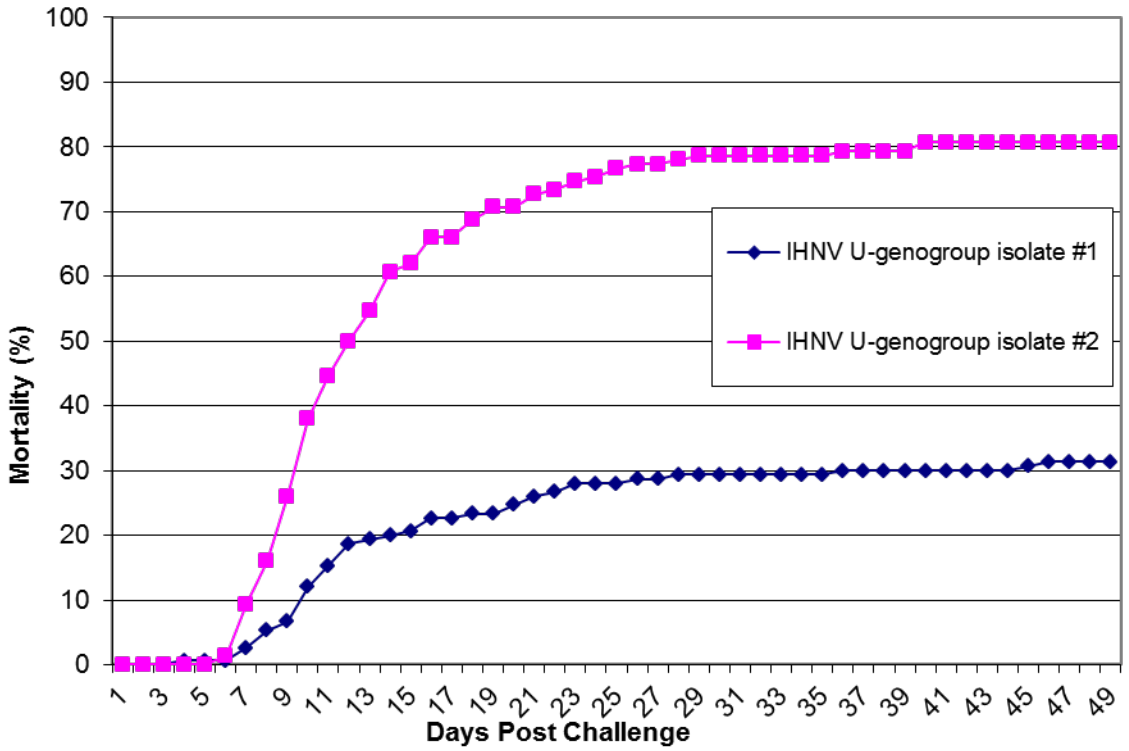

Figure 3. Cumulative percent mortality of Pitt River (Fraser River stock) Sockeye Salmon fry exposed through waterborne challenge to IHNV isolate #1 or #2 of the U genogroup.

Salmonid immunity and vaccinology

The history of exposure to IHNV can also significantly alter the host's susceptibility to disease. It is generally accepted that fish that have survived an IHNV exposure mount protective immunological responses often accompanied by measurable IHNV specific neutralizing antibodies in the plasma (Amend and Smith, 1974). Through laboratory studies, these antibodies have been shown to be protective against IHNV, either by lack of reinfection or from

naïve fish remaining free of disease after receiving a passive transfer of serum (LaPatra et al., 1993b). The ability to produce antibodies following exposure to the virus has been shown through multiple life stages ranging from fry to adult. In particular, neutralizing antibodies have been measured in wild adult returning Sockeye Salmon in the marine waters of BC (Traxler et al., 1997) and in general it is believed that fish surviving significant IHNV exposure remain protected against disease.

With the knowledge that salmon can mount a protective response to IHNV infections, researchers have employed various IHNV vaccine formulations to artificially induce this protective response to safeguard against IHN disease. Vaccine types employed against IHNV consist of a killed, attenuated, subunit, and DNA vaccine. The killed vaccine was produced by inactivating an isolate of IHNV using either β propiolactone (Amend, 1976) or formalin (Nishimura et al., 1985). The vaccine was best administered as an intraperitoneal injection. However, the use of these vaccines has not always proven effective. In BC, the salmon industry utilized a killed IHNV preparation that appeared to have little to no protection in the farm populations that used them during the IHNV epidemic occurring from 2001 to 2003 (Saksida, 2006). Researchers have also focused on development of an attenuated IHNV. The vaccine was produced through multiple passage of an IHNV isolate through steelhead trout cell culture (Fryer et al., 1976); however, due to safety concerns of residual virulence, the vaccine was never successfully implemented into a field scenario. As a safer alternative, researchers turned to developing a subunit vaccine consisting of a single IHN virus protein produced and purified from bacteria. Fish immunized with a crude lysate containing the viral glycoprotein showed protection against a lethal challenge of IHNV yet the subunit vaccine was never produced commercially. Recognizing the importance of the viral glycoprotein as a key antigen in stimulating an IHNV protective response in a host, researchers utilized recombinant DNA technology to develop a DNA vaccine consisting of a plasmid expressing the IHNV glycoprotein gene that when delivery into the muscle of a fish would synthesize the antigenic protein in recipient cells and initiate an immune response. The vaccine proved to be extremely protective against IHN disease in Rainbow Trout (Anderson et al., 1996; Corbeil et al., 1999; Corbeil et al., 2000a; Corbeil et al., 2000b; LaPatra et al., 2000; LaPatra et al., 2001a), numerous species of Pacific salmon species (Garver et al., 2005b), and Atlantic Salmon (Traxler et al., 1999). Moreover the protective immunity afforded by the DNA vaccine is long-lived (Kurath et al., 2006) with limited plasmid persistence and biodistribution (Garver et al., 2005a) making it a favourable candidate for commercial applications.

Being a DNA vaccine, APEX-IHN® does not use the whole virus but rather consists of only one viral gene incorporated into a plasmid which when expressed induces an immune response to protect against IHNV. The vaccine is directly injected into the musculature of fish whereby the viral antigen is then produced in the cells containing the plasmid prompting an immune response akin to a natural infection. Plasmids are not organisms, they are not self-replicating, nor are they infectious. Consequently, vaccinated fish themselves are not contagious and do not shed infectious vaccinated material.

In July 2005, the IHNV DNA vaccine, APEX-IHN®, produced by Elanco (formally Novartis) received licensure from the Canadian Food Inspection Agency. However, it wasn't until 2015 that APEX-IHN® was universally administered throughout the BC farmed Atlantic Salmon industry. Since licensure, over 60 million doses have since been administered in farmed Atlantic Salmon in BC and to date there has been no reported occurrence of IHNV in an APEX-IHN® vaccinated farm. Currently, all marine Atlantic Salmon farming companies voluntarily vaccinate their fish for IHNV (Wade, 2017).

IHNV IN BRITISH COLUMBIA

Pacific salmonids

In British Columbia, where IHNV isolates belong to the U genogroup, it is most common to detect the virus within Sockeye Salmon and kokanee populations. In some instances, IHNV infection has caused severe disease in this species that resulted in losses at fry and juvenile stages (Traxler, 1986; Traxler and Rankin, 1989). Albeit less common, IHNV in BC has been isolated from Rainbow Trout (Amend et al., 1969), steelhead trout (DFO Pacific Biological Station, Nanaimo, BC, Diagnostic Report 2014-11, unpublished data), Chum Salmon (Garth Traxler, DFO (retired), 3190 Hammond Bay Rd, Nanaimo, BC, Canada V9T 6N7, pers. comm.) and Chinook Salmon (DFO Pacific Biological Station, Nanaimo, BC, Diagnostic Report 2014-139, unpublished data).

Long-term monitoring of British Columbia Sockeye Salmon stocks from the Skeena, Fraser, and Columbia River watersheds has revealed that the annual prevalence of IHNV is highly variable both within and among stocks (Table 6). How the virus is perpetuated and what factor(s) drive the variable occurrence of IHNV in these populations remains unknown. However, recent identification of IHNV in newly smolted and apparently healthy Fraser River Sockeye Salmon during their marine migration supports the role of a subclinical carrier state in the life cycle of IHNV and a possible mechanism towards its perpetuation within a host population (Garver, unpublished data). Laboratory studies confirm persistence of IHNV in surviving Sockeye Salmon smolts (Müller et al., 2015), thus suggesting the infections detected in asymptomatic juvenile fish from the marine environment (Table 6) likely represent survivors from a naturally occurring IHNV exposure. Although the infectious nature of virus detected in these asymptomatic carriers is unknown, it is possible that these fish may be a potential source of virus to sympatric salmonid species such as farmed Atlantic Salmon held in marine cages.

Table 6. Prevalence (%) of IHNV in five populations of spawning adult Sockeye Salmon (O. nerka) in British Columbia based on cell culture results (Data provided by G. Traxler and K. Garver).

	Fraser River Watershed				Skeena River Watershed				Columbia River Watershed	
	Weaver Creek		Nadina River		Pinkut Creek		Fulton River		Okanagan River	
Year	N	Prevalence (%)	N	Prevalence (%)	N	Prevalence (%)	N	Prevalence (%)	N	Prevalence (%)
1984	0	-	0	-	98	87.8	324	63.2	0	-
1985	0	-	0	-	114	54.4	266	48.9	0	-
1986	0	-	0	-	146	43.8	429	66.4	0	-
1987	131	38.2	69	0.0	134	60.4	467	55.7	0	-
1988	101	3.0	111	2.7	171	48.5	491	71.7	0	-
1989	110	0.0	73	0.0	162	4.3	670	2.0	0	-
1990	115	26.1	91	0.0	218	1.8	636	4.7	0	-
1991	202	17.3	87	14.9	70	71.4	523	67.5	0	-
1992	147	0.0	66	0.0	61	34.4	283	60.4	0	-
1993	116	9.5	94	0.0	88	60.2	338	60.3	0	-
1994	131	0.0	37	0.0	106	17.0	327	42.0	0	-
1995	176	0.0	78	10.2	108	30.6	297	21.7	0	-
1996	70	20.0	79	1.3	81	62.9	327	10.0	0	-
1997	68	22.1	39	23.1	63	41.2	369	28.5	0	-
1998	96	2.1	108	0.0	75	72.0	490	45.5	0	-
1999	116	0.0	118	16.1	105	5.7	421	57.0	0	-
2000	108	50.0	129	33.3	72	34.7	466	0.9	109	86.2
2001	117	23.1	137	21.2	101	58.4	417	65.1	75	33.3
2002	111	0.9	76	43.4	101	58.4	340	21.2	59	8.5
2003	86	15.1	100	0.0	108	0.0	403	0.5	213	3.8

Year	Fraser River Watershed				Skeena River Watershed				Columbia River Watershed	
	Weaver Creek		Nadina River		Pinkut Creek		Fulton River		Okanagan River	
	N	Prevalence (%)	N	Prevalence (%)	N	Prevalence (%)	N	Prevalence (%)	N	Prevalence (%)
2004	120	0.0	126	0.0	100	0.0	456	0.0	185	5.4
2005	111	0.0	119	0.8	109	0.0	448	0.0	253	0.4
2006	120	0.0	113	0.0	103	0.0	448	0.0	222	0.0
2007	0	-	73	0.0	110	11.8	542	38.0	387	0.3
2008	21	0.0	99	12.1	135	0.0	294	26.6	569	0.0
2009	139	0.0	101	0.0	113	0.0	452	0.0	303	0.0
2010	100	2.0	108	52.8	106	0.0	437	0.0	250	24.4
2011	100	0.0	68	4.4	105	0.0	344	0.0	238	0.0
2012	17	0.0	103	62.1	82	0.0	503	0.8	260	0.4
2013	108	1.8	79	0.0	93	0.0	222	0.0	120	30.8
2014	0	-	0	-	0	-	0	-	323	1.9

Trawl and purse seine surveys were conducted in the Strait of Georgia and Discovery Islands in May, June and July of 2010 to 2015 as described in Neville et al. (2013) and Neville et al. (2016). Juvenile Sockeye Salmon from these surveys were selected at random and screened for IHNV following the National Aquatic Animal Health Program (NAAHP) validated diagnostic methods which are based on the IHNV molecular diagnostic test reported by Purcell et al. (2013). The fish were identified to stock by the Molecular Genetics Laboratory at the Pacific Biological Station in Nanaimo, British Columbia, using procedures outlined in (Beacham et al., 2014a, b). Between 2010 and 2015, 2,564 Sockeye Salmon were tested and IHNV prevalence varied considerably between years and among stocks. Across all stocks, IHNV prevalence ranged from 0% (2012, 2013 and 2015) to max of 10.5% (2014) (Stewart Johnson, DFO, 3190 Hammond Bay Rd, Nanaimo, BC, Canada V9T 6N7, pers. comm.). As a potential wild source of virus to farmed fish populations, such annual fluctuations in IHNV prevalence may in part explain the stochastic occurrence of IHNV in farmed populations (Kyle Garver, DFO, 3190 Hammond Bay Rd, Nanaimo, BC, Canada V9T 6N7, pers. comm.).

Farmed Atlantic Salmon

In addition to the detection of IHNV in Pacific salmon in BC, three outbreaks of the virus have occurred in marine farmed Atlantic Salmon. Due to the devastating economic losses incurred as a result of the first two outbreaks, the detection, eradication, and epidemiological assessment of these occurrences have been well documented (St-Hilaire et al., 2002; Saksida, 2006). This section provides a brief summary of the outbreaks and associated impacts; the salmon aquaculture industry fish health management and biosecurity practices are detailed in Wade (2017).

In 1992, IHNV was reported for the first time in Atlantic Salmon from sea cages in BC (Armstrong et al., 1993). The first infection occurred in the summer months (onset in July) at a farm located in the Discovery Islands area (growing Area 1) just east of Campbell River. The epidemic lasted for five years ultimately encompassing 13 farms within 20 km of the index case (St-Hilaire et al., 2002). Mortality during this time ranged from 18-78% with the majority of infected sites involving fish under six months post-sea water entry (St-Hilaire et al., 2002). Some near harvest size fish also became infected in the first outbreak. A study of the 1992-1996 incursion by St-Hilaire et al. (2002), showed that IHN was detected in all months of the year but August, September and October. During this the period from 1992-1997, at least one farm was infected for 45 of the 66 months that the outbreak lasted (July 1992 to December 1997). For this period, a monthly farm level prevalence of IHNV was estimated at 12.5% of

active farms (average excluding the months with no cases), with a minimum number of infected farms of 1/29 (3.4%), a maximum of 4/6 (66.7%) and a median of 12.0%. During this initial outbreak, little disease management practices were in place and practices such as movement of fish, co-habiting naïve fish with survivors of the viral disease, and movement of equipment, undoubtedly contributed to the farm to farm spread and prolonged nature of the outbreak. In 1996, the industry undertook various measures such as site fallowing, equipment disinfection, and in some cases, the stocking of the less susceptible Chinook Salmon in an effort to control IHNV in farms (St-Hilaire et al., 2002). From 1998-2000, the Atlantic Salmon farming industry remained free of IHNV.

In August 2001, IHNV was diagnosed in farmed Atlantic Salmon in BC (Saksida, 2006). This epidemic lasted for three years ultimately affecting 36 farms operated by five different companies. This constituted over 50% of all the marine Atlantic Salmon farms in the province with a total of 12 million fish either dying or being culled due to IHNV (Saksida, 2006). The cumulative mortality averaged 58% (Saksida, 2006). The highest mortalities were from populations of fish which had been in seawater for less than one year. Similar to the previous outbreak, the virus was first detected in farms in the Discovery where monthly farm level prevalence ranged between 2/16 (12.5%) and 6/16 (37.5%), with an average of 21.6% and a median of 18.8%. Interestingly, this epidemic was not limited to one growing area as in the 1992-1996 epidemic, but rather involved farms situated along the North Coast as well as on the west coast of Vancouver Island and the Discovery Islands area (Saksida, 2006). The identification of two IHNV U genogroup variants between these geographically distinct areas revealed that the widespread epidemic was not a consequence of farm to farm spread but rather due to at least two separate introduction events. Nonetheless through epidemiological investigations, it was demonstrated that farming practices at the time of this epidemic, contributed significantly to the spread of IHNV between nearby farms (Saksida, 2006). Potential sources of viral spread included moving fish from one site to another, and the movement of personnel and equipment between sites and delivery vessels. In particular, the practice of pumping water through well boats during transport of fish through areas positive for IHNV or which were harvesting IHNV positive fish was identified as a source of spread of infection. Further temporal and spatial analyses of infections within this epidemic suggests that natural waterborne transmission may have played a role in the spread of IHNV among farms located in close proximity to each other (Saksida, 2006).

After the 2001-2003 epidemic, the industry remained free of IHNV for eight years during which several advancements in IHNV management occurred. In 2005, a highly efficacious DNA vaccine for IHNV (known as APEX-IHN®) was licensed for commercial use in Canada. Another significant advancement in the control of IHNV within the BC salmon farming industry occurred in 2010 with the implementation of an industry wide viral management plan. Details of the viral management plan are discussed in Wade (2017).

In 2012 there was an incursion of IHNV on Atlantic Salmon farms in BC as well as in Washington State (WA). The index case occurred in WA in April at a farm located near Bainbridge Island in Puget Sound while in BC, the first farm infected was in Clayoquot Sound off the west coast of Vancouver Island in May. Upon confirmation of IHNV in the BC farm, the Canadian Food Inspection Agency placed the infected farm under quarantine and the industry eradicated the fish from the site and disposed of the carcasses at a land based composting facility suitable for inactivation of IHNV. Subsequently, in the summer (July/August) two additional farm sites in BC, one located on the Sunshine Coast and one in Clayoquot Sound were confirmed IHNV positive. Due to the physical separation and/or temporal nature of these detections it is believed that each occurrence represents a separate introduction from a wild source rather than farm to farm spread. Genetic typing of the IHNV isolated from infected

Atlantic Salmon farms in BC revealed an identical sequence to an isolate found in out-migrating Sockeye Salmon in Washington State and BC rivers.

The average percent daily mortality for each infected farm in 2012 was calculated at the farm level starting ten days (Garver et al., 2013) prior to the date on which samples that first tested positive for IHNV were collected until complete farm depopulation. The average percent daily mortality were 0.21% for Dixon (April 29 to May 22, 2012: 23 days), 0.04% for Millar (July 15 to August 15, 2012: 31 days) and 0.18% for Culloden (July 21 to August 9, 2012: 19 days) farms during the outbreaks. From confirmation of detection of IHNV, a span of four days occurred until complete depopulation of the Dixon farm, 14 days at the Millar farm and five days at the Culloden farm.

For Dixon Bay, five whole fish were received May 16th, 2012 at 14:30. Brain and kidney were necropsied the same day and cells were inoculated May 17th. Overall, the molecular testing (qPCR and conventional PCR) and sequencing for genotypes took less than three days. Cell culture was essential and was conducted in parallel and proved the presence of infectious virus seven days post inoculation. The time to first observed cytopathic effect (CPE) in cell culture is highly variable and dependent upon viral load in the specimen. Dixon Bay was eradicated by May 22nd.

It is worthwhile noting that the farms diagnosed with IHNV were not vaccinated against IHNV; however, with the existing government and industry surveillance programs as well as the viral management plan, early detection and isolation was possible before farm to farm spread could occur.

The lack of virus spread among and between farms is best evident from a review of the diagnostic report (case 12-3214) provided by the Animal Health Centre with permission from Grieg Seafood for the farm on the Sunshine Coast. This summary of infection for the July/August outbreak was derived from the final diagnostic report. The farm consists of an array of six cages in a rectangular configuration, cages numbered sequentially from one to six. Fish were only present in cages 2, 4 and 6. Cages 2 and 6 were approximately 100 m apart. On July 30, 2012 samples were taken from cage 6 and tested positive for IHNV. Ten days later, sampling of cages 2 and 4 revealed a definitive positive detection in cage 4 while cage 2 had only a suspect diagnosis at the limit of detection for the assay. Furthermore, there was no evidence of IHNV-related mortality in cage 2 suggesting that IHNV spread was restricted within the infected farm and a farm-wide epizootic never occurred within the ten days from the initial virus detection. These results provide evidence to support a management option on farms where mortalities are closely monitored and any fish with suspicious clinical signs are tested. If IHNV positive fish are culled immediately based on clinical signs of disease and positive PCR results, it may be possible to remove infected fish before farm-wide infection occurs (Gary Marty, Animal Health Centre, Ministry of Agriculture and Lands, 1767 Angus Campbell Road, Abbotsford, BC, Canada V3G 2M3, pers. comm.*)*.

TRANSMISSION PARAMETERS OF IHNV

Within and among wild salmon populations, the primary mode of IHNV transmission is horizontal; however, transmission of the virus can also occur vertically when virus particles on the surface of the eggs infect the developing embryo, except if virus is destroyed by the application of disinfectants as performed in a hatchery setting.

Through the use of broodstock screening for IHNV, egg disinfection, and virus free water sources to rear fish, the BC Atlantic Salmon aquaculture industry has maintained an IHNV free status during the freshwater stage of their production cycle. However, (as noted in the above "Farmed Atlantic Salmon" section) IHNV has been occasionally detected in farmed Atlantic

Salmon populations that have been moved to marine cages for the grow out phase of the production cycle. In an effort to quantify the risk of virus dispersion from infected farms, investigations on the infection dynamics of IHNV in Atlantic Salmon were conducted. Specifically, the amount of IHNV shed from an infected Atlantic Salmon was estimated; the stability of the shed virus in seawater was examined and; the virus dose required to initiate IHN disease was determined. These studies are summarized below.

IHNV shedding in Atlantic Salmon

Once infected with IHNV, Atlantic Salmon shed increasingly higher quantities of virus with the progression of IHN disease. Virus shedding peaks one to two days prior to death and can reach rates of upwards of 3.2×10^7 pfu/fish/hr. Shed virus is only detected among those individuals that develop disease, suggesting that Atlantic Salmon that appear asymptomatic and remain free of IHN disease are not a significant source of virus (Garver et al., 2013). Atlantic Salmon that did develop IHN disease after being immersion exposed to a lethal dose of virus expired 18 to 23 days later. During this acute disease period, shed virus was observed in the water of the tanks with the diseased fish for a period of 9 to 13 consecutive days (Garver et al., 2013). However, it is worthwhile noting that both onset and progression of IHN disease is highly dependent on the exposure dose and therefore is likely to be highly variable between outbreaks (Garver et al., 2013).

Virus decay

The environmental stability of IHNV is affected by water salinity, temperature, organic load, microbial content, and exposure to ultraviolet light. IHNV can survive up to one month in freshwater at “cooler” temperatures (OIE, 2012). However, in naturally occurring river and ocean waters that are not sterilized, high concentrations of IHNV are inactivated within days (Garver et al., 2013). This inactivation rate can be further accelerated such that high concentrations of IHNV are inactivated within minutes if virus contaminated water is subjected to sunlight (Garver et al., 2013). Consequently higher concentrations of waterborne IHNV are likely to accumulate during the winter months when water temperatures and sunlight are significantly less than that observed during spring and summer months (Garver et al., 2013).

Minimum lethal dose

As noted in the “Host range and susceptibility” section above, Atlantic Salmon have been shown to be highly susceptible to IHNV with disease being easily transmitted through cohabitation with IHNV infected fish (Traxler et al., 1991, 1993) or simply through exposure to virus contaminated fresh or seawater (Garver et al., 2013; Foreman et al., 2015a). Furthermore Atlantic Salmon are susceptible to virus isolates representing all genogroups of IHNV (Kurath et al., 2016) that may be a possible consequence of a lack of historic exposure as first postulated by Traxler et al. (1993). Through waterborne exposure, IHN disease and mortality was transmitted after fish were exposed to as little as 10 pfu/mL for one hour (Garver et al., 2013). In Sockeye Salmon, it was demonstrated through two separate experiments that the minimum lethal dose required to establish IHN disease and mortality in smolts ranged from 10 to 100 fold higher than Atlantic Salmon of similar life stage (Long et al., 2017). As noted in the “Host range and susceptibility” section of this document, the differential susceptibility between Atlantic Salmon and Sockeye Salmon is even more pronounced at the adult life stage when Sockeye Salmon are refractory to IHN disease while Atlantic Salmon remain susceptible to the disease.

Modeling waterborne dispersion of IHNV among BC salmon farms

Water movement in aquatic environments can facilitate pathogen transport over long distances. The Discovery Islands, a major farmed salmon producing region of BC, is characterized by a complex network of narrow channels and deep fjords that contain some of the strongest tidal currents in the world. Coupling a hydrodynamic ocean circulation model for this region with the virological transmission estimates described above has been performed to provide accurate geospatial predictions of risk for IHNV waterborne transmission in this area (Foreman et al., 2015a). Simulations of numerical particles released and inactivated at rates in accordance with laboratory derived estimates and dispersed by model oceanic flows demonstrate that Atlantic Salmon farms undergoing acute IHN disease can transmit an infectious dose to neighboring naïve Atlantic Salmon farms (Foreman et al., 2015a). The simulated release of virus from an infected farm is predominately driven by excessive virus shedding from a diseased population. Consequently, fish health practices such as vaccination which safeguard farm populations from IHN disease, substantially reduces risk of viral shedding and transmission (Foreman et al., 2015a).

Vaccination

Laboratory studies evaluating the APEX-IHN® vaccine efficacy in Atlantic Salmon demonstrated that fish 17 months after vaccination still maintained a protective advantage such that mortality among virus exposed fish reached approximately 76% in the control group while only reaching approximately 27% in the vaccinated group (Salonius et al., 2007). Further, recent vaccine efficacy trials conducted at the Pacific Biological Station demonstrated that Atlantic Salmon post smolts exposed to a lethal dose of IHNV five months post APEX-IHN® vaccination resulted in significant protection against IHNV with mortality occurring in only 2.7% (4 mortalities out of 150 fish) of the population as opposed to 96.7% (121 of 125) in unvaccinated controls. Additionally, vaccination in IHNV infected Atlantic Salmon completely abolished disease transmission to cohabitating naïve Sockeye Salmon and reduced virus spread among cohabitating naïve Atlantic Salmon (Long et al., 2017). Taken together, these results demonstrate that vaccination greatly reduces the infectious load and potential for IHNV transmission.

IHN DISEASE IMPACT ON SOCKEYE SALMON POPULATIONS

The impact of infectious diseases on wild fish populations is often difficult to quantify as the fish's environment and predation of debilitated hosts hinders the detection of mortality events. However, in the case of IHNV, several epizootics have been observed under natural conditions that have allowed for a direct measure of impact to the population through mortalities. A review of these episodic events has demonstrated that IHNV can have a considerable influence on the numbers of fish produced from a system, particularly during the fry and juvenile freshwater life stages.

The first confirmed epizootic of IHN disease occurred in Sockeye Salmon in Chilko Lake, BC. The outbreak occurred in the spring of 1973 and resulted in an estimated reduction of 23.7 million fry migrating into the lake. The effect of IHNV on this population was predominately observed in the egg to fry stage resulting in 3.8% survival, the lowest ever recorded in the period 1949 to 1973. However, with a normal fry to smolt survival of 57.1%, IHN had no apparent effect on the fry during the lacustrine stage of the life cycle (Williams and Amend, 1976).

The egg to fry stage also proved to be the most impacted in an IHNV epizootic in Sockeye Salmon in Weaver Creek spawning channel in 1987 (Traxler and Rankin, 1989). The prevalence of IHN virus in Sockeye Salmon fry was highest during the early part of the fry

migration from the spawning channel and resulted in an estimated loss of 8.3 million of the 16.8 million fry migrating from the channel. Traxler and Rankin (1989) held infected fry in a tank supplied with flow through creek water for several days. By doing so, they demonstrated that losses not only occurred within the channel but also several days after fry would have migrated from the channel.

Evidence of impacts to older life stages is uncommon, although IHNV epizootics in 1.5 year old Sockeye Salmon have been documented in Alaska. In two consecutive years, 1980 and 1981, mortality occurred in a portion of outmigrating Sockeye Salmon smolts from Hidden Lake (Burke and Grischkowsky, 1984). Moribund and dead fish were first observed in the last third of the migration period with mortality rates reaching upwards of 8% in the remaining 30% of the out-migrants.

No natural IHNV mortality events have been observed in the marine rearing and adult life stages of Sockeye Salmon. Nevertheless, laboratory exposure of twenty five Sockeye Salmon post smolts (one week acclimation in seawater) to IHNV through cohabitation with infected Atlantic Salmon undergoing acute IHN disease, resulted in 4% mortality (1 out of 25) after 12 weeks, suggesting that marine life stages are much less susceptible to IHN disease in comparison to early rearing freshwater stages (Traxler et al., 1993).

SURVEILLANCE AND DETECTION

Because IHNV is a reportable disease listed under the *Health of Animals Act*, the Canadian Food Inspection Agency (CFIA) is responsible for official surveillance and response to IHN disease when required. This does not preclude other organizations from performing tests for IHNV; however, if the virus has been detected or IHN disease is suspected then there is a legal obligation to notify the CFIA.

CFIA and DFO have an agreement that the testing of fish for the purposes of the National Aquatic Animal Health Program will occur at one of the three DFO references laboratories under the National Aquatic Animal Health Laboratory System (NAAHLS). The reference laboratory for IHNV is the Pacific Biological Station; however, testing can be done at both the Gulf Fisheries Centre and Freshwater Institute.

The testing requirements for IHNV in apparently healthy populations include primary testing using RT-qPCR assay targeting the IHNV nucleocapsid (N) gene. The IHNV N gene RT-qPCR had 100% diagnostic specificity (DSp) and sensitivity (DSe) and a higher estimated diagnostic odds ratio (DOR) than virus culture or conventional PCR. The RT-qPCR assay was highly repeatable within a laboratory and highly reproducible between laboratories and was found suitable for use in a diagnostic setting (Purcell et al., 2013). If the virus is detected via RT-qPCR, the extract or original tissue will be screened using both RT-PCR and virus isolation for confirmatory testing. Samples are screened for all genotypes of IHNV. Samples of whole animals or tissues may be submitted to the laboratory for testing depending on the size of the animal and situation. The target organs for RT-qPCR are anterior kidney and or other suitable tissues and/or fluids. For virus isolation, whole fish are utilized if the fish are ≤4 cm in length. If they are between 4 and 6 cm in length, viscera including kidney is used. For fish ≥6 cm, anterior kidney is sampled independently or a tissue pool, prepared from a standardized mixture of spleen, heart, liver or encephalon, is analyzed. In spawning adults, ovarian fluid or milt are tested.

CFIA surveillance

Beginning in 2012, the Aquatic Surveillance and Epidemiology section of the CFIA undertook a multi-year program to survey wild and enhanced anadromous salmonids in British Columbia for

infectious salmon anaemia virus (ISAV), infectious pancreatic necrosis virus (IPNV) and IHNV. Over the course of the program 1,272 fish were tested for IHNV including 94 Chinook, 342 pink, 332 chum, 191 coho and 313 Sockeye Salmon (Table 7). All fish tested negative for IHNV. Samples were collected directly from the wild, at processing plants and at enhancement hatcheries. Life stages tested included juveniles in freshwater and adults (Table 7).

Table 7. Summary of the numbers of each species of Pacific salmon tested for IHNV by CFIA as a part of the 2012-2014 anadromous salmonid surveillance program (SW=saltwater) (Data provided by CFIA).

Common name	Scientific name	# Animals tested for IHNV (2012-2014)		
		Juveniles (SW)	Adult	Total
Chinook Salmon	*O. tshawytscha*	85	9	94
Coho Salmon	*O. kisutch*	126	65	191
Pink Salmon	*O. gorbuscha*	50	292	342
Sockeye Salmon	*O. nerka*	61	252	313
Chum Salmon	*O. keta*	151	181	332
Total		**473**	**799**	**1272**

CFIA epidemiological assessment

In 2014, the CFIA completed an epidemiological assessment of farmed salmon with respect to ISAV (pathogenic and non-pathogenic), IPNV, and IHNV for the period of 2006-2011 inclusive. The purpose of the assessment was to provide the scientific basis for surveillance recommendations in support of the substantiation of health status of farmed Atlantic and Pacific salmon species in both fresh and marine culture systems (Canadian Food Inspection Agency, 2014).

This evaluation was conducted in collaboration with the BC Salmon Farmers Association (BCSFA), Mainstream Canada, Creative Salmon, Grieg Seafood, Marine Harvest Canada, the BC Ministry of Agriculture (BCMA), the Department of Fisheries and Oceans Canada (DFO) Aquaculture Management Division in BC, the BC Centre for Aquatic Health Sciences (BC CAHS), and DFO's National Aquatic Animal Health Laboratory System (NAAHLS) (Canadian Food Inspection Agency, 2014).

Included in the analysis was the evaluation of existing government and industry surveillance programs which existed between 2006 and 2011. For this period, over 550 farm audits were carried out by the government. This equated to 3,183 diagnostic tests for IHNV. The industry completed 31,086 tests for IHNV using a combination of molecular techniques, tissue culture, and histopathology testing (Canadian Food Inspection Agency, 2014). A scenario tree model was used to assess the sensitivity of the surveillance activities conducted as a part of the government Fish Health Audit and Surveillance Program as well as industry fish health monitoring programs. It was determined that additional surveillance beyond what is currently in place was not necessary to address the requirement for the early detection of clinical disease in farmed Atlantic Salmon in BC. During the time of this study, some farms began to use the IHNV vaccine (Canadian Food Inspection Agency, 2014). The impact of vaccination on surveillance was not included in the CFIA's epidemiological assessment.

Provincial/ DFO fish health audit and surveillance activities

Marine aquaculture

In 2002, the British Columbia Ministry of Agriculture (BCMA) began a Fish Health Audit and Surveillance Program. On December 18, 2010, the Department of Fisheries and Oceans

assumed management and regulatory responsibility for the aquaculture industry in British Columbia including the Fish Health Audit and Surveillance Program with some refinement from the original BCMA plan. Under this Program, ongoing testing occurs for many pathogens including IHNV. This program is discussed at length in Wade (2017).

Enhancement hatcheries

In British Columbia, Sockeye Salmon are enhanced through both hatcheries and spawning channels. Although some small community run facilities do exist, the Department of Fisheries and Oceans operates the major facilities, which produce millions of Sockeye Salmon annually for release into the natural environment (Table 8).

*Table 8. Production of Sockeye Salmon from DFO enhancement facilities. Data provided by DFO Salmon Enhancement Program (*indicates production of Fraser River stocks).*

Facility	Production years	Highest recorded annual production	Lowest recorded annual production	Average production 2003-2015
Fulton River spawning channel	1967-present	159,600,000	16,000,000	80,373,992
Gates Creek spawning channel	1969-present	19,701,066	21,000	3.668,940
Horsefly River spawning channel*	1990-present	29,400,000	85,000	10,452,568
Inch sockeye satellite (Cultus Lake)*	2004-present	1,199,253	48,378	439,040
Inch sockeye satellite (Pitt River upper)*	2004-present	2,467,732	1,528,310	2,022,689
Nadina spawning channel*	1974-present	19,162,000	373,000	6,195,303
Ouillet Creek hatchery (Sakinaw Lake)	1986-present	309,841	2,784	139,197
Pinkut Creek hatchery	1969-present	84,828,148	4,930,000	46,085,729
Rosewall Creek hatchery (Cultus Lake)*	2000-present	998,011	402	483,841
Rosewall Creek hatchery (Sakinaw Lake)	2006-present	953,285	39,000	416,451
Snootli Creek hatchery	2001-present	912,250	49,859	417,391
Weaver Creek Spawning Channel*	1966-present	56,054,000	434,000	30,452,115
Total	**-**	**375,585,586**	**23,511,733**	**181,147,256**

As a part of this enhancement program, a sub-sample of females used for egg production are tested for IHNV from both Rosewall Creek and Snootli Creek hatcheries. Testing of females from spawning channels is not a part of the enhancement program. However, DFO researchers have been testing returning adults from Weaver Creek, Nadina, Pinkut and Fulton spawning channels from as early as 1984 (Table 8).

Although there is concern over the spread of IHNV though egg-associated infection from positive females, if fertilized eggs are disinfected appropriately with iodine, infection and subsequent disease can be mitigated. This is common practice in hatchery facilities. Therefore it is not necessary to destroy eggs derived from IHNV positive females. Hatchery fry can therefore only become IHNV positive through contaminated water or breaches in biosecurity. Fry are not tested for IHNV before they are released from the hatchery; however, it is a requirement that unusually high mortality events be reported to the veterinarian responsible for DFO's enhancement facilities as these events occur. Disease status of animals in spawning channels is not monitored.

INACTIVATION METHODS

The chemical and physical inactivation of IHNV is discussed as it is pertinent to the farmed fish health management practices (Wade, 2017).

IHNV contaminated equipment, clothing, or vessels are inactivated by common disinfectants and drying (desiccation). IHNV can survive outside the host in freshwater for extended periods, particularly in the presence of organic material (OIE, 2012). In freshwater salmonid hatcheries, the use of UV or ozone on incoming water has proven to be effective in the management of IHNV (Winton, 1991; Foott et al., 2006; OIE, 2012). Pietsch et al. (1977) found that IHNV infectivity was significantly reduced at pH 5 and 9. Generally studies have shown that IHNV can survive for weeks at 15^{o}C but at 32^{o}C it becomes inactivated within hours. Watson et al. (1954) showed that a single freeze-thaw cycle reduced virus titres from 10^6 to 10^2, and suggested that proper freezing of fish products may be effective for pathogen inactivation. However, this is in direct contrast to LaPatra et al. (2001b) who demonstrated no significant reductions in concentrations of IHNV in brain and kidney tissue in injected adult Rainbow Trout after a freeze-thaw cycle at -20^{o}C for 7 or 14 days.

Chemical or physical disinfection of the IHN virus has been evaluated. Efficacy testing has been reported for heat, UV-C, acid, chlorine, ozone, iodophores, and Virkon Aquatic ® (Table 9).

Table 9: Evidence for the chemical or physical inactivation of IHNV (Karreman and Wade, 2011[1]). MEM= type of growth medium.

Method	Dose	Comment	Contact time	Reduction	Source
Heat	28^{o}C	MEM-1	330 min	90 %	Gosting and Gould (1981)
	32^{o}C	MEM-1	90 min	99.9 %	
	38^{o}C	MEM-1	15 min	99.9 %	
	35^{o}C	MEM-0	5 hr	Inactivated	Whipple and Rohovec (1994)
	40^{o}C	MEM-0	20 min	Inactivated	
	45^{o}C	MEM-0	10 min	Inactivated	
	50^{o}C	MEM-0	90 sec	Inactivated	
	55^{o}C	MEM-0	30 sec	Inactivated	
UV-C	10-30 J/m ^{2}	Strain dependent	-	99 %	Yoshimizu et al. (1986)
	20 J/m ^{2}	-	-	99.9 %	Sako and Sorimachi (1985)
Acid	pH 4 citric phosphate buffer	22^{o}C	7 hr	Incomplete	Whipple and Rohovec (1994)
	pH 3.8-4.3 fish silage	22^{o}C	30 sec	Inactivated	
Chlorine	0.1 mg/L	10^{o}C, aqua dest.	30 sec	Inactivated	Wedemeyer et al. (1978)
	0.5 mg/L	10^{o}C, soft lake water	5 min	Inactivated	
	0.5 mg/L	10^{o}C, hard lake water	10 min	Inactivated	
	1 mg/L	10^{o}C, hard lake water	30 sec	Inactivated	
	10 ppm	-	30 min	Inactivated	Amend and Pietsch (1972)
Formalin	0.2 %	-	60 min	Incomplete	Amend and Pietsch (1972)
Ozone	0.01 mg/L	10^{o}C, aqua dest	30 sec	Inactivated	Wedemeyer et al. (1978)
	70 mg/h/L	10^{o}C, soft and hard lake water	10 min	Inactivated	

[1] Karreman, G. A. and Wade, J. 2011. Biosecurity Procedures for Harvesting, Transporting and Processing of Aquaculture Fish in Nova Scotia. Nova Scotia Department of Fisheries and Aquaculture. Unpublished report. 196 p.

Method	Dose	Comment	Contact time	Reduction	Source
Iodophor	25 ppm	pH 7.0	15 sec	Inactivated	Amend and Pietsch (1972)
	12 ppm	pH 7.0	30 sec	Inactivated	
	0.1 mg/L	In deionized distilled water or hatchery water	7.5 sec	99.9%	Batts et al. (1991)
Virkon Aquatic	1:1000 at 20^{o}C	Cell culture fluid (rhabdovirus Strain 19)	15 min		Western Chemical Inc.
	1:1000 at 20^{o}C	Cell culture fluid (rhabdovirus Ban Pako Strain)	15 min		

SUMMARY

THE VIRUS

Laboratory studies have demonstrated that Atlantic Salmon suffering acute IHN disease shed virus into surrounding water with increasing amounts of IHN virus released at the terminal stages of the disease (i.e., one to two days prior to death). Conversely, Atlantic Salmon that have been exposed to IHNV but which do not develop disease do not shed detectable levels of virus into the water. Consequently, treatment methods and/or viral management practices aimed at preventing IHN disease are of upmost importance towards abating virus release from IHNV infected farms.

In 2005, a plasmid-DNA based vaccine (APEX-IHN®) was licensed for commercial use in Canada for the prevention of IHN disease. The vaccine is extremely efficacious with upwards of 100% protection. In laboratory studies, APEX-IHN® prevented an outbreak of IHN disease in a population of Atlantic Salmon exposed to a lethal dose of IHNV. Furthermore, the vaccinated fish were incapable of transmitting IHN disease to cohabitating Sockeye Salmon. Since its licensure, over 60 million doses of APEX-IHN® have been administered to Atlantic Salmon in BC and to date there has been no detection of IHNV in an APEX-IHN® vaccinated farmed Atlantic Salmon.

Laboratory studies exposing fish to IHNV via immersion in virus contaminated water or through cohabitation with IHNV infected fish, have demonstrated that IHNV is transmitted and spread through waterborne exposure. IHNV dispersion from infected net-pen Atlantic Salmon farms is dependent upon the number of diseased fish in the farm population (virus shedding fish), the decay rate of IHNV, and the water movement (currents) in the area of the infected farm.

The IHN virus is not stable outside of the host. The virus is quickly inactivated by sunlight and the microbial community present in seawater. The longevity of IHNV is increased with decreasing sunlight and colder water; nevertheless IHNV has an abbreviated lifespan when shed into seawater.

Model simulations of IHNV dispersion from outbreaks in farmed Atlantic Salmon performed using numerical particle releases in accordance with laboratory derived virus shedding and inactivation rates, demonstrate that in the absence of disease management procedures, neighbouring naïve farms can become exposed to IHNV via waterborne transport from an IHN diseased farm, with the greater ability of spreading an infectious dose of IHNV occurring during months with low sunlight hours. However, under current fish health management practices, which would result in rapid detection and depopulation of diseased fish within 14 days of confirmation of disease, the maximum IHNV concentration in net pens was determined to be significantly lower than the virus dose found to initiate disease in laboratory exposed juvenile Sockeye Salmon.

PRESENCE OF IHNV

Endemism

IHNV occurs naturally in the waters of western North America and can cause acute disease (IHN) in nearly all salmonid species given appropriate viral, host, and environmental conditions. In British Columbia, IHNV is commonly detected in Sockeye Salmon, occasionally in Atlantic Salmon, and has rarely been identified in Rainbow Trout, steelhead trout, Chum Salmon, and Chinook Salmon.

IHNV is endemic in the freshwater river systems of BC, and all recorded natural IHNV associated mortality in Pacific salmon species has occurred at the fry and juvenile life stage while rearing in the freshwater environ. Fry surviving an infectious exposure of IHNV in the freshwater environments can mount a protective immune response to subsequent IHNV exposure. Due to the endemic nature of IHNV in freshwater environs and its prevalence in BC Sockeye Salmon stocks, it is uncommon that Sockeye Salmon smolts would remain naïve to IHNV prior to initiating their ocean migration.

With increasing size and lifestage, Pacific salmon species become refractory to IHN disease. To date, no IHN disease has been reported in adult Pacific salmon despite testing positive for IHNV.

On Atlantic Salmon farms

IHNV has caused IHN disease in Atlantic Salmon farms in British Columbia. Atlantic Salmon are highly susceptible to IHNV and have shown high mortality at the post smolt stage within the first year of seawater entry. Additionally, Atlantic Salmon show little protective advantage to IHN disease with increasing age and size as observed in Pacific salmon species.

Epidemiological investigations of IHNV in Atlantic Salmon farms suggest viral releases occurred as evident by farm to farm spread of disease during historical outbreaks when disease management practices were not implemented industry-wide. In 2010, the BC Atlantic Salmon aquaculture industry universally adopted and implemented a viral management plan detailing practices and procedures employed to safeguard against the introduction and spread of IHN disease in Atlantic Salmon farms. Consequently, during the 2012 outbreak, under more modern management practices which include movement controls and eradication, no farm to farm spread occurred.

In BC there has never been a detection of IHNV in freshwater phase farmed Atlantic Salmon. IHNV in farmed Atlantic Salmon has only occurred in marine net-pen reared animals indicating that farmed fish are exposed to IHNV in the natural seawater environment. The occurrence of IHNV in BC Atlantic Salmon farms is sporadic, index cases occurring in 2001, 2002 and 2012. The spill-over of virus from a wild marine reservoir typically occurred in the spring (April/May) or summer (July/August) corresponding with the outmigration of Sockeye Salmon smolts and returning spawning adults, respectively.

Surveillance of Sockeye Salmon smolts in the marine environment while migrating through the Strait of Georgia has revealed the presence of IHNV prior to entering areas of net-pen Atlantic Salmon farming, suggesting that Sockeye Salmon smolts are a potential source of IHNV to farmed Atlantic Salmon.

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