The Cohen Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River

July 2011

TECHNICAL REPORT 1A

Assessment of the potential effects of diseases present in salmonid enhancement facilities on Fraser River sockeye salmon

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Preface

Fraser River sockeye salmon are vitally important for Canadians. Aboriginal and non-Aboriginal communities depend on sockeye for their food, social, and ceremonial purposes; recreational pursuits; and livelihood needs. They are key components of freshwater and marine aquatic ecosystems. Events over the past century have shown that the Fraser sockeye resource is fragile and vulnerable to human impacts such as rock slides, industrial activities, climatic change, fisheries policies and fishing. Fraser sockeye are also subject to natural environmental variations and population cycles that strongly influence survival and production.

In 2009, the decline of sockeye salmon stocks in the Fraser River in British Columbia led to the closure of the fishery for the third consecutive year, despite favourable pre-season estimates of the number of sockeye salmon expected to return to the river. The 2009 return marked a steady decline that could be traced back two decades. In November 2009, the Governor General in Council appointed Justice Bruce Cohen as a Commissioner under Part I of the *Inquiries Act* to investigate this decline of sockeye salmon in the Fraser River. Although the two-decade decline in Fraser sockeye stocks has been steady and profound, in 2010 Fraser sockeye experienced an extraordinary rebound, demonstrating their capacity to produce at historic levels. The extreme year-to-year variability in Fraser sockeye returns bears directly on the scientific work of the Commission.

The scientific research work of the inquiry will inform the Commissioner of the role of relevant fisheries and ecosystem factors in the Fraser sockeye decline. Twelve scientific projects were undertaken, including:

Project	
1	Diseases and parasites
2	Effects of contaminants on Fraser River sockeye salmon
3	Fraser River freshwater ecology and status of sockeye Conservation Units
4	Marine ecology
5	Impacts of salmon farms on Fraser River sockeye salmon
6	Data synthesis and cumulative impact analysis
7	Fraser River sockeye fisheries harvesting and fisheries management
8	Effects of predators on Fraser River sockeye salmon
9	Effects of climate change on Fraser River sockeye salmon
10	Fraser River sockeye production dynamics
11	Fraser River sockeye salmon – status of DFO science and management
12	Sockeye habitat analysis in the Lower Fraser River and the Strait of Georgia

Experts were engaged to undertake the projects and to analyse the contribution of their topic area to the decline in Fraser sockeye production. The researchers' draft reports were peer-reviewed and were finalized in early 2011. Reviewer comments are appended to the present report, one of the reports in the Cohen Commission Technical Report Series.

Executive Summary

The objectives of this report were; (1) to review disease data and reports from salmon enhancement facilities operated under the authority of Fisheries and Oceans Canada (DFO) and the Freshwater Fisheries Society of British Columbia (FFSBC) and evaluate the potential for a qualitative and/or quantitative assessment of the potential effect of diseases present in enhancement facilities on the production of Fraser River sockeye salmon (*Oncorhynchus nerka*) and, (2) if possible evaluate the disease risks posed by the operation of salmonid enhancement facilities on the production of Fraser River sockeye salmon.

The role of enhancement hatcheries in sustaining wild salmon populations is controversial. Salmonid enhancement is intended to improve the freshwater productivity of native salmonids. Concerns about negative effects from interbreeding of enhanced and wild salmon, ecological competition, and the impacts of mixed fisheries have been the subject of other reviews and remain unresolved. This report only considers the potential infectious disease risks of salmonid enhancement facilities in the Fraser River watershed and Strait of Georgia for approximately the past decade.

Two methods were used to assess the burden of evidence available for risk assessment and to attempt to evaluate the risks. First, a scoping literature review sought direct and indirect evidence of a causal relationship between salmonid enhancement related infectious diseases and Fraser River sockeye salmon production. Second, data provided by the Cohen Commission including, salmonid enhancement disease diagnostic data; hatchery-level health records and; production data were examined using a risk assessment framework.

The disease impacts of salmon enhancement facilities on Fraser River sockeye salmon are largely unexplored in the literature. The published literature failed to provide sufficient direct or indirect evidence to fulfill standard criteria for causation. Infectious diseases and disease causing microorganisms have been reported in the literature in both Fraser River sockeye salmon and other species of enhanced salmonids in British Columbia. These pathogens are capable of causing clinical and sub-clinical impacts on individual fish but the effects on population productivity remain speculative.

The literature was unable to provide sufficient information to determine the likelihood of salmonid enhancement-associated diseases impacting Fraser River sockeye salmon, the magnitude of the hypothetical impacts, or the ability of enhancement facilities to prevent or mitigate the risks. A small number of historic cases have associated the presence of pathogens in Fraser River sockeye salmon with acute and sometimes large scale mortality, but the hypothesized association between crowding at spawning channels and increased risk of disease have not been definitively proven.

The goal of determining the impact of a specific disease on wild fish productivity is largely unachievable due to the high variability in exposure settings, environmental conditions and biological responses; high level of uncertainty due to infrequent or inaccurate measurements; and large number of unknown interacting factors. Past reviews of the impacts of enhancement hatcheries have suggested a negative effect on wild salmon, but supporting evidence is lacking.

Limitations in research designs and the challenges of studying fish disease under natural settings are significant obstacles to understanding the impacts of disease and to establishing with sufficient precision that free-ranging fish are exposed to pathogens of enhancement facility origin. There is biological and epidemiological plausibility that diseases, under certain environmental conditions, could affect wild fish population dynamics and there is experimental evidence that certain pathogens can cause death, disease and impaired physiological function in individual fish. However, there is insufficient information and understanding in the published literature to establish the proportional contribution of infectious diseases alone or in combinations with other host and environmental stressors to Fraser River sockeye salmon production.

We could not find an evidence-based, non-zero standard to define an acceptable frequency or amount of transfer of pathogens from enhanced fish to wild fish that could be used in a risk assessment.

We know of no legal fish health standard that establishes an acceptable level of fish pathogen risk for enhancement operations except for legislation dealing with the exclusion of foreign or exotic disease from Canada. A single standard for acceptable exposure cannot currently be defined as the capacity for individuals and populations to cope with a disease is context specific and would be affected by things such as the pathogen, host species, life stage, habitat quality, water temperature and many other factors.

A health standard of no infectious or parasitic microorganisms or diseases in Fraser River sockeye salmon is unattainable because; infection and disease are normal in wild fish populations and a variety of infectious agents are ubiquitous in aquatic environments or common in cultivated or wild fishes.

Disease data from enhanced salmon in British Columbia did not allow for the construction of a complete hazards list for use in a risk assessment or for estimating the frequency and abundance of infection in enhanced fish populations. The nature of the diagnostic systems restricted our knowledge to the more common infections that are capable of causing overt clinical signs in a sub-set of the population as well as to a small number of pathogens in returning broodstock. The data did reveal that a variety of pathogenic hazards exist in enhanced salmon in British

Columbia; none of which were unexpected or exclusive to enhanced salmonids. Enhanced salmon in the province do harbour viruses, bacteria and parasites capable of causing severe clinical disease in infected fish under experimental or culture conditions. We were able to document cases where fish with known or suspected infections were released from salmonid enhancement operations into fish bearing waters. In no case were we provided evidence that post-release monitoring of surrounding wild fish was undertaken. There was no evidence found to assess if these releases did or did not result in exposure or impacts on other fish.

For a risk to exist, an individual or population must be exposed to a hazard. Generally, there are three variables that affect the probability of exposure to an infectious hazard; (1) the geographic distribution of the escaped pathogen; (2) the abundance of the pathogen in the receiving environment and; (3) the frequency with which the fish are involved in an exposure that results in transmission of the pathogen. As there are no data for these 3 variables, exposure assessment was not possible. Fraser River sockeye salmon reared in enhancement hatcheries or spawning channels have the most plausible route of exposure to diseases present in hatcheries or spawning channels. Exposure of Fraser River sockeye salmon outside of enhancement facilities to infectious enhanced salmonids has not been monitored. Biologically plausible routes of exposure exist, but none have been measured.

Federal and provincial salmonid enhancement programs do many things to reduce the risk of disease to wild fish by managing disease abundance in their facilities. Diagnostic services provided to salmonid enhancement facilities allow for identification and treatment of infections; movement restrictions limit the translocation of pathogens; and broodstock screening allows for the reduction of certain vertically transmitted diseases. The operating procedures for risk reduction at the enhancement hatcheries and spawning channels focus on two elements; reducing the prevalence of disease within groups of fish to be released from salmonid enhancement operations; and pre-release assessments of groups of fish with previous disease or infection histories. There is no routine assessment of the infection status of groups that are either not showing clinical signs and/or are not progeny of fish with vertically transmitted infections or at risk of having known vertically transmitted infections. A population-wide fish disease surveillance program does not exist.

All major DFO and FFSBC hatcheries have Fish Health Management Plans that are intended to support the goal of not releasing fish with known infections. The Plans have not been audited. There are inadequate resources to allow fish health professionals to visit enhancement facilities to help adapt Fish Health Management Plans to local conditions, audit their practices and develop ongoing disease prevention programs. The Plans vary in detail and in their adaptation to local conditions. There is little opportunity to apply Fish Health Management Plans to spawning channels and it did not appear that the Community Economic Development Program or Public

Involvement Project hatcheries have comprehensive fish health management plans. The amount of risk reduction to Fraser River sockeye salmon realized by these efforts has not been investigated but it is reasonable to assume that reduction of infection in salmonid enhancement facilities will reduce the level of exposure for wild salmonids from this potential source.

The current system for reporting and recording fish health in salmonid enhancement facilities or for documenting the suitability of fish for release, lack consistency, quality and accessibility thus limiting external review and public assurance.

A risk assessment could currently only conclude that the risk of transfer of infectious agents is biologically and epidemiologically plausible. There is a suite of pathogenic hazards present in fish in enhancement facilities and evidence that pathogens have viable means to escape spawning channels and hatcheries via fish or water releases; thus entering fish bearing waters potentially occupied by Fraser River sockeye salmon. The probability and consequence of an exposure to released infectious agents on Fraser River sockeye salmon cannot be specified using the current scope of scientific knowledge.

We could not determine if diseases present in salmon enhancement facilities (hatcheries or spawning channels) present potential for serious or irreversible harms to Fraser River sockeye salmon. Limitations in scientific understanding, lack of ongoing surveillance of wild and cultured fishes, and deficits in data provided to us were the primary reasons for our inability to make specific cause-effect conclusions and to qualitatively or quantitatively assess risk.

We provide management and research recommendations that may improve the effectiveness of fish health programs in risk management as well as increase oversight of fish diseases to provide public assurances that undue disease risks are not arising from salmonid enhancement facilities. Management recommendations fall into 3 themes: (1) shifting the emphasis and organization of fish programs from diagnostic services for disease treatment to comprehensive health management for health promotion and disease prevention; (2) promoting a systems perspective that allows for fish disease and population data to be integrated and (3) improving auditing and oversight. Research recommendations are intended to support these management objectives by developing evidence for strategic management decisions and to create new understandings to better characterize and monitor disease interactions between cultured and free-ranging fish.

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Introduction

Introduction to BC salmonid enhancement

Salmon hatcheries were initially developed to mitigate declines in wild salmon populations and fisheries catches (Heard et al., 2007). The British Columbia (BC) Salmonid Enhancement Program (SEP) was established in 1977 to restore salmon and anadromous trout to their historic levels of abundance (Anonymous, 2009) to "address conservation concerns and provide fishing opportunities" (MacKinlay et al., 2004). The main goals of the SEP are to (MacKinlay et al., 2004):

1. Restore depleted stocks to higher levels of abundance by increasing freshwater survival directly using hatcheries and spawning channels or indirectly through habitat improvement;
2. Mitigate for major habitat losses including those from dams and urbanization;
3. Provide for harvest opportunities especially for terminal or selective fisheries;
4. Re-establish extirpated stocks by introduction of fish from similar stocks into abandoned and presumably under-utilized habitat

Various techniques have been used for salmonid enhancement (Table 1), all of which depend on "collaborations between DFO, First Nations, non-profit organizations, community groups and various other stakeholders" (Anonymous, 2009). This report will focus on two techniques, namely, hatcheries and spawning channels as they provide a means to control or directly affect the health of enhanced fish and have some capacity to influence, measure or manage fish diseases.

In 2009, the SEP was comprised of 23 major fish enhancement facilities, 22 of which were owned and operated by Fisheries and Oceans Canada (DFO), and one jointly funded by SEP and Aboriginal Fisheries Strategy (Anonymous, 2009).

The Community Involvement Program is a component of the SEP. It includes the Community Economic Development Program (CEDP) involving 21 hatcheries and the Public Involvement Program (PIP) which supports 350 projects composed mostly of education, outreach, stream-keepers activities, and the operation of small volunteer run hatcheries (Anonymous, 2009). Although the volunteer run hatcheries are generally considered "small" (Anonymous, 2009), others have been called "quite substantial" (MacKinlay et al., 2004).

Table 1: Description of salmon enhancement techniques (adapted from MacKinlay et al., 2004).

Technique	Definition
Hatcheries	Provision of controlled spawning, protected incubation and, usually, rearing to fry or smolt size
Spawning channels	Groundwater or river fed, manned and unmanned manmade structures created to increase the available spawning and incubation area and improve conditions for spawning and in-gravel incubation
Semi-natural fish culture structures	Incubation boxes, side-channel spawning/rearing, etc. to increase freshwater survival with low-tech/low-cost intervention
Fishways	Placement of structures or removal of obstructions to improve fish passage past barriers
Habitat improvements	Placement or removal of structures to increase spawning and rearing productivity
Lake and stream enrichment	Addition of nutrients/carcasses to lakes and streams to increase primary productivity, leading to greater food availability for salmon
Public education	Classroom and educational activities, outdoor-club, First Nation and other community-based activities to increase awareness and stewardship of fish stocks and habitat and to provide economic opportunities in remote communities

PIP and CEDP programs have similar data reporting requirements. CEDP facilities are operated by local community groups under government contract, whereas PIP projects are run by volunteers and part-time staff (MacKinlay et al., 2004). Both PIP and CEDP programs are supported by DFO community advisors. Current locations for these hatcheries and programs are summarized in Table 2 and detailed in Appendix 3. Just under half (9/19) of the CEDP programs and 65% (13/20) of DFO hatcheries are located within the geographic scope of this assessment (i.e. Fraser River Watershed and Strait of Georgia). Seventy-five percent (200/265) of PIP programs are located within the Fraser River Watershed and Strait of Georgia.

The Freshwater Fisheries Society of BC (FFSBC) is a non-profit organization that works in conjunction with the province of BC to support its fish stocking program and conservation fish culture activities. It was established in 2003 to provide this service, formerly undertaken by the provincial government. FFSBC works with the Ministry of Environment regional fisheries staff to manage and create freshwater fisheries and undertake some public education. The FFSBC operates five hatcheries: Kootenay Trout Hatchery, Summerland Trout Hatchery, Vancouver Island Trout Hatchery, Clearwater Trout Hatchery and Fraser Valley Trout Hatchery. Kootenay and Summerland hatcheries are located outside of the geographic scope of this review. Between these 5 hatcheries, some 6 million trout, Arctic char (*Salvelinus alpinus*) and land-locked sockeye salmon (*Oncorhynchus nerka*) (kokanee) are produced and stocked into nearly 850 lakes and streams in British Columbia each year under the direction of the BC Ministry of Environment (http://www.gofishbc.com/ourhatcheries/default.htm, accessed June 27, 2011). Additional information on salmonid enhancement programs in BC is presented in Appendix 3.

Fraser River sockeye salmon enhancement includes; lake enrichment, spawning channels and hatcheries. Lake enrichment involves adding nutrients to surface waters to increase the plankton food available to juvenile sockeye salmon. An assessment of the potential for lake enrichment to affect disease patterns was outside of our scope of work (Appendix 1).

Five sockeye salmon spawning channels have been created in the Fraser River drainage and are located in Weaver Creek, Nadina River, Horsefly River, Inch Sockeye Satellite and Gates Creek. There are also two hatchery programs for the Upper Pitt River and Cultus Lake stocks. Based on production data provided through the Cohen Commission, Weaver Creek was responsible for 67% of the sockeye salmon produced in enhancement facilities, followed by 14% at Nadina River. All other facilities provided less than 10% of the enhancement facility production. Based on DFO major facilities and CEDP facility production, ninety-seven percent of all enhanced populations of sockeye salmon released from SEP facilities between 2005 and 2009 were reared in spawning channels (Figure 1).The vast majority of sockeye cultivation in enhancement facilities occurred in south-western BC (Figure 2).

Table 2: Summary of the locations of all SEP enhancement facilities within (Fraser River watershed and Strait of Georgia) and outside (other watershed) the scope of this review (data from Cohen Commmission, March 2011).

SEP Enhancement Facility	Location	#
Aboriginal Fisheries Strategy (AFS)	Fraser River/Strait of Georgia	0
	Other	2
	Sub-Total	2
Community Economic Development Program (CEDP)	Fraser River/Strait of Georgia	9
	Other	10
	Sub-Total	19
Department of Fisheries and Oceans (DFO)	Fraser River/Strait of Georgia	13
	Other	7
	Sub-Total	20
Public Involvement Program (PIP)	Fraser River/Strait of Georgia	200
	Other	65
	Sub-Total	265
Grand Total		306

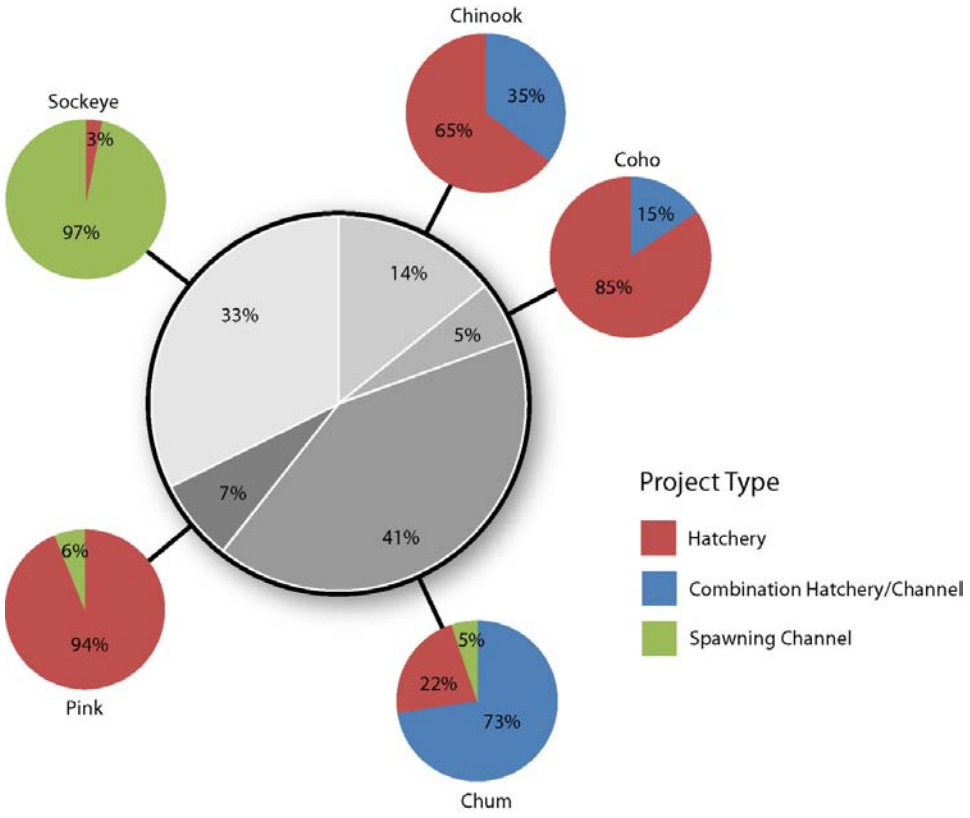

Figure 1: Total DFO enhancement facility salmonid releases (%) for all years (2005-2009) by species; satellite pie charts represent the total number (%) of each species (2005-2009) by DFO salmonid enhancement "project type".

The role of salmonid enhancement hatcheries in sustaining wild salmon populations is controversial (e.g. see Myers et al., 2004). Levin et al. (2001), stated, "While the release of hatchery fish into marine waters has occurred for decades, the central issue of the impact of hatchery fish on wild populations has rarely been seriously evaluated." The Salmon Specialist Group of the International Union for the Conservation of Nature (IUCN), however, has listed "negative effects of hatcheries and construction of artificial spawning habitat," including the spread of disease to wild salmon as one the their three major threats to sockeye salmon (Rand, 2008). Potential negative effects from salmonid enhancement include interbreeding of enhanced and wild salmon, ecological competition, mixed fisheries and/or disease transmission. The negative perceptions of hatcheries are countered by reference to the role of salmonid enhancement programs in maintaining runs that would otherwise have perished in the face of multiple pressures on their freshwater survival. It is not within the scope of this report to resolve or examine the controversy about the overall effects of salmonid enhancement on sockeye salmon: Instead we will only examine the potential effects of diseases present in

British Columbia enhancement facilities and enhanced salmonids on Fraser River sockeye salmon production as outlined in the scope of work provided by the Cohen Commission of Inquiry into Decline of Sockeye Salmon in the Fraser River (Appendix 1).

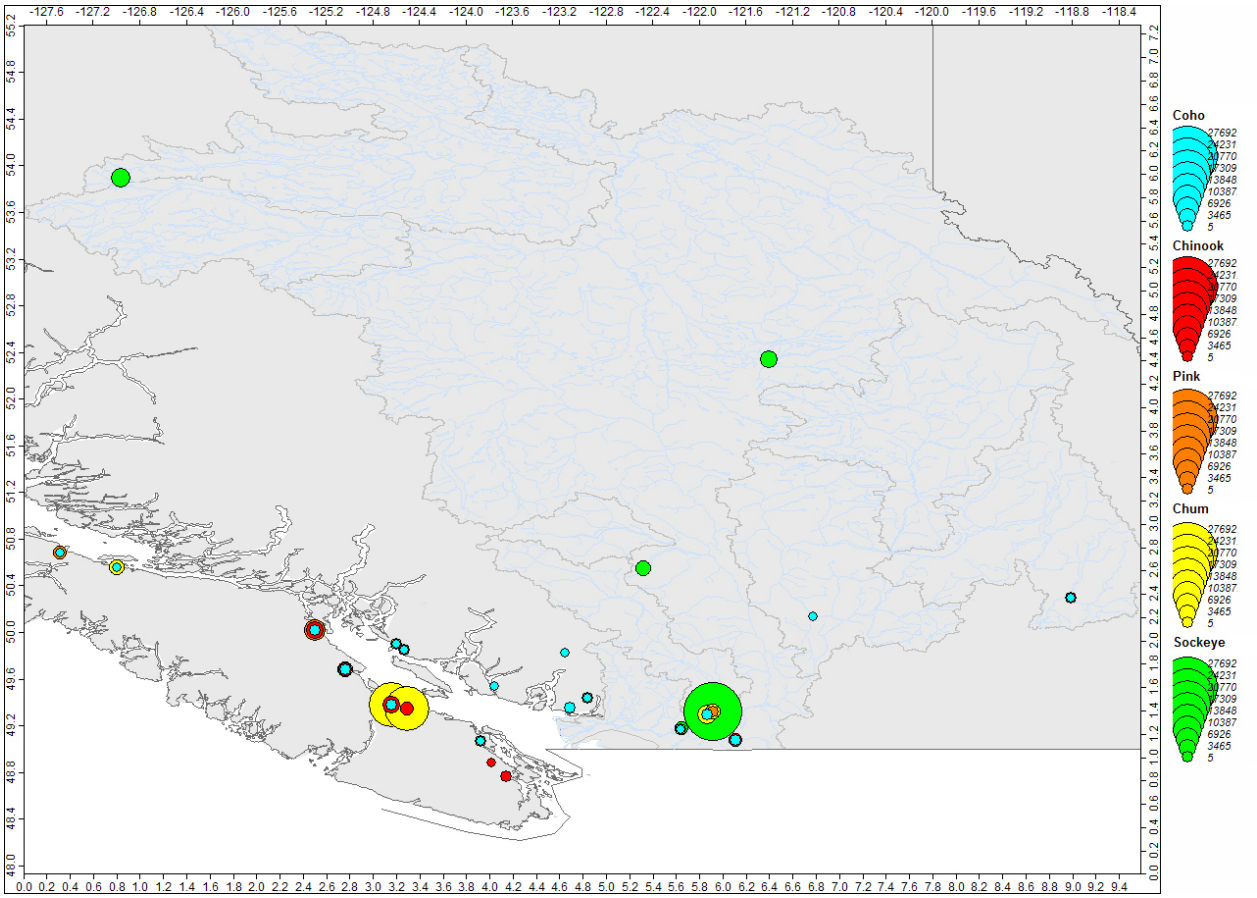

Figure 2: Average annual release statistics for DFO and CEDP facilities in the Fraser River watershed and Strait of Georgia, 2005-2009.

GIS information from DFO Spatial Data Holdings; production data from DFO records (provide by Ryan Galbraith) as requested from the Cohen Commission; map generated in SAGA (System for Automated Geoscientific Analysis).Sockeye salmon production is indicated by the green circles. Circles are scaled in proportion to production

Introduction to disease

Words like infection, disease and health are often used inconsistently or incorrectly in fish health discussions. Often, when papers or programs speak of fish health, they are really restricting their discussion to fish diseases and sometimes only to infections in individual fish. Disease is defined as an abnormality of form or function and can be caused by a suite of infectious, non-infectious and inherent factors. Disease is different than health or infection. Disease can only occur if a pathogen and/or the host's response to the pathogen causes an adverse effect in physiological functions, behaviour or integrity of the fish's organs. An infectious disease is caused by the presence and growth of a microorganism in or on a fish in such a way that it affects the form or function of that fish. Infectious diseases can be caused by a variety of microorganisms including viruses, bacteria, rickettsia unicellular parasites, multicelluar parasites and other organisms. In this report, we will use the word pathogen to include bacteria, viruses, rickettsia, and unicellular and multicellular parasites capable of causing disease. In some cases we will use the term parasite separately to indicate the occurrence of a microorganism on or in a fish without measurable impacts on form or function of the host. The presence of an infectious microorganism within a fish does not always equate to disease.

All vertebrate animals carry with them their own suite of normal microorganisms. There is a long history of collecting and identifying parasites in Pacific salmon, sometimes even using normal variations to track specific stocks (Mosquera et al., 2003). Surveys have shown that wild Pacific salmon do harbour disease causing organisms such as those causing bacterial kidney disease (BKD), vibriosis and enteric redmouth as well as a variety of parasites (Arkoosh et al., 2004; Rhodes et al.; 2006; Kent et al., 1998).

There are three possible outcomes to an infection in an individual fish. In the first outcome, the fish's immune function may eliminate the pathogen allowing the fish to recover before the fish shows obvious signs of illness. The second possible outcome of an infection is disease. Disease can be clinical (where a fish shows observable signs that it is sick) or sub-clinical (no outwards signs of disease). Sub-clinical disease is not necessarily benign because it can affect factors such as growth rate, feed conversion, ability to evade predators and ability to meet full reproductive potential. Fish that survive a sub-clinical or clinical disease may become persistently infected with the pathogen. In the persistent stage, affected fish may carry and shed the pathogen into their environment. Because most fish disease research has been conducted in laboratories under artificial conditions and because most diagnostic tests used on fish require a fish to be killed to collect samples, sub-clinical effects of diseases have been under-studied. A fish can recover from a sub-clinical or clinical infection, however, once clinical signs are seen in fish, the animal is often significantly ill and near death leading to the belief that an infected fish becomes sick fish and a sick fish dies. Such a belief is premature until new diagnostic methods can be developed to

measure and monitor fish for all manifestations of disease and the progression from pathogen colonization to final outcome can be studied under natural conditions. The third possible outcome of infection is *death*. The fish is unable to combat the effects of the pathogens and/or cope with adverse effects of the body's attempt to rid itself of infection and it dies.

In order for an infectious disease to impact population distribution or abundance, it must affect population health. Health is more than the absence of disease.

Health is determined by an individual's or population's:

1. Access to the needs for daily living, such as food and habitat;
2. Resilience to stressors and the ability to thrive and survive in the face of change and;
3. The ability to meet expectations

The latter feature of health is often the most subjective and can include anything from defining a healthy stock as one that is large enough to support a commercial fishery, to being consistent with our understanding of how long a fish might live. In general terms, infectious diseases can affect health by reducing the ability to cope with stress (e.g. reducing energy availability or negatively impacting form or function) or by impacting the ability to meet expectations (e.g. reduced reproduction).

A population can be healthy in the presence of diseased individuals in the population, therefore, the absence of disease does not equate to a population (e.g. lack of food or habitat can reduce health). Infectious disease is a normal component of ecosystems and all species live in association with a broad suite of pathogens.

As will be described in more detail below, there are many mechanisms through which disease may reduce health, from large scale die-offs, to reductions in general fitness, to reduced egg production. For this report we will assume the following when assessing health and infectious diseases:

1. Exposure to an infectious agent is necessary for a fish to become infected but is not sufficient to result in infection
2. An infection is necessary but not sufficient to cause infectious disease in a fish
3. Having a proportion of the fish population infected is necessary but not sufficient for an infectious disease to impact population health

Methods

Literature review

We undertook a scoping literature review to find direct and indirect evidence that diseases associated with salmonid enhancement facilities could negatively affect Fraser River sockeye salmon. Scoping studies are intended to explore the amount, breadth, and characteristics of primary research in a broad area and facilitate knowledge translation and identification of knowledge gaps (Davis et al., 2009) in contrast to the systematic literature review which is better suited for more focussed questions (Sargeant et al., 2005).

A scoping approach to research synthesis requires: (1) the development of a study question, (2) the development and pre-testing of the search strategy to identify potentially relevant articles, (3) relevance screening of extracted papers, and (4) information extraction (Sargeant et al., 2005). Our project team derived questions to refine our literature search strategy from the questions that were prescribed by the scope of work (Appendix 1). Our team was composed of a veterinary epidemiologist with experience in fish health, risk assessment and critical literature review; a fish health biologist with advanced training in environmental management; a veterinarian with experience in environmental impact assessment and data analysis; and a fisheries biologist.

An initial search for articles and websites on the topic of "salmon enhancement, hatcheries, disease, wild fish and related terms" was performed in the Google search engine to help identify key themes and topics to identify keywords. This was used to developed literature reviews in 3 thematic areas:

1. The exchange of pathogens between cultured salmonids and other fish, especially other salmon
2. The impacts of infectious diseases on free-ranging salmon
3. Salmon pathogen surveillance and pre-release risk assessment management

Literature was found using a combination of BIOSIS, Google Scholar, PubMed and Ringtail. Search terms included combinations of common and Latin names for Pacific salmonids, terminology specific to salmon husbandry and rearing facilities, lists of known pathogens, past reviews of salmon enhancement facility impacts and clinical/descriptive terms for disease and infection. The initial search strings are presented in Appendix 4.

The initial searches on Ringtail performed by the Cohen Commission on our behalf generated a very large number of hits so some terms were combined to refine the results. More focused searches were submitted to BIOSIS, Google Scholar, and PubMed. Our search was restricted to English language documents including peer reviewed papers and grey literature from

government or non-government agencies with preference for papers that provided data in support of their conclusions and opinions. Abstract-level relevance screening was used to identify potentially relevant literature. Articles were collected and reviewed to confirm their relevance within the context of British Columbia and the questions outlined in the statement of work (Appendix 1).

Risk assessment

The Cohen Commission was asked to request from DFO and the FFSBC information clustered around 3 objectives:

Objective 1: To assemble a representative list of infectious hazards present in salmonid hatcheries in BC that are within the movement corridor of the Fraser River and Strait of Georgia.

> Data requested included: Pacific Biological Station (PBS) Fish Health database relevant to all DFO hatcheries (n=11), spawning channels (n=5), all Community Economic Development Program (CEDP) facilities (n=9), a random sample of 30 Public Enhancement Project (PIP) facilities within the movement corridor for 2005-2010. From the same facilities, we also requested facility level records to capture conditions diagnosed without PBS involvement including low level endemic problems, conditions managed without drug treatment and other in house or externally diagnosed infectious conditions. The random sample of PIPs was based on identifying facilities within the Fraser River drainage and in the Strait of Georgia and calculating a sample size that would allow us to identify diseases found at 10% prevalence or more with 95% confidence. A secondary list of 15 randomly selected PIP facilities was included to compensate for non-responders to the data request. We also requested the diagnostic records for FFSBC hatcheries within the study area (n=3).

Objective 2: To understand the possibility of exposure of free-ranging sockeye salmon to pathogens of hatchery origin.

> We requested from the Cohen Commission, population data on all hatcheries and spawning channels included in Objective 1 including:
>
> 1. Number of salmon released and returned to each hatchery (average and range for 2005-2010)
> 2. Locations of smolt release if different than the geographic location of the hatchery
> 3. Average date of release and return to the hatchery
> 4. Information on the average movement patterns of free-ranging Fraser River sockeye salmon

Objective 3: Characterize the practices in place to reduce the prevalence, movement or release of pathogens from salmonid enhancement hatcheries.

> From the hatcheries and spawning channels included in Objective 1, we requested that the Cohen Commission obtain Fish Health Management Plans (FHMP), their date of implementation and any reports of audits of the implementation of the Plans. We also requested information on requirements for and procedures for pre-release risk assessments. In the absence of an approved FHMP, we asked for standard fish health operating procedures. A request was also made for waste management plans including information on the current status and changes in the past 10 years regarding source and treatment of incoming water; wastewater treatment and methods of release; fate of carcasses (including disposal methods); and if carcasses are used for stream enrichment, the locations where the carcasses were deposited.

In certain circumstances, the Cohen Commission, DFO, or the FFSBC were unable to fulfill these data requests because in some cases the requested data did not exist or there were data deficits, and in other cases time constraints for the review precluded the submission of data to the reviewers. Challenges with data acquisition and use are provided in the risk assessment section below.

In response to these requests, we were provided with 3153 files; 2590 dealt with diagnostic records, screening test results and health management plans for DFO hatcheries, spawning channels, CEDP and PIP facilities. An additional 486 files represented similar data for the FFSBC. A single file could contain 1- 50 pages of datasets, handwritten notes or data sheets. The remaining files were scientific papers, emails, copies of slide presentations and some reports.

Diagnostic and fish health data and records were provided from the DFO and the FFSBC, from their fish health units and from a selection of hatcheries (details provided below in risk assessment section). These records came as PDF scans of computer generated and handwritten records which we re-entered into Excel for analysis.

DFO major facility and CEDP hatchery release data for Pacific salmonids were obtained via a Cohen Commission request to the DFO. All data were manipulated in Microsoft Excel using pivot tables, and exported to SAGA (System for Automated Geoscientific Analysis) for mapping. Geospatial data for DFO and CEDP locations were obtained from DFO Spatial Data

Holdings, online at <http://www.pac.dfo-mpo.gc.ca/gis-sig/themes-eng.htm>. FFSBC hatchery georeference codes were manually located through an address search on Google Earth.

Meetings were arranged through the Cohen Commission with the following personnel in order to discuss details of the data and practices:

Pacific Biological Station and Fisheries and Oceans Canada:

- Dr. Christine MacWilliams – Veterinarian, DFO Pacific Region
- Dorthee Keiser – Biologist and former head of the fish health diagnostic unit
- Mark Higgins – Biologist and member of Introductions and Transfers Committee
- Dr. Stewart Johnson – Head, Aquatic Animal Health Science
- Dr. Simon Jones – Fish Health Research Scientist

Freshwater Fisheries Society of British Columbia

- Sherry Mead – Section Head, Fish Health Services

Framework for reviewing the literature for casual relationship between salmonid enhancement and productivity of Fraser River sockeye salmon

It was not possible, based on available literature, to quantify a proportional contribution of disease effects attributable to salmonid enhancement facilities on the productivity of Fraser River sockeye salmon. There were 2 cases of disease outbreaks in sockeye salmon spawning channels that described or estimated relatively large losses within the population in the affected year but neither provided long term follow-up or evidence of the proportional contribution of the enhancement activities as a risk factor for the outbreaks (1 involving a parasite in spawning fish, the other involving a virus in fry -details provided below). We therefore were required to combine indirect evidence to see if causal relationships between salmonid enhancement associated diseases and Fraser River sockeye salmon production were plausible and likely.

There are three general ways in which questions of cause and effect are studied for fish health problems: in the laboratory, in a one-to-one diagnostic setting and population-based observational studies. Until very recently, fish disease research has focused almost exclusively on the first two settings particularly as they relate to parasitology and microbiology. Early fish health researchers were more involved in parasite taxonomy than pathology. It was not until the 1950s that fish disease research shifted away from its preoccupation with identification and characterization of parasites and began to examine the role of other pathogens and toxins in fish disease (Mitchell, 2001). The desire to use fish as bioindicators of pollution, and the growth of commercial and public fish culture, served as driving forces for an intensification of fish disease

research (Moller and Anders, 1986). By the 1960s the role of environmental stressors in fish disease became an important subject of investigation and a key consideration for diagnosticians. Fish disease investigators began to consider the interaction of host, agent and environment necessary to bring about disease.

Fish health research has largely focused on the experimental examination of impacts on individuals rather than populations, in large part due to the relative ease of examining the effects of pathogens under controlled laboratory conditions in comparison to measuring the suite of disease determinants under natural conditions (Hedrik, 1998). The role of disease as a population regulating factor in wild fish remains significantly under-researched in the fish health literature. In 2001, a review of 10 issues of two prominent fish health journals found that out of 194 articles, 28% dealt with the pathological response of a fish to an infection, 27% dealt with aspects of microbiology, 12% were concerned with treatment of individual fish, and 9% were concerned with the transmission and epidemiology of infectious diseases (Stephen and Thorburn, 2004). None dealt with the effects of disease on populations or ecosystems. This is in contrast to standard approaches to ecological risk assessment where population survival and interactions, ecosystems, and biodiversity are the subjects of concern rather than individuals (Firestone, 2006; Gochfeld and Burger, 1993).

More attention to the regulating effects of parasites and some pathogens can be found in the ecological and parasitological literature. Dobson and May (1987) adapt general epidemiological models to support the hypothesis that pathogens can affect and are affected by fish population processes. Sindermann's (1987) summary of the effects of fish parasites indicate the capacity for infections to cause direct mortality as well as cause pathology in individual fish, rendering them incapable of coping with environmental stressors.

In our search for information on the role of diseases in wild fish, we often encountered quotes such as; "The practical difficulties in measuring the prevalence, incidence, and pathogenicity of diseases in wild [salmon] populations cause serious problems in determining the possible implications of disease" (McVicar, 1997) or "The state of the science for understanding the impacts of pathogens on wild salmon in British Columbia is minimal" (Kent, 2011). Brown (2003) concluded that, "Although several pathogens and diseases are widely present in west coast hatcheries and watersheds, and can cause severe problems to salmonid populations, there is little empirical evidence of widespread transfer of disease and pathogens from hatchery to wild fish. However, there have been relatively few studies to determine if this is a serious problem". Due to prevailing knowledge gaps, Naish et al. (2008) concluded that "The role that hatchery fish play in affecting the disease ecology of wild salmonid populations is highly equivocal." Finally, Gardner et al. (2004) concluded, "There is very little information available on the incidence of disease in wild populations, so it is almost impossible to know whether it is higher

or lower in wild fish exposed to hatchery fish. If new diseases did appear in wild populations, it may be uncertain whether these are endemic diseases that have simply never been observed before or an exotic disease introduced to the population".

Major limitations in studying disease in free-ranging fish include; the inability to follow individual fishes over their life course, problems in accessing fish for diagnostic sampling in a non-lethal manner, biases in finding and capturing wild animals (the effective of these biases on measurements of impact are largely unknown in wild fish disease studies); and a void of information on specific fish histories of pathogen exposure. The general lack of baseline information that has been collected long enough to account for environmental and epidemiological variability complicates attempts to conclude that disease in free-ranging fish are increasing or decreasing (Ward and Lafferty, 2004). Bakke and Harris (1998) suggested that the lethality and rapidity of mortality of some fish diseases plus the challenges of being able to trace the natural history of diseases (in part due to delays in detection and response) lead to failures in our ability to fulfill epidemiological criteria for causation. For example, McVicar et al. (1993) attempted to correlate the presence of pathological lesions and pathogens to decreases in body condition and population size in seatrout (*Salmo trutta*) in Scotland. Despite detecting significant pathology, associations between pathological lesions and health effects were inconsistent and prevented the authors from drawing conclusions on the effects of diseases in these populations. Similar problems have been encountered when trying to explain pre-spawning mortality in Fraser River sockeye salmon. Investigations have indicated that gill and kidney diseases play a significant role in mortality, in particular those caused by *Parvicapsula minibicornis, Loma* spp., columnaris disease and *Saprolegnia* spp., but the search for a single infectious cause of pre-spawning mortality has been unsuccessful (Patterson et al., 2009). Rather, pre-spawning mortality appears to be complex and can likely result from a multitude of factors (not all pathogen specific) whose effects can change both annually and seasonally.

Research on the abundance, distribution and impacts of diseases on Fraser River sockeye salmon was sparse in the literature, limited to a small number of endemic pathogens and generally unsuited for meta-analysis (a statistical technique to summarize and amalgamate the results of a number of quantitative studies). Long term studies of salmon diseases were rare and typically not repeated over time with the same methods or effort. Examples of long term studies include; a 10 year study of resistance to *Gyrodactylus salaris* in Finnish salmon farms (Rintamäki-Kinnunen and Valtonen 1996); monitoring returns and diagnoses of IHN or *Ichthyophthirius multifilis* in Weaver Creek and Nadia River (Garver, 2010); a 27 year review of whirling disease in Montana Rivers. Marty et. al., (2003) noted the lack of long-term studies of marine fish disease.

We were required to use analogy, limited local information and information from outside of British Columbia to indirectly examine the plausibility of causal connections between infectious

diseases, salmonid enhancement facilities and Fraser River sockeye salmon productivity. The extrapolation of information from other settings to this case must be done with caution. Biotic heterogeneity leads to high local variability of disease transmission and infection patterns in nature (Kitron, 2000), which in turn complicates how well we can extrapolate case studies and observations from other geographic settings to the Fraser River system. It is unwise to think of disease as a single issue as each specific disease will be different. For example, one tapeworm (*Eubothrium salvelini*) of sockeye salmon has 15 known fish hosts while another sockeye salmon parasite (*Philonema oncorhynchi*) has five known fish hosts, four of which are Pacific salmon (MacDonald and Margolis, 1995). The ecologies of these two different parasites will undoubtedly be different.

There are widely accepted criteria that are used to try to identify causal relationships in the health sciences. Although widely accepted, each has its limitations. There is no single criterion that can definitively establish the relationship between a specific infectious agent, a corresponding disease and impacts on a population`s health and productivity. There is a variety of overlapping approaches that are emphasized and criticized to greater or lesser degrees across various disciplines. For this review, we were guided by Evan's postulates of causation and Hill's criteria for causation (Evans, 1976; Morabia, 1991) as the basis for our framework to assemble and consider published research (Table 3). The authors of these criteria made it clear that final decision on causation in natural populations is often a matter of judgement. The criteria do not prove a cause-effect relationship, but rather help to examine the burden of evidence and determine if a cause-effect association is sufficient to warrant action. We used Table 3 as an aid to organize, assemble and assess the literature for evidence on the causal relationship between Fraser River sockeye productivity and infectious diseases associated with salmonid enhancement.

Table 3: Causal criteria used to examine the literature and consider the association between Fraser River sockeye salmon productivity and diseases of public salmonid enhancement facilities.

Number	Postulate
1	The disease can be experimentally reproduced with the suspected cause
2	Cause precedes effect
3	The proportion of cases (prevalence) of the disease is higher in exposed than non-exposed populations
4	The amount of exposure should be higher in populations with the disease than those without
5	It can be shown prospectively that exposure to the causative agent increases the number of new cases of the disease (incidence)
6	Exposure to the putative disease causing agent is higher in those with disease than those without, all other factors being equal
7	The level of exposure increases, so too does the amount of disease (dose-response)
8	Preventing the hosts response or eliminating the suspected cause eliminates the disease
9	The strength of the association of the putative cause and the effect of concern should be statistically strong and make biological and epidemiological sense
10	The relationship between the suspect cause and the effect have been consistently observed by more than one researcher and in more than one way

Literature Review Results

Linking effects with causes: how do we define effects?

Causation is multi-factorial

It is tempting to think of a cause as a single entity, event or condition which inevitably leads to a specific outcome. This is rarely the case in biomedical situations, especially when population

health and disease are being considered. The presence or absence of a disease typically requires a complex interplay of factors. When referring to wildlife populations, Holmes (1995) said, "Looking for a single, consistent cause for population regulation is not only wishful thinking, but also hinders our efforts to understand population dynamics. Population regulation is not only multifactorial, but interactions among those factors are important; single-factor experiments can miss important interactions. In addition, the ecological context constantly changes, so that regulatory processes track a moving target; experiments can have different results if the context differs." Similarly, the goal of determining the impact of a specific disease on the survival of wild fish is frequently unanswerable due to the high variability in exposure settings and in environmental conditions and biological responses; high level of uncertainty due to infrequent or inaccurate measurements; and large number of unknown interacting factors (Hammell et al., 2009). The reliability of estimating the magnitude (or the probability of error for the estimate) of the impact of infectious disease in isolation is questionable when the background mortality rate is highly unpredictable. Particularly relevant to migrating populations like salmon, there is considerable population fluctuation due to natural mortality caused by many influences and interactions on overall survival (Noakes et al., 2000). The likely non-linear relations between causal factors and population regulation would make postulate 7 in Table 3 hard to fulfill as it often assumes a linear dose-response relationship.

Population versus individual level causation

Many of the prevailing epidemiological models look for fish disease risk factors at an individual level (e.g. the presence of pathogen X increases the risk of a fish getting disease Y). The individual risk factor approach assumes populations are a collection of individuals and the nature of the interactions between individuals does not alter patterns of risk (Koopman and Lynch, 1999). But, in this report, we are interested in effects on populations and not isolated individuals. Individual-level risk models fail to recognize that individuals interact with other individuals and other pathogens in a dynamic and variable environment. The wide variety of immune responses and population responses that occur in the face of disease make it very unlikely that coexisting diseases will act independently (Adler and Brunet, 1991). For example, the virus responsible for erythrocytic inclusion body syndrome is thought to increase the susceptibility or effects of other disease agents in salmonids (Rodger et al., 1991). As another example, the pre-existing richness and saturation of fish hosts with parasites can affect the establishment of introduced parasites (McIntyre, 1996).

Population interactions can be more important driving factors in determining the pattern and impacts of disease than the interaction of a host and its pathogen. Changing the connections between exposed and unexposed individuals within a population can often affect population infection levels more than changing the exposure status of individuals (Koopman and Lynch,

1999). Disease dynamics can be changed by altering the proportion of a population that is susceptible to disease (Anderson and May, 1979). Some authors have hypothesized that the release of a large number of naive susceptible fish from hatcheries may change disease risk to wild salmon (Naish et al., 2008) not because they introduce more infections, but rather because the increase in the proportion of the population susceptible to infections increases the risk of a disease outbreak. These naive fish could also become infected and serve as a source of infection to the wild cohort with which they commingle (Naish et al., 2008). Methods such as network analysis are now being used in health research in people and terrestrial animals to better understand infection dynamics within complex systems but they have not yet gained prominence in fish disease research.

Potential mechanisms for impact on Fraser River sockeye salmon by diseases associated with salmonid enhancement

Table 4 introduces hypothesized mechanisms by which enhancement operations could influence the disease status of Fraser River sockeye salmon. The purpose of Table 4 is to illustrate that we can conceive of a variety of plausible routes beyond the direct transmission of pathogens from enhancement fish to Fraser River sockeye salmon as a means to affect disease patterns.

Examining the criteria in Table 3 requires careful consideration of the exposures and outcomes for which associations are being made. Fish health literature concerned with outcomes other than individual fish disease can be roughly categorized in three types (Stephen and Thorburn, 2004).

Table 4: Some hypothesized mechanisms by which salmonid enhancement could affect diseases that in turn affect the health and productivity of Fraser River sockeye salmon.

Putative causal variable	Hypothesized effects
Infectious microorganisms are released with fish, wastes or other material in/from a salmonid enhancement facility	The prevalence of infection increases in Fraser River sockeye salmon; this increased prevalence increases the rate of lethal disease which in turn directly reduces the number of sockeye salmon
	The prevalence of infection increases in Fraser River sockeye salmon; this increased prevalence increases the rate of non-lethal disease which in turn indirectly reduces the number of sockeye salmon by impacting population regulating variables
	The prevalence of infectious disease increases in animals ecologically important to Fraser River sockeye salmon (e.g. prey species or other salmonid species), which in turn affects disease risks or food availability for Fraser River sockeye salmon
Selection pressures on sockeye salmon reared under enhanced conditions affect the genetic composition of the population	Interbreeding between the enhanced and non-enhanced sockeye salmon increases their susceptibility to infectious disease
Ecological interactions between enhanced salmonids and Fraser River sockeye salmon	Competition or social interactions could affect the exposure of Fraser River sockeye salmon to infectious agents in their environment
The operation of a salmonid enhancement facility alters the environment	The stressors associated with the altered environment negatively effects the immunocompetance of Fraser River sockeye salmon making them more susceptible to infectious diseases

First, there are publications concerned with the effects of disease on the survival, growth and carcass quality/safety of cultured fishes. Second, there are publications interested in documenting the pathological and community structure response of fish to pollutants. In these cases, the aim is not necessarily to examine fish health *per se*, but instead to use fish health as an indicator of effects on ecosystems. Finally, there is a small body of literature examining the effects of microorganisms on ecologically important functions in wild populations. These publications typically attempt to examine isolated effects of specific parasites or other biological agents on variables such as reproduction, predation or growth. Recent work in parasitology has been studying or modelling population impacts of fish parasites (Kent, 2011). Although it is becoming more widely accepted that infectious diseases can affect the size and structure of wild animal populations (including fish), thorough documentation of these effects are minimal in the literature (Arkoosh et al., 1998; Hammell et al., 2009). The fish health literature often relies on a small number of case studies to demonstrate the possibility for diseases to cause significant mortality in wild fishes: Examples include; *Ichythophonus hoferi* in herring, Viral hemorrhagic septicaemia in freshwater fishes, herpes-type virus in pilchard in Australia and *Gyrodactylus salaris* in Atlantic salmon Norway (Johnsen and Jensen, 1991; Marty et al., 2003; Kent, 2011).

As Kent (2011) pointed out in his technical report to the Cohen Commission (Infectious diseases and potential impacts on survival of Fraser River sockeye salmon), despite there being several surveys for pathogens in wild Pacific salmon, there are only a small number of cases where the pathogen has been associated with large scale mortality. Most observations of large-scale mortality in wild Pacific salmon are based on en route or pre-spawning mortality (Kent, 2011) and most of what we know of free-ranging Pacific salmon diseases comes from fish leaving or returning to enhancement facilities (Stephen and Thorburn, 2004). Most reports are of overt die-offs rather than the chronic or sub-clinical effects that influence the ability of fish to reproduce or survive. The most likely diseases to continue to spread within a population are those that infect fish but allow a fish to live with its cohorts for longer effective contact times. This stands in contrast to most historical fish disease research that has focussed on rapidly lethal diseases and not on the dynamics of chronic, endemic disease. There has been a small number of case studies wherein a pathogen has been associated with epidemics and declines in population size in wild fish, but almost all studies looked at short term effects; long term studies (e.g. >4 years) are not available (Stephen and Thorburn, 2004; Marty et al., 2003).

How might salmonid enhancement facilities affect Fraser River sockeye salmon disease status?

Establishing direct transmission of pathogens as a route of exposure

Salmonid enhancement facilities typically aim to release their fish timed with the movement of outmigrating wild fish (MacKinley et al., 2004). This could allow for commingling of wild and enhanced fish and thus present possible opportunities for transmission of infections between these groups (Rhodes et al., 2006). Transmission of pathogens may also occur with indirect contact, such as was evident when wild fish passed Infectious Hematpoeitic Necrosis virus (IHNv) to hatchery fish despite the presence of physical barriers between the fish (Anderson et al., 2000). The practice of placing dead spawned salmon carcasses into a stream to provide nutrient enrichment to the stream has also been nominated as a potential way for pathogens to be released into the aquatic environment (Anon, DFO) and thus a possible route of exposure for diseases such as whirling disease (e.g. Arsan, 2006).

Despite these possibilities, establishing that a Fraser River sockeye salmon has been exposed to a pathogen originating from or increased by a salmonid enhancement facility would require the ability to distinguish different strains of a pathogen at a genetic level (i.e. "genetic fingerprinting") and to relate the different strains to specific sources. Molecular and genetic diagnostic methods have most often been used in fish disease work to make more refined diagnoses, for taxonomic identification of microorganisms and to describe variations in pathogens extracted from affected fishes. There are only a few published cases where these methods have been used to assist in the epidemiological investigation of the sources or spread of specific salmon pathogens. Anderson et al. (2000) used these methods to demonstrate that IHNv was introduced to hatchery kokanee from wild kokanee in Oregon; Troyer and Kurath (2003) demonstrated co-circulation of IHNv strains between private trout farms and enhancement hatcheries in Idaho; and St- Hilaire et al. (2002) used molecular methods to help describe the movement of IHNv between British Columbia Atlantic salmon farms. Nylund et al. (2007) used genetic diagnostic methods to examine the transmission of Infectious Salmon Anemia virus (ISAv) on Norwegian Atlantic salmon farms. Hanninen et al. (1995) used the methods to gather data on the origins and spread of *Aeromonas salmonicida* in Finland; suggesting that the organisms in Finland was likely derived from Swedish fish farms that were on the same bay as those in Finland. Our review, however, failed to find molecular epidemiological studies of pathogen exchange between and within Fraser River sockeye salmon in or out of enhancement facilities. Interviews with DFO and FFSBC fish health staff confirmed molecular epidemiological studies have not been done in BC salmonid hatcheries.

Evidence of exposure of wild fish to pathogens of enhancement facility origin is often indirect and/or assumed. In his report to the State of the Salmon organization, Dr Jim Winton postulated that the release of infected hatchery salmon could be a pathway for exposing wild fish to diseases of hatchery origin (Winton, unknown date). He suggested that release of infected fish at times when wild fish would not normally encounter infections, could be important in determining impacts of diseases from hatcheries, citing IHNv as an example. The capacity for sick fish to leave an enhancement facility is greater in spawning channels where fish are free to move out of the enhancement area into the rivers or lakes on their own. Traxler and Rankin (1989) surmised that fish that were part of an IHNv outbreak in a BC spawning channel left the spawning channel still infected. They estimated that 8.3 million fish died of the disease after migrating out of the spawning channel. Although the prevalence of infected fish and the mean viral titre in sampled fish decreased as the date post-outbreak increased, remaining fish continued to shed virus for over a month. Work by Maule et al. (1996) showed how fish released from salmonid enhancement facilities in the Unites States could enter rivers with high levels of *Renibacterium salmoninarum* (the etiology of bacterial kidney disease) and that the prevalence of this infection could increase or decrease post release, possibly in relation to conditions in the river. Conversely, work by Rhodes et al. (2006) found that location of capture was a much better predictor of whether or not a chinook salmon (*Oncorhynchus tshawytscha*) caught in Puget Sound would have bacterial kidney disease than whether or not the fish was of hatchery origin. That study found no difference in prevalence of infection in marked (hatchery origin) versus unmarked (presumably wild) Chinook salmon, leading the authors to conclude the probability of infection was the same for both types of salmon. Halpenny and Gross (2008) found samples of enhanced steelhead trout (*Oncorhynchus mykiss*) from northern Vancouver were free of pathogens that their diagnostic protocols could detect when the fish were still in the hatchery. The fish were monitored for 3 months post release. Infection levels became similar to a small number of local wild steelhead smolts as the released fish acquired infections in the stream. Their post-release infection status was similar to a published survey on wild juvenile coho (*Oncorhynchus kisutch*) and chinook salmon infection (Arkoosh et al., 2004 as cited in Halpenny and Gross, 2008). These authors concluded that the "clean" hatchery fish were exposed to background pathogens in the receiving waters and soon acquired infections "normal" in the fish community.

The pressure to release fish at times that mimic natural salmon migration patterns has been suggested as a reason why fish may be released from enhancement facilities while still infectious (Naish et al., 2008). However, there is little published evidence of the practice of releasing infected fish. It is important to note that the preceding examples of infect fish release should not be interpreted as the accepted standard of practice by hatchery health managers. Published cases and abstracts show that hatchery managers in the Pacific Northwest have taken disease status

into account and destroyed groups of infected hatchery fish to avoid exposing wild fish (e.g. Strom et al., 2002). The Pacific Northwest Fish Health Protection Committee of the USA developed guidelines in 1992 stating that cultured salmon that are infected with any of the virulent salmonid viruses, such as IHNv, should never be released (Flagg et al., 2000). The Committee did note that this standard is virtually impossible due to limitations in diagnostic testing. DFO staff reported that they strive to follow and adopt the guidelines of this committee. Details of BC practices are discussed in the risk assessment section below.

Impacting the susceptibility of Fraser River sockeye salmon to pathogens: Genetic effects

Fraser River sockeye salmon could be affected by diseases if salmonid enhancement facilities created situations or released material or substances that made them more susceptible to endemic diseases. Susceptibility to infection can vary and is affected by innate, acquired and external factors. Salmon genetics is one means to influence disease susceptibility. Susceptibility to infection can be different between species as was demonstrated in Chilean research, which showed how susceptibility to the parasite *Caligus* spp. varied between farmed rainbow trout (*Oncorhynchus. mykiss*), Atlantic salmon (*Salmo salar*) and coho salmon (Gonzalez et al., 2000). Genetic differences can also occur within a species and can affect the severity of infections, as was seen in Norwegian strains of wild and farmed Atlantic salmon and their response to infection challenges with sea lice *Lepeophtheirus salmonis* (Glover et al., 2004). Arkush et al. (2002) concluded that the loss of genetic variation in winter-run Chinook salmon would increase pathogen susceptibility. Allendorf and Phleps (1980) concluded that loss of genetic variability in hatchery reared cutthroat trout (*Oncorhynchus clarkii*) rendered them more susceptible to disease. Kaufman et al, (2010), undertook challenge experiments of first generation hatchery and wild steelhead trout and diploid versus triploid rainbow trout. They found that the hatchery fish were not disadvantaged in terms of susceptibility to experimental disease challenge but that triploid fish did die faster. Brown (2003) quotes, a 1996 NRC report which stated, “loss of genetic diversity due to hatchery practices could result in loss of the genes that help salmonids fight infection” and Hilborn and Hare (1992) stated that disease resistance of wild fish has been “eroded by crosses with hatchery fish.” “Infectious diseases might be both mitigated by and rapidly change the genetic composition of host populations (which) gives new significance to the role of host genetic diversity in species conservation” (Altizer et al., 2003). The validity of claims regarding the genetic effects on disease susceptibility in Fraser River sockeye salmon from inter-breeding or selection pressures within enhancement facilities remains undetermined. It was beyond the scope of our work to examine genetics effects other than impacts on infectious disease susceptibility.

Impacting the susceptibility of Fraser River sockeye salmon to pathogens: Environmental change

It has been suggested that salmonid hatcheries may change the environment in such a way that the susceptibility of wild fish may be affected (Naish et al., 2008). It has long been known and well documented that the manifestation of fish diseases can be influenced by environmental determinants and that the susceptibility of fish can be affected by environmental stressors. Environmental variability can range from local factors such as crowding in a hatchery pond to global processes such as climate change. Indeed, increases in water temperature associated with climate change have been hypothesized as a reason for increased opportunities for Fraser River sockeye salmon to be exposed to pathogens in freshwater upon their return to spawn (Hinch and Martins, 2011). Environmental conditions fluctuate greatly from year to year presenting greater or lesser challenges to growth and survival of wild Pacific salmon (Noakes et al., 2000), thus it could be expected that conditions influencing the patterns of disease in populations also changes regularly.

Snieszko (1974) reviewed the impacts of environmental stressors such as industrial pollution, temperature and fish metabolic wastes on the incidence of fish diseases. Variations in water and habitat quality undoubtedly affect the number and types of fish at particular locations and thus the potential to exchange pathogens. For example, *Vibrio anguillarum* outbreaks in Australian estuarine fish have been associated with rainfall, salinity and water temperature (Rodgers and Burke, 1981). Crowding that occurs under culture conditions or when fish aggregate in their natural environment can influence the occurrence and severity of disease outbreaks (Reno, 1998). These observations have been supported by work that documented how environmental stressors affect the fish immune system and/or the transmission of infectious agents (e.g. Mazur and Iwama, 1993). Rhodes et al. (2006) presented data suggesting water temperature and salinity, and the site and month of capture were significant predictors of chinook salmon bacterial kidney disease status in Puget Sound. Stressors from enhancement facility operations to the local environment could potentially include changes in stream temperature by large inputs of hatchery water or eutrophication from phosphorous or organic matter inputs that can increase algal growth or lower dissolved oxygen levels. We did find some evidence that wastewater from salmon hatcheries can impact water quality parameters in receiving waters (Michael, 2003); but these findings were not from BC hatcheries. No studies were found on the impacts of hatchery waste water on salmonid immune systems.

Impacting the susceptibility or exposure of Fraser River sockeye salmon to pathogens through ecological changes

Release of enhanced fish could be hypothesized to change population interactions and ecological conditions which in turn affect the way a Fraser River sockeye salmon and a pathogen come together. Non-disease related variables such as life-stage, spawning behaviour, feeding patterns and life-history can affect opportunities for pathogens to be transmitted and maintained within sub-groups of the same species and therefore further complicate the prediction of the effects of introduced pathogens. Competition can magnify differences in habitat and food selection, resulting in segregation of fish rather than commingling (Burgner, 1991) and thus result in different exposures to different pathogens in different fish communities. One group of fish can be virtually separate from other members of a biological community because of its habitat requirements and behaviours. New competitive interactions may result in patchy population distributions, rather than a homogenous mix of fishes. Even in the absence of intense competition, fish in the same habitat may partition resources so as to reduce interactions with other fish (McMichael and Pearson, 1998). For example, Bailey and Margolis (1987) showed that, within the same lake, there could be ecologically isolated groups of juvenile sockeye salmon that use different parts of their environment and thus have different parasites.

As habitats affect biological community compositions and species interactions, they also affect opportunities for transfer of pathogens. Studies by Dowling et al. (2002) in the Madison River, USA concluded; the "effects of whirling disease on rainbow trout populations are governed by a complex interaction between the timing and location of key life history events (spawning, emergence, and early rearing) and the spatial and temporal variation in the presence of the infectious stages of *M. cerebralis*" (the etiological cause of whirling disease). Scott and Hall (1997) found that undisturbed streams were characterized by a diverse array of fishes that utilized a variety of habitats, whereas disturbed streams were characterized by a lower relative abundance of fish and by a relative increase in the proportion of fish species that were habitat or trophic specialists. Patchy distributions cause densities to vary by ecosystem and life stage which will affect disease transmission and introduce more uncertainty in disease risk models (Reno, 1998). In their executive summary to the Cohen Commission, Nelitz et al. (2011) noted that there was a general lack of information that could be used to reliably define dynamic changes in condition across sockeye salmon spawning, rearing, and migratory habitats, which implies that epidemiological forecasting of the effects of change in sockeye salmon habitats on disease patterns cannot currently be reliably made.

We have insufficient information, to determine the indirect impacts of disease on sockeye salmon through effects on ecologically important species, such as prey species. Food web relationships can affect the distribution and abundance of parasites and pathogens (e.g.

Marcogliese, 2002; Ostfeld and Holt, 2004). When in freshwater, juvenile sockeye salmon feed mainly on zooplankton, amphipods and insects. A small number of papers described the detection of salmonid pathogens in these smaller creatures (e.g. Faisal and Winters, 2011), but none on the effects of those pathogens on the insects or amphipods. There is evidence that parasites can affect the distribution and abundance of aquatic insects and amphipods (Moore, 1995; Marina et al., 2005); that aquatic insect populations can be regulated by diseases (e.g. Kohler and Hoiland, 2001); that diseases in aquatic invertebrates can affect stream communities (Kohler and Wiley, 1997); and that a fish's access to specific prey species can affect its parasite status (Bailey and Margolis, 1987; Berube and Curtis, 1986); but, we could not find information specific to salmon pathogens and impacts on zooplankton, amphipods or insects in freshwater systems and subsequent impacts on sockeye salmon predator-prey interactions. When in saltwater, sockeye salmon continue to feed on plankton but also grow to eat other fishes and squid. We know very little about the true host range of many fish pathogens because fish health research and surveys have historically focused on commercially and recreationally important species (Stephen and Thorburn, 2004). There are some surveys of other species which have shown that salmon pathogens are found in non-salmonid hosts (Kent et al., 1998; Jones et al., 2006), but none have examined the effects of these infections on predator-prey relationships. Increasingly, we are finding fish pathogens affecting a wide array of hosts with different effects (Kent et al., 1998).

Ecological and epidemiological variability prevent consistent prediction

Even with a more complete knowledge of the abundance and distribution of pathogens in the Fraser River sockeye salmon ecosystem, attribution of the impacts of disease would not be consistent from year-to-year and still be elusive to estimate. Epidemic theory tells us that five variables determine whether or not an infectious or parasitic agent will persist in a population (Anderson, 1991):

1. The density of the hosts
2. The probability of transmission per contact between susceptible and infectious hosts
3. The disease-induced mortality rate
4. The per capita death rate of uninfected hosts
5. The rate of recovery from infections

We lack these data for Fraser River sockeye salmon (and most wild fish) and can assume they will change throughout the life history of sockeye salmon. For example, per capita mortality rates due to causes other than infection can be assumed to be different (not necessarily better or worse) when sockeye salmon migrate upstream to spawn than when they are foraging in the open Pacific Ocean. Population dynamics affect disease dynamics; making disease dependent on

population ecology. For example, the extent to which predators remove clinically sick fish may be an important determinant of wild fish population disease dynamics (Mesa et al., 1998). Early removal of infectious fish by predators may reduce the duration that an infected fish will co-exist with other susceptible fish and thereby may reduce the effective contact required for transmission of the disease agent. Simplistically, the number of new infections that each infected individual can cause would be lowered (Reno, 1998), but the effects would be speculative. If, as Halpenny and Gross' (2008) paper suggests, hatchery reared-salmonids have a lower complement of pathogens upon release, a large cohort of naïve fish could enter the environment. Increasing the proportion of susceptible individuals in a population can increase the possibility of an outbreak of a circulating disease (Anderson and May, 1979). Alternatively, vaccination of hatchery fish could reduce the proportion of the population that is susceptible. While vaccination may increase post-release survival of enhanced salmonids (Balfry et al., 2011), it's impact on disease dynamics of enhanced salmonids and other salmonids with which they interact has not been examined. Habitat features which affect the likelihood of a salmon interacting with another infectious fish, such as when residing in critical habitat areas like an estuary, could affect the impacts of disease. Omitting or overlooking interactions between and within species in the ecosystem likely could affect outcomes of disease transmission models and predictions of disease impacts.

Epidemiologists have been working to predict infectious diseases in people and animals for centuries, most often with the goal of predicting the timing and size of outbreaks. Most epidemiologists will admit though that accurate predictions of the details of "who, what, where, when and how much" for disease outbreaks is notoriously difficult to achieve. Without doubt we can recognize conditions that are conducive to outbreaks of disease in general. It is almost common knowledge that, whether we look at fish, people or pigs, factors such as poor nutrition, inadequate biosecurity, and crowding are all factors predisposing for disease outbreaks. But predisposition does not mean predetermination. In the case of Fraser River sockeye salmon, knowledge of predispositions is inadequate to estimate an attributable impact of infectious diseases from salmonid enhancement hatcheries on population productivity. While there is scientific literature dealing with; (1) how the abiotic environment is critical to disease outcomes in cultured fish, (2) the synergistic role of environmental pollution and infectious diseases on fish health and (3) how ecological interactions between species affect fish infections and infestations; we found no papers specifically examining or measuring how environmental or ecological changes attributable to salmonid enhancement facilities could affect disease outcomes or survival in Fraser River sockeye salmon.

Experimental evidence

Experimental studies have been useful for determining if a specific microorganism is capable of causing specific pathological outcomes; how much of the microbe is required to cause an infection or death; how differences in strains of fish or the pathogen affect disease outcomes; and candidate treatment methods. Kent (2011) used controlled experimental studies of virulence as one of his key features for assigning the level of risk specific pathogens present to Fraser River sockeye salmon.

Experimental studies and causation of disease

Koch's postulates have been used extensively as a benchmark in fish health to help researchers identify microbiological causes of specific diseases (Table 5). An example is the confirmation by Koch's postulates of a rickettsia-like organism as the etiological agent of a systemic disease causing significant mortality among coho salmon in Chile (Cvitanich et al., 1991). Researchers used a variant of Koch's postulates to demonstrate that plasmacytoid leukemia in Chinook salmon in British Columbia was transmissible, even though they could not isolate the causative agents (Eaton and Kent, 1992). Laboratory based infection studies have helped to identify a wide variety of pathogenic agents and to understand several aspects of infectious disease biology in fishes, such as virulence and lethal dose of a pathogen.

Table 5: Koch's postulates (Evans, 1976).

Postulate
The microorganism must be present in every case of the disease
The microorganism must be isolated from the host with the disease and grown in pure culture
The specific disease must be reproduced when a pure culture of the microorganism is inoculated into a healthy susceptible host
The microorganism must be recoverable from the experimentally infected host

Due to time constraints of this project, we could not independently review all of the experimental evidence upon which to create a list of known infectious agents capable of causing disease in sockeye salmon. Instead, we have chosen to rely on Kent's (2011) report to the Cohen Commission with the proviso that Kent's list of sockeye salmon pathogens is most likely incomplete. Kent (2001) reminded the reader that past studies have inadequately documented or

measured the chronic, sub-clinical or milder effects of an infection. In addition, the suite of pathogens we can detect is often limited by our detection methods, as was illustrated by Kent's accounts of a putative novel (and still undetermined) virus of sockeye salmon and of the cause of plasmacytoid leukemia. Kent (2011) also used reported prevalence or past detection of a pathogen in sockeye salmon as a criterion for assigning risk, while at the same time stating that efforts to detect pathogens in wild sockeye salmon have been relatively few and have been challenged by limitations on following and sampling wild fish. Never-the-less, Kent's report provides an overview of the experimental and observational studies needed to assemble a preliminary pathogen list for sockeye salmon in British Columbia.

Experimental studies may be necessary to associate a specific pathogen with a disease, but they are insufficient to prove causality and they do not speak to how variation in ecological, host, pathogen and environmental conditions affect the possibility of a pathogen becoming the cause of a disease or resulting in population impacts under natural conditions (Hanson, 1988). Many intervening factors affect the likelihood of a specific microorganism causing disease including: the prior history of the host; the host's behavioural patterns, environmental conditioning, and disease history; the circumstances of exposure; the environmental factors related to the host and the parasite; individual variation (genetic, physiologic, immunologic, etc.) of the host and the individual variation (strains, immunogenicity, pathogenicity, virulence, etc.) of the pathogen (Hanson, 1988). All of these interact in a complex dynamic system, making experimental studies insufficient for drawing conclusions about the effects of a pathogen in a wild population. The effects of some of these variables have been studied more under culture conditions than in the wild, again due to the comparative ease of observing, controlling and measuring these variables under a laboratory or hatchery setting as opposed to in the wild (Hedrick, 1998). While experiments may allow us to relate the effects of a single factor to the well-being of a single fish, it is another matter to interpret the response of multi-species fish communities to a suite of stressors, of which disease is only one component (Kelso et al., 1996).

Experimental impacts other than death and disease

Some researchers have tried to bridge the gap between laboratory studies and population impacts by examining outcomes other than death or overt disease in experimental settings. For example, Tierney and Farrell (2004) found that sockeye salmon swimming ability and oxygen uptake was impeded by severe infection with *Saprolegenia* spp., *Ichythophonus* spp. or by lethal injury, but not with mild or moderate disease. Wagner et al. (2005) found a similar relationship with experimental infections of *Parvicapsula minibicornis* in sockeye salmon and extrapolated their experiments to conclude that the severity of infection required to negatively affect swimming performance would not have been achieved before the returning fish passed the last hydroelectric obstacle on the Thompson River. Moles and Heifetz (1998), compared levels of *Myxobolus*

arcticus infections in the brains of sockeye salmon smolts captured in Alaskan rivers and concluded that neither smolt size nor ability to osmoregulate were affected by infections but some effects on swimming speed could be measured. Mesa et al. (1998) demonstrated that chinook salmon challenged with *Renibacterium salmoninarum* were more susceptible to predation by northern squawfish (*Ptychocheilus oregonensis*) and smallmouth bass (*Micropterus dolomieu*) under experimental conditions. Interpretation of these and similar findings is complicated by the conclusion by Donaldson et al. (2010) that physiological condition differently affected behaviour and survival of different populations of Fraser River sockeye salmon. Paradoxically, not all infections or infestations always produce negative effects as was demonstrated in an experimental study wherein three-spined stickleback (*Gasterosteus aculeatus*) were infected with a tapeworm (*Schistocephalus solidus)* and found to grow faster and were in the same or better condition than their uninfected cohort (Arnott et al., 2000). Extrapolating the experimental studies of physiological impairment due to infection must be done with some caution, but they are important in that they demonstrate that looking only to death or overt clinical diseases to measure disease effects in fish would misrepresent the potential impacts of disease.

Experimental approaches have also been used to demonstrate that the removal of a suspected casual microorganism could prevent disease, thus contributing to criterion 8 in Table 3. These have mostly been laboratory studies on the effectiveness of drug or chemical treatments. The clinical trial methodology is thought to be the most rigorous means to assess the effects of a treatment on the patterns of disease in natural conditions (Dohoo et al., 2003), providing a more realistic assessment than a laboratory experiment. Methodological challenges of clinical trials have not been overcome in fish culture settings and thus this method has rarely been used in hatchery settings. The clinical trials that have been conducted in aquaculture could best provide evidence that a suspected pathogen could cause disease in individual fish and/or on the rates of disease in fish held under culture conditions.

We concluded that experimental evidence has provided important insights into the types of pathogens to which Pacific salmon, including sockeye salmon, are susceptible, and the pathological processes by which they cause death, disease or infirmity in salmonids. Experimental studies have provided a solid basis from which to develop hazard lists for risk assessments. Two pathogens most extensively studied under experimental settings in Fraser River sockeye salmon have been Infectious Hematopoietic Necrosis virus (IHNv) and *Parvicapsula minibicornis*. The inability of experimental studies to account for natural conditions and variability and their lack of focus on population health outcomes (like reproduction) limits their utility for drawing casual conclusions about the effects of disease on Fraser River sockeye salmon productivity.

Diagnostic and observational studies

Observational studies, fish disease and causation

In order to satisfy most of the remaining criteria for causation in Table 3, study designs need to allow for comparisons under natural conditions. Comparisons need to be made between groups of different disease and/or exposure status. These states need to be compared at more than one point in time. Our overview of fish disease research found that the vast majority of non-experimental studies could best be described as descriptive studies (case reports, case series or surveys); study designs lacking comparisons and thus lacking in capacity to be make causal associations between exposures and effects (Dohoo et al., 2003). Many studies were hampered in their ability to meet our first casual criterion (Table 3), that cause precedes effect, as exposure and effect were measured concurrently, most often at a single point in time. Few studies could pinpoint where and when a fish was exposed to pathogens as many of the pathogens are ubiquitous or endemic and could come from multiple sources. A second temporal challenge to consider is the potential delay between exposure and the manifestation of adverse effects. This has been observed for invasive species (Firestone, 2006) and can be assumed to be likely for pathogens wherein there must be sufficient effective transmission within a large enough pool of susceptible fish to allow outbreaks to occur or to allow the pathogen to be maintained in a population.

Cohort and case-control studies are most often used to compare rates of exposure and outcomes in epidemiological studies (Dohoo et al., 2003). When the words “cohort study salmon hatchery enhancement” was searched in Google Scholar and PubMed databases no reports were found in the first 100 references in the former and no hits were returned from the latter database. When the words “case control salmon hatchery enhancement” were entered, equally low returns were found. In one 2006-07 survey of pre-spawning mortality in sick and apparently healthy sockeye salmon were compared for the presence or intensity of an infection. No differences in severity or intensity of *Parvicapsula minibicornis* infections in moribund and ‘healthy’ fish were found, leading the investigators to conclude the pathogen was not playing a causal role in mortality in those years (Hinch et al., in Hinch and Gardner (Eds.), 2009)

Sometimes authors described their studies as case-control designs when comparing fish exposed to one area of risk and not another (such a design is better described as a cohort study as fish are defined on the basis of exposure rather than outcome). There are methodological challenges in accounting for unmeasured variability in exposure interactions when exposure status is determined on place of capture or residence alone. This is especially true for species such as sockeye salmon that can move over very large areas within their life. The debate on the issue of cohort and case-control study design for wild salmon research is beyond the scope of this report,

but we can conclude that study designs usually required to fulfill criteria 5 and 6 from Table 3 were absent in the literature on salmonid enhancement hatchery disease in general and specifically as it relates to risks to Fraser River sockeye salmon.

Cross-sectional studies and surveys

Cross-sectional studies and surveys of wild fish have added to experimental studies that concluded infections can result in adverse impacts other than premature death or clinical disease. In these study designs, a sample of fish is examined and both exposure and outcome status were measured at the same time. These types of studies can be used to seek associations between exposures and outcomes (Dohoo et al., 2003); thus, contributing to casual criteria 3, 4 and 9 in Table 3.

The cross-sectional or survey study design has provided some evidence about the association between infectious diseases and ecologically important outcomes in fish. Traxler et al. (1998) reported an association between pre-spawning losses of sockeye salmon in spawning channels due to the parasite *Ichthyophthirius multifiliis* and reduced fry production. In the 2 years where pre-spawning mortality was high due to this parasite, fry production was estimated to be 153.6 million fewer than historic averages, whereas in the year where mortalities due to *Ichthyophthirius multifiliis* were not evident, fry production returned to average levels. Traxler et al. (1998) speculated that the accumulation of a large number of fish held below weirs at the entry to spawning channels allowed for the parasite, likely residing in local wild fish (kokanee and peamouth (*Mylcheilus caurinus*)), to increase to epidemic proportions in the returning sockeye salmon. While a reasonable hypothesis, their data could not prove this association and the authors recognized that complicating factors such as elevated water temperature and migratory stress may have affected the relationship. Of interest was their detection of other populations of sockeye salmon in the same areas that had high prevalence of this same parasite with no apparent impact on pre-spawning mortality. This report made mention of 4 instances over a 30 year period where Fraser River sockeye salmon with pre-spawning mortality also had *Ichthyophthirius* infections. Other cross-sectional studies of pre-spawning mortality in Fraser River sockeye salmon have concluded that aggregation at entry to the river and temperature effects both impact the rates of *Parvicapsula minbicornis* infections in returning fish (Jones et al., 2003).

Other examples of fish population impacts recognized through surveys and cross-sectional studies include: the ectoparasite *Ichthyophthirius multifiliis* apparently affecting mate choice by stickleback (Apanius and Schad, 1994); depression in female fecundity in Pacific hake (*Merluccius productus*) which increased with increasing levels of infections with *Kudoa paniformis* (Alderstein and Dorn, 1998); and the impacts of parasites on susceptibility of fish to

predation in a Manitoba lake where parasitized fish were smaller than non-infected fish and thus more susceptible to predation (Szalai and Dick, 1991). A similar relationship was noted in the Netherlands where cormorants caught a disproportionately higher number of fish infected with a tapeworm (*Ligula intestinalis*) than non-infected fish (van Dobben, 1952). Amos and Thomas (2002) used commonalities across case studies of salmonid hatchery, salmon farm and wild fish disease episodes in Washington State to circumstantially conclude that exposure to wild fish is a risk factor for IHN in hatchery salmonids and that IHNv and VHSv occur in wild fish but salmon hatcheries were not a source of these viruses for wild freshwater fish. Despite making these conclusions, the authors acknowledged that problems in tracking and documenting wild fish diseases and exposures threaten the validity of their conclusions.

Limits of surveys and cross-sectional studies

A major drawback of cross-sectional surveys is that the timing of exposure and disease can rarely be ascertained (Dohoo, 2003) and thus criterion 2 from Table 3 cannot be fulfilled. The role of time in changing both exposure and outcome status was illustrated by Foott et al. (2006) who demonstrated that the IHNv infection status of hatchery reared chinook salmon was different when they were in the hatchery than when they were captured many kilometres downstream after release.

Most of the sockeye salmon adult lifecycle has been inaccessible and unexplored from a pathological and epidemiological perspective. Wild salmon are not subject to ongoing surveillance. Rarely can such surveys in wild fish meet the required assumption of random sampling needed to estimate the frequency of exposures and prevalence of outcomes in the population (Dohoo et al., 2003). Hatchery fish tend to be under the watchful eyes of fish culturists, but regular and systematic monitoring of pathogen prevalence is not done routinely. The costs of high seas sampling of fish and the loss of infected fish to predation before they can be sampled means that most of what we know about salmon diseases is derived from surveys of fish when they leave and return to their natal rivers and from samples of fish in aquaculture conditions such as enhancement hatcheries or salmon farms netpens. One would need to extrapolate those findings with caution to free-ranging Fraser River sockeye salmon as their differing life histories could very plausibly mean different pathogens and exposure opportunities exist.

Pathogen surveys are likely to have biases against finding certain pathogens. The capture method used can itself affect what pathogens one can detect. For example, capture methods and post-capture handling used to survey wild salmon have been shown to cause ectoparasites to be dislodged from the fish (Bristow and Berland, 1991). While there has been some work conducted on the effects of specific sample methods in cultured fish (Thorburn, 1992) very little is known

about how different field sampling methods affect the reliability of extrapolation of results of health assessments on sampled fish to their source population. Most diagnostic tests and pathogen surveys do not included all life stages of the fish. Many disease surveys in salmon have been done on the more accessible, but demographically less important returning adults in freshwater, rather than on early life history stages (marine or freshwater) (Bakke and Harris 1998). Pathogens may cluster in different sub-groups within populations and communities due to different histories of exposure and susceptibility. Good et al. (2001), for example, found that pathogens clustered by species and age groups within Ontario salmonid hatcheries. Extrapolation of diagnostic results from limited social or age sub-groups to the entire population or region must be done with great caution when one lacks sufficient knowledge on how the disease clusters in location, species and age (Stephen and Ribble, 1995). When decisions as to where to collect fish for sampling are based on convenience (e.g. ease of access, ability to launch boats or set sampling gear) rather than on knowledge of how disease and susceptible hosts cluster, we run the risk of generating anomalous survey results with respect to the abundance and distribution of certain pathogens. By not exploring the full complement of species in a community for the presence of pathogens, we may fail to accurately characterize the infection status of that community.

The selection of which pathogen to test for also limits the utility of some surveys. Often, surveys focus on one specific pathogen or group of pathogens, excluding consideration of others. The specific media for viral or bacterial culture, the lack of anaerobic culture methods and the use of specific molecular methods further restricts the scope of pathogens we could expect to be detected. The clinical performance of most diagnostic tests (false positive and false negative rates) has not been validated for all the species of fish studied or under typical field conditions. Fish diagnostic tests have most often been developed and validated under laboratory conditions in a single or small variety of host species that have been exposed to a single (often high dose) pathogen. Many factors affect the ability of diagnostic tests to detect pathogens such as; host tissue type, strain, cross-reacting pathogens, stage of infection, management conditions, minimum detection limits of tests, prevalence of carrier fish, concentration of the pathogen in the various fish tissues and the variation in all of these factors between ages and species (Gozlan et al., 2006; Greiner and Gardner, 2000). The effects of host variation on the interpretation of tests was re-enforced by Thorburn (1996) who found the homogenous approach used in many diagnostic laboratories for screening fish for disease is unlikely to achieve the same results for all species and all pathogens. The effects of differing prevalence on the interpretation of diagnostic tests is so significant that the World Organization for Animal Health (OIE) recommends that tests should be re-validated after substantial changes in health, disease or abundance occur in target populations (OIE, 2006).

Mathematical models

Mathematical modeling of infectious disease transmission began over a century ago and it has given significant insight into the epidemiology, impacts and means to control disease. The value and use of mathematical models are subjects of debate in both the scientific and management worlds. They have given us critical insights into the role and interactions of causal factors and their control for a wide suite of diseases in the veterinary and medical sectors. Modelling is used as a foundational tool in ecology and epidemiology. However, disease models have often been erroneous or imprecise in their capacity to predict disease events as was seen for foot and mouth disease in the United Kingdom, the spread and impacts of Mad Cow Disease (bovine spongiform encephalopathy) and AIDS, as well as the epidemiology of H1N1 influenza. It is beyond the scope of this paper to review the pros and cons of modelling as a means of revealing new insights into how diseases could affect Fraser River sockeye salmon. Suffice it to say, when the terms "disease model sockeye" or 'disease model salmon hatchery" were entered into Google Scholar and Pub Med, no research was found that examined disease in Fraser River sockeye salmon apart from a few models wherein disease was hypothesized to play a synergistic role on pre-spawning mortality, along with increased water temperatures and low flows. Other papers reported on experimental models to predict pathogen genetic sequence variation or dealt with the commercial fishing sector.

Literature review conclusion

The disease impacts of salmon enhancement facilities on Fraser River sockeye salmon are largely unexplored in the literature. The literature shows that infectious diseases have historically existed and are known to be present in both Fraser River sockeye salmon and enhanced salmonids in British Columbia. These pathogens are capable of causing clinical and sub-clinical impacts on individual fish. The literature was unable to provide any information on the likelihood of a salmonid enhancement facility affecting Fraser River sockeye salmon through infectious diseases, a measurement of the magnitude of the hypothetical impacts, or the ability of enhancement facilities to prevent or mitigate the risks.

While past reviews of the impacts of hatcheries on wild salmon have hypothesized, explored or suggested a possible role for disease as a negative impact on wild salmon, direct evidence is lacking (e.g. Rand 2008; Naish et al.; 2008; Myers et al.; 2004; Levin et al.; 2001; Gardner e. al.; 2004). Our literature review failed to find sufficient direct or indirect evidence to fulfill the criteria for causation outlined in Table 3 (Table 6). Research designs and the challenges of studying fish disease under natural settings generally precluded the fulfillment of the criterion that cause preceded effect (apart from experimental studies and impacts on individual fish) or

allowed for measurements of exposures sufficiently precise to determine the origins of infectious agents.

There is biological and epidemiological plausibility that diseases, under certain environmental conditions, could affect ecologically important features of wild fish and experimental evidence that pathogens can cause death, disease and impaired physiology in individual fish. A small number of historic cases have associated the presence of pathogens in Fraser River sockeye salmon with acute and sometimes large scale mortality, but these events have not been linked to salmonid enhancement facilities except wherein spawning channels have increased fish density in the short term and supposedly increased infection rates (See Traxler et. al. 1988 and Traxler and Rankin, 1989). However, even this latter association is supposition and not proven definitively.

Table 6: Summary of evidence relating to criteria for causation (Table 3) and the relationship of salmonid enhancement facilities in British Columbia and the health of wild Fraser River sockeye salmon.

Postulate	Summary conclusions	Criteria fulfilled
The disease can be experimentally reproduced with the suspected cause	Experiments reveal a wide suite of possible hazardous microorganisms to which both cultured and wild salmon are susceptible, but experiments could not determine population impacts	Partially fulfilled
Cause precedes effect	Study designs typically measured disease and exposure concurrently; studies could not demonstrate pathogens were of hatchery origin using molecular epidemiology; past exposures were challenging to document outside of hatchery settings	Not fulfilled

The proportion of cases (prevalence) of disease is higher in exposed than non-exposed populations The amount of exposure should be higher in populations with the disease than those without	Some surveys, cross-sectional studies and outbreak investigations provided evidence that diseases can be associated with changes in ecologically important functions in wild salmon, but these approaches have not been applied to the question of Fraser River sockeye salmon productivity and disease effects associated with salmonid enhancement facilities	Partially fulfilled
It can be shown prospectively that exposure to the causative agent increases the number of new cases of the disease (incidence)	Study designs did not allow for the differentiation of old and new cases	Not fulfilled
Exposure to the putative disease causing agent is higher in those with disease than those without, all other factors being equal	Challenges to measuring exposure prevent this criterion from being fulfilled. Surveys rarely measured other risk factors when looking at disease	Not fulfilled
The level of exposure increases, so too does the amount of disease	No dose-response studies were found under non-laboratory conditions	Not fulfilled
Preventing the hosts response or eliminating the suspected cause eliminates the disease	Experimental work allowed for some of this to be fulfilled for effects of a pathogen on individual fish, but no work was found on impacts on Fraser River sockeye salmon populations or effects of treatments on impacts on free-ranging populations	Not fulfilled
The strength of the association of the putative cause and the effect of concern should be	Biological and epidemiological analogy were the strongest and most reasonably fulfilled casual	Fulfilled

statistically strong and make biological and epidemiological sense	criteria	
The relationship between the suspect cause and the effect have been consistently observed by more than one researcher and in more than one way	Studies of the effects of disease on wild fishes were rare under natural settings and were generally not repeated; no repeat studies were found for Fraser River sockeye salmon apart from follow up studies of 2 disease outbreaks in spawning channels	Not fulfilled

Risk assessment

Introduction

In this section, we use a risk analysis framework to review fish health data specific to British Columbia salmonid enhancement operations within the Fraser River watershed and Strait of Georgia. Data were provided by Fisheries and Oceans Canada (DFO) and the Freshwater Fisheries Society of BC (FFSBC) through request to the Cohen Commission. Health risk analysis is a process intended to characterize the nature, likelihood and severity of potential adverse effects of exposure to hazardous agents or activities. It is composed of the following steps:

1. Identify the health standard
2. Hazard identification
3. Exposure assessment
4. Consequence assessment
5. Assessment of capacity to prevent or mitigate risk

Valid quantitative risk assessments are rarely possible for wild animals because the information required is usually too imprecise or approximate (Leighton, 2002). Our literature review suggests the same is true for wild salmon. The World Organization for Animal Health (OIE) recognized that the poorly understood life-cycles and survival of fish pathogens makes risk assessment difficult, even to the most studied models (Stephen et al., 2007). We undertook this risk analysis to determine if we could gain additional insight into the determinants of disease risk associated

with salmonid enhancement operations to Fraser River sockeye salmon rather than to quantify the risk.

Risk assessments are typically based on data of the current or historical situation and may not be well suited for forecasting future impacts, such as under different scenarios of climate change or management. The effects of the new Pacific Aquaculture Regulations, for example, cannot be determined from retrospective assessment. Modelling, uncertainty analysis and foresight methods may be helpful for that purpose but still require data and are based on a number of assumptions that are often hard to validate for the natural situation. The timelines for this project did not allow for risk forecasting through modelling or participatory approaches.

We were limited to undertaking a generic risk assessment for salmonid enhancement facilities as opposed to undertaking site specific assessments. The differences in the populations being reared, infrastructure, water sources and discharges, surrounding ecosystem, staff training and other factors can conceivably make the nature of risks different between enhancement facilities. While we endeavoured to consider the differences between hatcheries, spawning channels and community programs, we were unable to compare each site individually due to lack of site specific data and time to evaluate sites individually. This risk assessment, therefore, will be a qualitative overview of trends based on the information provided to us as described in our risk assessment methods.

The major limitations and challenges of the data provided to us by the Cohen Commission are summarized here to provide some context for the risk assessment. While we could not conduct additional research to validate the accuracy and completeness of the data provided to us, we could find several data limitations. The amount and quality of data varied significantly across facilities. Hatchery names for example, were not used consistently between the production data, fish treatment records and fish diagnostic records; making integration and interpretation of the data difficult. There were significant inconsistencies in the types, amounts and details of information provided in hatchery fish health and treatment records. For example, some hatcheries and spawning channels provided no fish health or in-house treatment records. Whether this reflected their true status (i.e. no health problems existed at the facility), lack of record keeping, or misunderstanding of the data request is unknown in a number of cases. A more detailed review of limitations of the diagnostic data is discussed below in the data quality section. Delays in receiving data prevented us from visiting specific facilities to help clarify ambiguities or gaps in their records including determining whether missing information was stored somewhere else or in records that did not specifically fit our data request. Data were provided to us as PDF versions of paper documents rather than as a database ready for analysis. This created problems in linking and enumerating records. For example, information on treatments could be found in paper versions of the diagnostic records, hatchery treatment

records, hatchery husbandry records and prescriptions records. Manual sorting of these files was required to try to avoid duplicating or missing information. We have confidence that our conclusions are reasonable as we did use multiple sources of information to explore risk analysis questions and none of our findings were inconsistent with past reports, reviews and the literature. However, we acknowledge that site specific details may be lacking and encourage future risk assessments be provided the time and resources to independently gather and validate data.

Identifying the health target

A health target is the standard against which to judge if the level of risk determined by the risk assessment is acceptable. Our literature review indicated that infection and disease is normal in wild fish populations; pathogens that are present in enhancement facilities can occur in the natural environment; and pathogens have been present in Fraser River sockeye salmon in enhancement facilities and in the wild, therefore, a health standard of no infections or diseases in Fraser River sockeye salmon is an unattainable standard.

Our literature review could not find an evidence-based standard to define an acceptable level of infection in terms of the frequency or distribution of transfer of pathogens from enhanced salmon to Fraser River sockeye salmon. The literature would suggest a single standard is not biologically reasonable as the capacity for individuals and populations to cope with a disease is context specific and would be affected by factors such as the pathogen, host species, life stage, habitat quality, water temperature and other variables. We know of no legal standard that establishes an acceptable level of fish pathogen risk except for legislation dealing with the exclusion of foreign or exotic disease from Canada. Significant legislation, conventions, policies and standards recognize that it is economically and ecologically undesirable to import or introduce foreign pathogens. DFO has as part of their policy goals, the prevention of the spread of pathogens into areas where they are not known to occur. In the absence of scientific evidence to define an acceptable level of risk that results in acceptable levels of harms, the health standard for disease in Fraser River sockeye salmon will need to be defined through negotiation or consensus.

Hazard Assessment

The goal of a hazard assessment is to develop a complete, inclusive list of all possible health hazards that might be associated with the proposed introduction of fish into the receiving environment (Leighton, 2002). For this report, a hazard is defined as any infectious biological agent that could adversely impact Fraser River sockeye salmon productivity.

Data source for hazard assessment

There were 3 possible sources of data available for this assessment.

1. The BC Salmon Farmers Fish Health Database
   1.1. The BC Salmon Farmers have a fish health database which stores information provided by its member companies on fish health diagnostics pertaining to farmed salmon including their hatcheries. Data regarding salmon farms were outside the scope of this review (See Appendix 1 Statement of Work).
   1.2. The FFSBC and DFO SEP agreed to provide fish health information to the BC Salmon Farmers Fish Health Database. The 2003 reporting guidelines indicated that 13 federal and 2 FFSBC facilities provided data (http://www.agf.gov.bc.ca/ahc/fish_health/bcsfa_database.htm). The data provided are aggregated on a geographic basis and do not separate public from private facilities. They, therefore, could not be used to describe fish health patterns in enhancement facilities.
2. Pacific Biological Station (PBS) and Freshwater Fisheries Society of BC (FFSBC) fish health sections- Diagnostic records
   2.1. Federal facilities (1 laboratory at the Pacific Biological Station)
      2.1.1. The PBS provides a diagnostic service (1 laboratory) to federally supported salmonid enhancement projects including their major hatcheries, the Community Economic Development Program (CEDP) and Public Involvement Program (PIP) facilities.
      2.1.2. PBS can provide a suite of diagnostic capabilities including gross pathological and histopathological examinations, bacteriology, virology, parastiology and molecular diagnostics.
   2.2. FFSBC has diagnostic laboratory services (1 laboratory at the Vancouver Island Trout Hatchery) for its hatcheries which functions similarly to the PBS program.
   2.3. These data were available for our review
3. Federal enhancement facilities- Hatchery production records
   3.1. Records were provided from 10 major hatcheries, 5 spawning channels, 2 facilities classified as combinations of hatcheries and spawning channels, and 9 CEDP facilities within the geographic area of this study, as well as a subsample of 30 of the 214 PIP facilities within the same area. A random sample of 30/214 facilities would allow us to be 95% confidence that a disease is present at less than 10% prevalence if no facilities reported the disease (10% prevalence can be high for diseases in natural populations. An original request for data from all facilities or a sample weighted by fish production was not felt by DFO to be feasible within the projects time line). The request was increased to 45 PIP facilities to account for possible non-compliance with the request by these volunteer operations. These records were reported to contain fish health information on mortality rates and records of endemic pathogens diagnosed on site. A similar request

was made for FFSBC facilities but we only received information from federal enhancement facilities.

3.2. Of the 45 PIP facilities from which data was requested, only 17 were said to be involved in fish culture (as per the explanatory note for fish health records provided by DFO)

3.3. The information provided from the enhancement facilities was not consistent across facilities. Some provided spreadsheets with mortality records, treatment records and comments on suspected causes of diseases while other provided hand-written notes. The names used for facilities differed between the PBS diagnostic records and DFO production records, resulting in challenges in reconciling these two data sources.

Data quality and quantity for identifying hazards

The quality of a hazard assessment is dependent on the quality and quantity of data available. In animal health risk assessments, monitoring and surveillance programs typically provide the bulk of information used with supplements from the published literature. There are several standard features of monitoring and surveillance systems that can be used to characterize the quality of data available for a risk assessment (Table 7).

It is important to first note that neither the federal nor provincial systems has been designed or funded to be a surveillance or monitoring program. They provide diagnostic support in response to active disease concerns in hatcheries. Epidemiologic surveillance is the "ongoing systematic collection, recording, analysis, interpretation, and dissemination of data reflecting the current health status" (Breslow and Cengage, Eds., 2002), of a population. Fish health data available for enhancement facilities were derived from investigations of unusual health events reported to central diagnostic facilities, from screening returning broodstock for a selection of specific pathogens and from some pre-release screening. Available data are largely case focused and are not subject to ongoing analyses. Never-the-less, these were the only data available to us to generate the hazards list. Assessment of many surveillance attributes require special studies independent of the monitoring and surveillance activities such as special surveys designed for evaluation purposes. Such studies were beyond the scope of this project (Appendix 1). We were, therefore, left to use information derived from our examination of the fish health data provided by PBS and the FFSBC fish health sections and hatcheries, discussions with personnel in those programs, and resources found on the internet.

Table 7: Attributes to evaluate when considering the quality of information for risk assessment (summarized from CDC, 2001; Strak and Salman, 2002).

Attribute	Goal of the attribute
Completeness	All relevant information is recorded to describe the sources and attributes of a case
Validity	Sample size and methods for collection do not result in sampling error (affected by sample size) or sample biases (affected by the source and testing of submitted samples)
Sensitivity	Monitoring and surveillance program detect all cases and/or outbreaks of disease
Representativeness	The data accurately describes the occurrence of a hazard over time and its distribution in the population by place, species and lifestage
Timeliness	The interval between the onset of a problem, its recognition and diagnosis and risk reduction strategies is adequate to prevent, reduce or characterize risks

Completeness

All PDF case records were entered into a new spreadsheet and the information fields from the PBS and FFSBC records used as columns in the spreadsheet. The diagnostic record content was transferred from the records to the spreadsheet, allowing for data elements to be summarized and analyzed. Both sets of diagnostic data did a good job at recording the species, age class and location of the fish tested (Table 8), but they varied in providing final reports in their records. PBS fish health staff reported to us that diagnostic results on cases and treatment advice were often made over the telephone or by email and that written final reports were not always provided or entered into the records. This practice was especially pronounced during broodstock screening season when the fish health staff was pressed to the limits of their human resource capacity to keep up with the diagnostic workload.

Table 8: Completeness of salmonid disease data from diagnostic laboratories for a selection of data elements.

Data element	% records at PBS	% records at FFSBC non-anadromous (anadromous)
Final reports/interpretation	47	80 (94)
Species involved	100	100 (97)
Age of fish sampled	91	91 (93)
Location (facility) name provided	100	100 (97)
Source (Wild or cultured)	88	27 (19)
Case history or reason for submission*	99	99 (97)

*The amount of historical information provided in these fields varied between submissions.

It was not possible to classify the PBS records into categories such as broodstock screening, pre-release screening and diagnostics without making assumptions based on our perceived understanding of the reason for submission as written in their records. For this reason, the relative percent of records that were diagnostic versus screening is not presented.

There were significant gaps in other data fields in the diagnostic records. For example, description of the virological method used for FFSBC fish was absent in 57% and 84% of cases (for anadromous and non-anadromous salmonids respectively). Information on whether or not tissues were taken for histology by FFSBC and their results were absent in 88-100% of the fields. One third of the FFSBC records had blank fields for whether a gross pathological examination was conducted. Similar gaps were present for many diagnostic fields and paralleled our findings for DFO records. We did not have time or resources to investigate whether these gaps reflected the diagnostic practices or the data recording practices. A blank field could mean that; (1) the test was not needed; (2) it was not done; or (3) it was done but not recorded.

Of the 1045 diagnostic and treatment record sheets received from CEDP and PIP facilities, 5 could not be conclusively identified as facility level ('In-House') or PBS diagnostic lab ('PBS)' records and were therefore excluded from any further analysis. Of the 1040 records that could be attributed to 'In-House' or 'PBS' (Table 9), 645 had a month and year date recorded or legible. Records were found for only 4 of the 17 PIP facilities (Table 9). DFO reported this was the case because most PIP facilities either had no disease outbreaks and/or no reason to treat fish**.** All of the CEDP facilities (n=9) were reflected in the PBS diagnostic database. Two CEDP

hatcheries were reported to have no formal process for recording hatchery-level health records. Based on letters or emails amongst the files we received, some facility level records were kept at Cowichan River and Deadman River facilities, but these were not reflected in the data provided to us and therefore unavailable for review.

Table 9: Summary of the sources and total numbers of fish health records provided for CEDP and PIP facilities.

Hatchery name	Hatchery type	In-House	PBS	Total
Cowichan River	CEDP		9	9
Deadman River (at Spius)	CEDP		8	8
Gwa'ni	CEDP	198	12	210
Kanaka Creek/Bell Irving	PIP	2	2	4
Marble River Hatchery	PIP	140	4	144
Nanaimo River	CEDP		32	32
Port McNeil/Kokish	PIP	48		48
Powell River Salmon Society	CEDP		2	2
Quatse Hatchery	CEDP	541	21	567
Sechelt Indian Band/Mclean Bay	CEDP		1	1
Seymour River Hatchery	CEDP	3	5	8
Sliammon Hatchery	CEDP		2	2
Sunshine Coast Salmonid Enhancement Society/Chapman Creek Hatchery	PIP		10	10
Total		**932**	**108**	**1040**

Validity

Sampling bias: The information on completeness is relevant to our consideration of sampling bias. It is not reasonable to expect all tests to be done on all fish – this is generally seen to be an inefficient way to undertake biomedical examinations (Sackett et al., 1991). Diagnosticians are better informed by history and gross examination to select subsequent tests when trying to determine the cause of a specific disease problem. However, the suite of diagnostic tests used will affect the probability of finding pathogens. For example, if a fish is not tested for a virus, we

cannot conclude it was free of a virus. These gaps in recorded data reflect the fact that the diagnostic system for salmon in enhancement facilities was created to help hatchery workers deal with clinical problems and not for complete surveys for pathogens or for ongoing surveillance.

Diagnostic cases are submitted at the discretion of fish culture staff at FFSBC hatcheries and DFO major hatchery or by the community advisor for CEDP and PIP projects. Recommended triggers at federal facilities (Personal communication, 2011, Dr. C. McWilliams, PBS; and as reported in some Fish Health Management Plans) for sample submission to the federal system include;

1) A daily mortality rate >0.1% per day for several days;
2) An unexplained daily mortality rate >0.5%;
3) Clinical signs indicative of the start of an endemic/annual disease; or
4) Unfamiliar clinical signs

The FFSBC program reportedly does not have pre-defined thresholds for reporting or for sample submission but their informal standard of practice encourages hatchery staff to contact the lab at the first signs of diseases, including abnormal behaviour and/or the presence of clinical signs. Like for DFO, FFSBC hatchery staff have some capacity to conduct diagnostic tests for common problems – mostly external parasites. But the 'culture' of the FFSBC was said to be that hatchery staff contact the fish health staff when a diseases or infection is suspected or detected and not to manage the problem on their own. FFSBC fish health staff have only anecdotal information that this system works and that they are being informed of all cases of diseases.

There is no requirement for routine visits by fish health staff or veterinarians to hatcheries or spawning channels and no formal requirements for surveillance apart from broodstock screening and management of bacterial kidney disease in federal facilities. Although facilities are able to use other diagnostic services, the fact that PBS services are free was considered a motivation for submission to PBS by DFO supported facilities and thus the PBS fish health staff believed they were capturing the majority of diagnostic samples being sent from major hatcheries, CEDP and PIP facilities – however this is an untested assumption. PBS staff interviewed believed that low level mortality events; disease issues that are endemic and readily recognized by fish culture staff; diseases due to non-infectious causes; and cases of reduced growth without clinical disease would not come to their attention.

The submission biases limit the ability to generalize the data from the diagnostic labs thus making them a poor source of data for describing the amount, frequency and distribution of pathogens in the entire enhanced salmonid population. The record of the presence and distribution of pathogens in enhanced fish is biased towards sick fish – biasing estimates of the

frequency and amount of clinical disease upwards. Mild, endemic or subclinical infections are unlikely to be submitted or detected except for broodstock screening which only looks for selected pathogens and thus likely under-represents other diseases. The diagnostic data can only be used to identify the causes of fish health events severe or unusual enough to warrant investigation and, for a few pathogens, their prevalence in a sample of broodstock. Population level surveys were not represented in the diagnostic data. Outcomes of some surveys done as part of research are presented in the literature review.

Validity of the diagnostic tests: Very little work has been done to validate the predictive value of many fish disease diagnostic tests as they are used in typical diagnostic settings, preventing assessment of false positive and negative rates for diagnoses. This is not a problem unique to fish health; it also occurs in other animal diagnostic tests but more test validation has been done for terrestrial species.

The diagnostic system is heavily dependent on the ability of the hatchery staff to recognize signs of changing fish health status and engage the diagnostic service. The fish health staff at PBS and the FFSBC spoke very highly of the staff at the major hatcheries. The PBS staff noted that for community enhancement projects, a DFO community advisor was the main point of contact with the diagnostic service. The current capacity and level of training of community advisors was said to vary from region to region. This creates a potential for variation in fish health oversight and advice being provided to individual facilities. The majority of FFSBC hatchery staff has at least a 2-year fisheries and aquaculture diploma during which some fish health training is provided. DFO major hatchery staff are offered a short course in fish health which is offered once every 2 years. We reviewed the content of this course and found it to be a good introduction to the principles of fish health management but limited in its ability to develop diagnostic skills.

The vast majority of the diagnoses made by the PBS and FFSBC laboratories were provided, in the records available to us, by a non-veterinarian. At PBS, diagnosis was most often provided by technical staff. The staff at the diagnostic labs are very experienced and have been engaged in the fish health community in BC, and the northwest United States for many years. Much of their training has been on-the-job as opposed to having specific post-graduate training in fish diseases or diagnostics. PBS has one veterinarian involved in diagnostics, management advice, treatment recommendation and prescribing medication. The FFSBC contracts one veterinarian who provides prescriptions as required in consultation with the FFSBC staff. In both cases, the veterinarians are unable to visit the hatcheries on a regular basis, limiting the development and maintenance of the expected veterinarian-client-patient relationship. None of the diagnosticians involved have advanced degrees in fish health. PBS staff does have access to fish health researchers and the FFSBC does periodically use diagnostic services at the BC Animal Health Centre, where there are 2 veterinarians with PhD level fish health training. The opportunities and

budgets for professional continuing education are few for the fish health staff although they are part of a regional network of fish health professionals.

PBS staff use the same infrastructure for their diagnostic work as used in their "sister" labs within the Aquatic Animal Health Unit which began the process of receiving ISO-17025 accreditation two years ago. The labs have developed their quality control process and infrastructure updates as required by ISO-17025 but are awaiting final accreditation. This accreditation was required for the Aquatic Animal Health Unit to support Canadian Food Inspection Agency's (CFIA) National Aquatic Animal Health Program (NAAHP). Activities done for enhancement program diagnostics follow the Fish Health Protection Regulations Manual of Compliance (DFO, 1984). Technical staff used for work done under the Manual of Compliance are crossed-trained to conduct work under the ISO level standards and thus likely to exert the same standards regardless of whether the work was for salmonid enhancement diagnostics or the NAAHP. Laboratory capacity available to the diagnostic lab is capable of undertaking genetic analysis of pathogens which could be used for source attribution, but this is not routinely done. The FFSBC facility has been recently upgraded but it has no external accreditation.

Precision of estimates of frequency of disease: Because the data available in the diagnostic records is biased towards sick fish, it is a poor source of data for estimating disease frequency and distribution. We therefore turned our attention to asking "what is the likelihood that the diagnostic system could detect diseases in the submitted fish groups?" The number of fish sampled from a population affects the confidence one has that a specific disease is present or not. The PBS diagnostic system historically received 500-700 cases per year; in recent years this has decreased to approximately 300/yr (Personal communication, 2011, Dr. C. McWilliams, PBS) (a case means an event rather than an individual fish). This estimate exceeds the number of cases in the diagnostic records provided to us of approximately 75 submissions per year (442 submissions 2005-2010). This difference may be explained in part by the fact that; (1) we did not request records for all facilities in BC; (2) we only requested submissions involving infectious diseases; and/or (3) not all submissions may have entered the diagnostic system database.

At the FFSBC facilities, the number of fish submitted per case averaged 22 for anadromous and 28 for non-anadromous salmonids but ranged from 1 to 107. For anadromous fish, the number examined on average for diagnostic or screening submission was not different but for non-anadromous fish, an average of 44 (range 7-87) were tested for screening purposes and 23 (range 1-102) for diagnostic cases. DFO diagnostic records contained 27 fish on average ranging from 1-284 fish per submission. For populations greater than 500 fish, a sample size of 25 fish would allow one to be 75-100% confident they would detect at least one fish with the disease if the true prevalence was 5%; with the same population level and sample size, one could be 25% confident

to find 1 positive if the true prevalence was 1% and 12% confident if the true prevalence was 0.5% (calculated using WinEpiscope: Sample size for detection of disease). Therefore, the average number of fish sampled provides a reasonable confidence of finding diseases that are common in the sampled population, but less confidence in finding uncommon diseases or diseases at lower prevalence. Fenichel et al.'s (2008) models support this conclusion that moderately low prevalence diseases are most likely to be those that escape detection and can be released or translocated with an infected fish population.

Case definitions (criteria used to make a diagnosis) were not provided to us. Diagnostic labs are now required to report named disease under the National Aquatic Animal Health Program (not in place for the time period of the retrospective data available for this assessment) and there was a historic database of certain diseases that were reported to Ottawa to the National Aquatic Disease database. In both cases, specific laboratory standards were/are required for diagnosis. For surveillance systems, standard case definitions are best practices to ensure that cases are consistently enumerated and described. For diagnostic purposes, diagnosticians may or may not use the same criteria to come to a diagnosis. For example, in the PBS records, a diagnosis of bacterial kidney disease may have resulted from different tests for broodstock (ELISA test) than for fry or smolts (DFAT tests) and in some cases was made only on staining of kidney tissue imprints (Gram stain). Similarly, often the diagnosis of myxobacteriosis was based on Gram stain but sometimes diagnosis also involved growing and identifying the causative agent. The accuracy and consistency of the case definitions used have not been assessed and thus the potential misclassification bias of the diagnoses cannot be characterized.

Sensitivity

We cannot estimate the likelihood that the diagnostic laboratories recorded all cases and outbreaks. The sensitivity of neither diagnostic system has been assessed. Based on our interviews with staff at the diagnostic laboratories, in cases where hatchery workers are familiar with the manifestations of the symptoms of endemic conditions, samples will not necessarily be sent to the laboratory. Because of inconsistencies in the hatchery level records, we could not determine if hatcheries varied in what they recorded and thus could not conclude if all fish health events were entered.

In the FFSBC system (Table 10), of all cases submitted, 46% were for salmonid broodstock screening, 42% for investigation of a disease issue and 6% for "health checks". The reminder were unrecorded or for other purposes. The "health check" category included some reports for pre-release assessment, some for risk assessment and others for research purposes. Broodstock testing represented 70% of cases from anadromous salmonids but only 18% for non-anadromous salmonids– a group for which diagnostic testing represented 63% of cases.

Table 10: Categories of submissions to the Freshwater Fisheries Society of BC laboratory (2000-2010).

Salmonid	Life stage	# cases/diagnostic submission	# cases/screening submission
Anadromous	Broodstock	10	123
	Juvenile	28	5
Non-anadromous	Broodstock	11	26
	Juvenile	48	4

In the PBS database, 57% of the cases could be categorized as investigations of increased fish losses or poor production due to recognized disease; 27% were involved in BKD or virus screening; 6% were called health checks. The remaining categories were pre-transfer screening (4%), pre-release screening (2%) and other or unstated (6%). The BKD/virus screening category seemed to be a combination of broodstock screening and BKD pre-release screening. This categorization of cases must be taken as a crude estimate as the historical information and descriptors of why a fish was submitted to the lab was inconsistent and used a wide variety of terms.

The fish health staff at both laboratories did not appear to have regular access to production records and therefore were unable to independently assess if trends in morbidity, mortality or growth were changing. They required notification from hatchery staff in order to become aware of on-site fish health problems. The hatchery staff are, therefore, critical points for surveillance and risk reduction programs.

Representativeness

FFSBC fish health staff reported that they dealt largely with cultured salmonids in the hatcheries of interest to this report. Their diagnostic records did have a field for classifying a sample's source as being wild or cultured fish (recorded in only 27% of the records). Broodstock collected from lakes were considered "semi-wild." The FFSBC was not involved in wild fish disease investigations.

The PBS records classified the salmonids submitted as cultured for 62% of submissions, wild for 23%, semi-wild/semi-cultured for 4% and unstated for 11%. Their criteria for this classification were not provided. A proportion of their returning broodstock or broodstock collected from nearby streams can be considered wild. Work done by DFO fish health scientists on IHNv and

Parvicapsula minibicornis were considered in the literature review above. There were reports of wild fish disease investigations each year in the PBS diagnostic data base. Because wild fish diseases were outside of our scope of work and were a small proportion of the diagnostic submissions and because the timeline for data review prevent a full assessment of hatchery results as well as wild fish results, wild fish records were not included in the hazard assessment. The opportunistic nature of wild fish investigations (often initiated by field staff or the public), the comparatively small number of cases of wild fish disease investigations and the limited spectrum of pathogens sought in broodstock screening restricts the capacity of these data sets to reflect the patterns of diseases in wild fishes.

The distribution of diagnostic material was not in proportion to the size of the populations (Tables 11 and 12). We can hypothesize that the relationship between the amount of fish produced at a site and the amount of samples sent for diagnostic examination could be affected by; (1) the pattern of disease at a site; (2) the interest, ability and/or willingness of the hatchery staff to submit samples to the lab; and/or (3) the ability of hatchery staff to recognize situations requiring sample submissions to the laboratory. No audits were available to determine the reason for the pattern of case submissions. Rates of disease or their spatial and temporal distributions were not routinely assessed and reported by either the FFSBC or PBS therefore changing disease patterns have not been interpreted in light of changing patterns of the general populations. The fish health diagnostic labs reported that trend analyses are not routinely done except in an informal manner which relies heavily on the corporate memory of key individuals.

Table 11: Average number of salmonids released/year (2000-2010) from Freshwater Fisheries Society of BC hatcheries in relation to the proportion of cases in the FFSBC diagnostic records.

Location	Average number of fish released	% of total fish released	Proportion of cases submitted (%)
Fraser Valley	865,983	19	63
Vancouver Island	351,352	8	19
Clearwater	3,363,757	73	13

Table 12: Average number of salmonids released/year (2005-2009) in DFO salmon enhancement facilities in relation to the proportion of cases in the PBS diagnostic records (2005-2010).

Location	Average number of fish released	% of total fish released*	Proportion of cases submitted (%)
Spius Creek Hatchery	722,430	0.5	39
Rosewall Creek Hatchery	Not provided	?	11
Puntledge Hatchery	8,316,790	6	10
Inch Creek Hatchery	9,283,177	6	9
Chilliwack Hatchery	4,832,231	3	8
Quinsum Hatchery	11,564,660	8	8
Capilano Hatchery	1,261,334	1	5
Cultus Lake Hatchery	Not provided	?	4
Big Qualicum Hatchery and Spawning Channels	27,616,775	19	2
Chehalis Hatchery	8,084,873	6	2
Nadina Spawning Channels	5,920,000	4	1
Weaver Creek Spawning Channels	37,510,200	26	1
Horsefly River Spawning Channels	4,025,000	3	0
Little Qualicum Hatchery and Spawning Channels	23,938,731	17	0

*Calculation made excluding values from Rosewall and Cultus Lake hatcheries. Proportion of fish released is calculated to examine the relative relationship between proportion of fish released and proportion of tests submitted.

The broodstock screening programs have a restricted role in identifying and describing disease events and patterns for three reasons. First, testing is restricted to only fish that have returned to spawn. Second, broodstock screening is limited to targeted pathogens, in a sample of the population, such as BKD and some IHNv sampling at DFO. The FFSBC screens 60 fish from all of their broodstock groups by virus culture. Fish from brood lakes and one facility exporting eggs to the Unites States also have broodstock tissues cultured for bacteria using 2 types of

bacterial culture media. Neither case could be considered a complete pathogen screening (e.g. parasite examination is not included). DFO's selection criteria for broodstock screening depend on past history of the group. High risk sites identified by technician's opinion and substantiated by fish health records are targeted for screening. Any facility with stock diagnosed with BKD during juvenile rearing will be asked to submit samples for at least the next lifecycle or until a return of the facility to low risk based on ongoing screening results and loss investigations. Periodic checks are done at major facilities to confirm their BKD status or can be done at the request of DFO hatchery or operations staff. The third reason for lack of representativeness of broodstock screening is the tests used have inherent false positive and false negative rates (sensitivity and specificity) that affect the predictive value of these tests. It is important to note that the intent of broodstock screening in this context, is to identify and remove (or reduce) the individuals from the breeding population that can transmit certain infections from parent to offspring and not to serve as population surveillance.

Timeliness

The record keeping and reporting system made it challenging to determine the timeliness of the data. Federal diagnostic lab staff noted that it can be common that reports are telephoned or emailed to hatchery staff. This communication is inconsistently recorded and may occur at the start, end or times in between the case investigation. Errors in the records created further challenges. For example, in the FFSBC database, 13 entries recorded the final report date before the submission date. Incomplete recording was another challenge; in the FFSBC data, only 118 records had both the submission and reporting date entered. In 92 cases, there was a notation or email that showed that the lab staff emailed screening results within days of final results being ready, but that it took weeks to generate those results due to the nature of the testing and confirmation process.

Data quality conclusions

The PBS and FFSBC laboratories have data quality problems that are not unique to fish health. Their data can best be used to describe the types of problems causing increased morbidity and mortality in hatcheries, but the data do not reflect the infection status of the hatchery population as a whole or allow the generation of information on rates of disease. They rarely provided information with which to determine the infection status of hatchery populations prior to their release. The data quality is not audited or systematically evaluated.

We can conclude that the diagnostic laboratory records would not detect all disease events or pathogens resident in a enhancement facility because the criteria used to encourage submissions to the laboratory reduces the likelihood that diseases that are endemic and familiar to the hatchery workers would be submitted; and diseases causing low level or sporadic mortality would not be subject to laboratory investigation. The lack of diagnostic capacity on-site in hatcheries and spawning channels and limited fish health training of facility managers suggests the possibility exists of misdiagnosis of infectious diseases that cause low level mortality, sub-clinical infections or diseases with multiple etiologies. Neither diagnostic laboratory systematically tracked wild fish except as part of their broodstock collections, which were focussed on targeted pathogens or pathogen types.

These deficits must be interpreted within the context of the current design and intent of the fish health programs – namely as diagnostic support rather than surveillance and risk management programs. Many of the problems in data quality are not unique to these 2 laboratories. Rarely do public animal health labs invest in assessment and monitoring of their data for the purposes of surveillance, monitoring and event detection. This reflects the typical purpose of diagnostic labs to provide problem solving services rather than surveillance services.

The nature of the on-site hatchery records precluded assessment of their data quality. There was no common record keeping between hatcheries (mortality, husbandry, treatment, calculations, release records, movement, and maintaining prescription and diagnostic records). Some PIP facilities had no health records. Some DFO hatcheries did not provide us with treatment records. The nature of the data recorded and its completeness varied widely between and within the major federal and CEDP hatcheries. Husbandry and treatment records were often (but not consistently) tracked by brood year (spawn date) rather than current date. Most hatcheries did not provide daily husbandry, mortality, or release records. Some hatcheries provided copies of fish health diagnostic information performed by PBS.

Information provided to us by the Cohen Commission included a Sept 23, 2010 email from David Celli, Regional Manager, Enforcement Operations, DFO to Cindy Harlow and others in which Mr. Celli expresses concerns about the “apparent lack of capacity [of CEDP and PIP facilities] to adhere to minimum fish culture standards, including adequate documentation (records and reporting) for hatchery operations.” This indicates that DFO is aware of deficits in records keeping at CEDP and PIP facilities but we had no further information as to the follow-up to this email.

Hazards identified by reviewing hatchery and diagnostic laboratory records

Tables 13 and 14 summarize the pathogens noted in FFSBC and PBS fish health diagnostic records. These tables indicate the numbers of times a diagnostic report mentioned a specific pathogen. The diagnostic records could contain no report of an infectious disease finding, a report of a single infectious agent in one fish, or multiple infectious agents in multiple fish. Sometimes, the records indicated the proportion of fish with the infection of diagnosis, other times it did not. The number of reports per group cannot, therefore, be used to determine the prevalence of infections. They may be used as a surrogate for issues that come to the attention of and are of concern to hatchery managers who are responsible for submitting cases to the laboratories.

Our review of FFSBC records found 162 reports of pathogen isolation or diagnosis of infectious diseases amongst the records we reviewed. *Flavobacterium* , especially *Flavobacterium psychrophilum*, was the most commonly reported infectious agent (62% of reported infections; n=100/162) (Table 13). Kent (2011) found no evidence of this species or other *Flavobacterium* spp. causing death or infections in the Fraser River sockeye salmon 'in recent times' and recognized it largely as a disease of hatchery fish: But he classified *Flavobacterium* spp. as a moderate risk pathogen as *F. psychrophilium* can be a primary pathogen and he hypothesized it could cause disease in Fraser River sockeye salmon when water conditions were poor. *Gyrodactylus* was the most common parasite (n=12 reports) in the FFSBC diagnostic records. The FFSBC laboratory diagnostic records noted the first isolation of herpesvirus salmonis-1 in the Fraser River from Fraser Valley Trout Hatchery. Kent (2011) did not include this virus or *Gyrodactylus* in his risk review.

Gyrodactylus is the name for a group of external monogenean parasites that comprise over 400 species (Cone et al., 1983). Diagnostic records did not state the species of *Gyrodactylus* diagnosed. There are at least 3 species that are specific to salmonids and others can be acquired by a wide range of hosts including some salmonids. Response to treatments can vary with the parasite species (Cone et al., 1983). Treatments for freshwater fish can include saltwater baths or treatments with chemical baths such as formalin. Commonly referred to as "skin flukes" or "gill flukes", these parasites can cause irritation, excessive mucous build up and skin or gill lesions. Their presence on cultured fish can reflect overcrowding, poor sanitation and/or poor water quality conditions (Noga, 1996). *Gryrodactylus* gained prominence in debates on the impacts of fish culture because *G. salaris* was disseminated into a variety of rivers in Norway and its introduction was linked to salmon stocking programs. The introduction of these parasites was associated with reduced Atlantic salmon returns and population size (Johnsen and Jensen, 1991). In response, the Norwegian government undertook dramatic risk eradication efforts, including ridding affected rivers of fish through chemical poisoning. It must not be assumed that the same

risk applies in BC because (1) *G. salaris* has not been found in BC waters and (2) other *Gyrodactylus* species (not *G. salaris*) are ubiquitous in many BC waters. The practice of restricting the movement of enhanced fish outside of their transplant zone or local waters is believed to greatly reduce the risk of a similar incident in BC. Also, because saltwater can be an effective treatment, exposures in marine environments would be minimized.

Table 13: Pathogens or diagnostic categories described in the Freshwater Fisheries Society of BC fish health data (January 2000-July 15, 2010).

General Taxonomic Group (# reports)		# Reports	Diagnostic findings included in the group
Bacteria (n=134)	Myxobacteria	100	*Flavobacterium psychrophilum* (confirmed and presumed); *Flavobacterium;* Myxobacteriosis
	Aeromonas spp.	10	Furunculosis; *Aeromonas hydrophila;* *Aeromonas* spp.*; Aeromonas* (presumed)
	Pseudomonas spp.	8	*Pseudomonas* spp. (presumed)
	Vibrio spp	4	*Vibrio anguillarum* type 1; *Vibrio* spp.; *Vibrio* (presumed)
	Yersinia spp.	4	*Yersinia ruckerii;* Enteric redmouth
	Bacterial gill disease	8	Bacterial gill disease; Yellow pigmented bacteria
Viruses (n=4)	*Herpesvirus salmonis*	4	

Parasites (n=24)	External parasites	22	*Gyrodactylus; Epistylis; Trichodina; Trichophyra*
	Internal parasites	2	*Philonema;* Unknown cestode

Table 14: Pathogens or diagnostic categories described in the Pacific Biological Station fish health diagnostic data (January 2000-May 13, 2010 – includes 2 cases from 1998).

General Taxonomic Group (# reports)		**# Reports**	**Diagnostic findings included in the group**
Bacteria (n=355)	Myxobacteria	163	**Flavobacterium* spp., Cold water disease; Tail rot; *^Myxobacteriosis (systemic and external)
	Aeromonas spp.	33	*^Furunculosis; **Aeromonas hydrophila;* **Aeromonas sobria;* Motile aeromonad
	Pseudomonas spp.	2	*Pseudomonas* spp.; *^*Pseudomoas aeurginosa*
	Vibrio spp.	1	*^*Vibrio vulnificus*
	Yersinia spp.	1	Enteric redmouth
	Bacterial gill disease	54	*Bacterial gill disease; Fusiform gill disease

	Renibacterium	97	*^Bacterial kidney disease; *Renibacterium*
	Other bacteria	4	*^Bacteria unidentified; Mixed bacterial infection
Viruses (n=2)	Viruses	2	*^Infectious hematopoietic virus
Parasites (n=130) (126 plus 4 unknown parasites)	External parasites	81	*^*Salmonicola;* *^copepod (unidentified); *Dermocystidium; *Ichthyophthirius; *Costia; Trichodina* ; *Turbea; *Trichophyra*
	Internal parasites	45	*^*Diphylobothrium;* *^Tapeworm; *^Cestode; Parasitic worm; *^Helminth unknown; *Sphaerosphora;* *^Myxidium; *^*Parvicapsula;* *Kudoa;* ^**Loma*; PKX; *Microspordia; *^Myxosporidia; *Chloromyxum*
Fungi (n=50)		50	*Saprolegnia;* *Internal Fungus; *^External fungus; *Phoma herbarium; *^Ichthyophonus*

* indicates diagnoses made in sockeye salmon (any classification); ^ indicates diagnoses in sockeye salmon classified as "wild" or "semi-wild" in the PBS diagnostic database

Herpes salmonis is poorly described in the literature. The American Fisheries Society Fish Health Section Bluebook (AFS, 2007) reports that the virus was first isolated from ovarian fluids of moribund adult rainbow trout that suffered up to 50% post spawning losses, but that it has also been isolated from apparently healthy fish. The role for this virus in causing disease under natural conditions remains unclear but pathology can be induced with experimental infections (AFS, 2007).

Myxobacterial infections, which include flavobacteria, were the most common bacterial diagnoses from the DFO diagnostic reports (46%; n=163/355) and were responsible for 30% of the 537 reports of infectious and parasitic diseases. The DFO data included 97 reports of bacterial kidney disease (BKD) – 27% of their bacterial diagnoses. This may reflect the active screening DFO does for this disease. Kent (2011) classified BKD as a high risk disease because sockeye salmon are particularly susceptible and the disease progresses when smolts enter seawater. There was insufficient history in the fish health records to allow us to correlate a fish's (or group of fish) history with its disease status, but the cases of *Ichthypotherius* and *Parvicapsula* were from returning adult fish. Because *Ichthypotherius multifiliis* and *Parvicapsula minibicornis* have been associated with high level pre-spawning mortality in sockeye salmon, Kent (2001) classified them as high risk infections. The fungal infections caused by *Saprolegnia spp.* and *Ichthophtherius hoferi* were classified as moderate risk by Kent (2011), however he noted that the former was prevalent in the environment and the latter had not yet been recognized in sockeye salmon. Proliferative kidney disease (PKX) was also classified as a moderate risk pathogen.

Two pathogens deemed by Kent (2011) as high risk– Furunculosis and *Vibrio anguillarum* - were diagnosed in FFSBC and DFO hatcheries. Five percent of reported diseases for FFSBC hatcheries were furunculosis and *Vibrio anguillarum* diagnoses made up 1% (with an additional 1% presumed to be this pathogen). For DFO, 5% of reports were for furuncluosis and but no *V.anguillarum* was reported. The finding of *V. anguillarum* at the FSBC should be interpreted with caution because *V. anguillarum* is typically associated with marine environments; the FFSBC tests only fish from freshwater; and interviews with lab staff indicated concerns over the reliability of the diagnostic test used to detect *Vibrio*.

As noted in the literature review, the accuracy of the Kent (2011) risk categories has not been validated. Kent noted the problems in trying to extend these risk categories (largely based on experimental and fish culture experience) to impacts on wild salmonids or wild Fraser River sockeye salmon. Moreover, Kent did not take into account the seasonal occurrence of these diseases in hatcheries and whether or not wild fish would be in an exposure pathway when the diseases were present in the hatchery.

Of the data used to generate Table 13, 82 cases were from sockeye salmon and are denoted by the asterisk in the table. Two-thirds of these reports (66%) came from 2 facilities (40 cases from Rosewell and 15 from Cultus Lake). Chilliwack Hatchery provided 10, Inch Creek 7, Weaver Creek 5 and Nadina River facilities 5 submissions. Of the 82 case reports for sockeye salmon in the PBS database, 48% were for investigations of some loss (mortality event) or disease issue, 15% for BKD or viral screening, and 17% for health checks. The most common diagnosis in sockeye salmon was BKD (8 reports) followed by *Parvicapsula* (7 reports) and *Salmonicola* (7 reports). All other diagnoses provided less than 5 reports each. While *Parvicapsula* has been found in Weaver Creek sockeye salmon, it is believed that the parasite is acquired in the estuary of the Fraser River as opposed to a freshwater source further upstream (Jones et al., 2003). Two internet references provide additional information of pathogens in enhanced Fraser River sockeye salmon, but did not expand the hazards list:

- A 2009 report stated that BKD has been increasing in incidence in captive Cultus Lake broodstock (CSAS, 2010).
- A 2003 COSEWIC report on Cultus Lake sockeye salmon stated that this endangered group was affected by pre-spawning mortality associated with *Parvicapsula* and early freshwater migration since the 1990's (COSEWIC, 2003)

Of the 1040 fish health records dealing with diagnosis and/or purpose for treatments from CEDP and PIP facilities; 978 were from CEDP facilities, 62 from PIP. Review of these facility level records did not add any new pathogens to our hazards list except for one record of *Piscirickettsia*. Most commonly, the diagnosis was not determined or not given (n=474 records). Next most common were clinical diagnoses of fin or tail rot and flashing (n= 120 and 90 respectively). These signs can be associated with external parasites or diseases like myxobacteriosis. Myxobacterial infections were mentioned in 89 of the hatchery records and fungal infections (unidentified species) were mentioned in 35 records. Because of the lack of consistent case definitions and the presence of more than 1 diagnosis per record, we could not confidently itemize the proportion of diagnoses for these records.

Spawning channels

Spawning channels produce the majority of sockeye salmon from salmonid enhancement in British Columbia (Fig.1). Diagnostic reports from spawning channels were rare in the data we received (Appendix 7). Two reports of *Cryptobia* spp. were found in the diagnostic data provided to us. While attributed to Weaver Creek and Cultus Lake, the source of these fish was unclear in the report (fish were classified as wild). Other pathogens found in wild or semi-wild sockeye salmon are indicated in Table 14.

We founds 2 reports from the Oceans, Habitat and Enhancement Branch (DFO) that presented the results of *Ichthyophthirius* spp. and *Loma* spp. screening on fish gathering at 2 northern BC spawning channels and the Nadina River spawning channel. *Ichthyophthyrius* spp. levels were higher in 2008 than 2005-06 samples in the Nadina system, with 2008 ranging from 75-100%. The higher 2008 levels were attributed to early returns and high loading of the spawning channel which resulted in higher densities for a longer time, which facilitated the parasite transmission. *Loma* spp. prevalence ranged from 0-28% positive samples. Escapement levels were higher in 2008 than in the 3 previous years (Donas, 2008).

DFO no longer routinely screens for IHNv due to the poor correlation between screening outcomes and subsequent diseases. Garver (2010) summarized historic long term monitoring (1986-2009) of 3 pathogens in spawning channels; IHN, *Ichthyophtherius multifilis* and *Parvicapsula minibicornis*. This summary included results reported by Traxler et. al. (1989) which described a 50% loss of Weaver Creek sockeye salmon due to IHN within days of leaving Weaver Creek in 1987. The only other large scale IHN die-off reported by Garver (2010) was one in Chilko Creek in 1973. IHN levels have varied within and between stocks in the monitored populations in Weaver Creek and Nadina River. IHN prevalence during that time has ranged from 0-80% per year. Garver reports that IHNv has not been recovered from Weaver Creek or Nadia River for the past ten years and 16 years respectively.

Within these same monitored populations, *Ichthyophythirus multifilis* prevalence has not changed in the past 10 years. This pathogen has been associated with increase pre-spawning losses (Weaver Creek in 1995; 30% loss) and in Nadia River in 1978, 1987, 1995 and 2008 with losses ranging from 25-70% (Garver, 2010). Garver, (2010) described these relationships only as associations and did not attribute the losses directly or solely to the pathogen. His conclusions were consistent with the data Traxler et al. (1988) presented for a spawning ground *Ichthyophthyrius multifilis* outbreak.

Within the data provided to us, there were 17 surveys for *Parvicapsula minibicornis* in sockeye salmon. These surveys were generally conducted in the Strait of Georgia or near the mouth of the Fraser River and not in enhancement facilities. This parasite was first described in Weaver Creek sockeye salmon by Kent (1997) and has been the topic of surveys in waters before spawning grounds (St. Hilaire e. al., 2002). Bradford et al. (2010) looked at sockeye salmon caught for broodstock for Cultus Lake and provided evidence that *Parvicapsula minibicornis* can cause prematurity mortality in spawning salmon as well as evidence that pre-mature mortality is affected by more than one factor. Additional information on this parasite is provided elsewhere in the literature review.

Hazard assessment summary

The data collected on diseases in enhancement salmonids does not allow for a complete hazards list to be developed or for an estimate of the frequency and abundance of infection across the entire enhancement fish population. The finding of only 2 reports of IHN in the PBS diagnostic records (Table 14) seemed surprising to us as DFO does have a practice of doing some IHNv screening (as was indicated in some of the facility level records we reviewed). This further emphasizes our caution to not use these findings to determine prevalence of infections in hatcheries.

The nature of the diagnostic system restricts our knowledge to the more common infections that are capable of causing overt clinical signs in a sub-section of the population as well as the presence of a small number of pathogens in returning broodstock. The data did reveal that a variety of pathogenic hazards do exist in enhanced salmonids; none of which were unexpected or exclusive to enhanced salmonids. Enhanced salmonids do harbour viruses, bacteria and parasites capable of causing severe clinical disease in infected fish under experimental or culture conditions.

Exposure assessment

The purpose of the exposure assessment is to describe, estimate or quantify the probability that Fraser River sockeye salmon are exposed to the hazards of concern. There remain major gaps in our understanding of disease transmission pathways and conditions that facilitate transmission of fish pathogens because most research has been done under laboratory conditions. For example, we do not know how close a naive fish must be to an infectious fish and the necessary length of that exposure under natural conditions to result in the effective transmission of infectious agents. We can reasonably assume that environmental conditions will affect the probability of an effective exposure because we know that the survival and viability of a pathogen can be affected by environmental conditions. Water quality (temperature, salinity, pH, turbidity etc.) and aquatic ecology (presence of intermediate hosts) are examples of environmental variable that will affect pathogen survival and thus opportunities for environmental survival (Stoskopf, 1993). The immune state of a fish (which is affected by factors such as age, nutritional status and stress) will also affect the probability that exposure to a pathogen will result in an infection (Stoskopf, 1993).

Exposure possibilities associated with enhancement facilities

Exposure of Fraser River sockeye salmon in enhancement facilities

On average, 46 million sockeye salmon were released per year from enhancement facilities between 2005 and 2009 (range= 37 and 63 million (See Appendix 5 and 6). Weaver Creek Spawning Channel had average annual releases of 27.7 million sockeye salmon, followed by Nadina River Spawning Channel (5.8 million average). Shuswap River hatchery, Inch Sockeye Satellite, Gates Spawning Channel and Horsefly Spawning Channel released a combined annual average of 11.6 million sockeye salmon between 2004 and 2009. In 2009, 12 million sockeye salmon were released, as opposed to average releases of 46 million annually. This was mostly due to the decrease in sockeye salmon released from Weaver Creek Spawning Channel.

We can conclude that a proportion of Fraser River sockeye salmon have been exposed to pathogens associated with enhancement facilities because (1) a portion of the Fraser River sockeye salmon population is derived from hatcheries and spawning channels and (2) our hazards assessment and literature review found pathogens in those operations.

We were not provided with data on the rates or distribution of infections within cultured sockeye salmon populations. There were also no data provided to determine how an infection present and/or propagated in a sockeye salmon enhancement facility would be transmitted (or not) to Fraser River sockeye salmon not reared in an enhancement facility. We found no studies apart from Traxler et al.'s (1989) work on IHNv in the Weaver Creek spawning channel that followed enhanced Fraser River sockeye salmon post-release to determine how long infections may be carried outside of the enhancement facility (see literature review for further discussion). Spawning channels are substantially different than hatcheries in that managers have less influence on the life history of the fish apart from restricting their entry into the spawning channel and regulating water flows, unlike the conditions for hatchery rearing.

Release/escape of pathogens from enhancement facilities

FFSBC fish health staff were aware of two possible scenarios where fish with infections were released into fish bearing waters. The first scenario was when salmonids could be released still infested with the external parasite *Gyrodactylus* spp. as they may have been released soon after their treatment ended but before the fish could be screened for the parasite. In another scenario, anecdotal evidence suggested some fish were released with skin lesions, presumably due to *Flavobacterium* spp. infections. This could not be confirmed as the lesions were detected by fishermen on stocked fish and were not submitted for diagnostic investigation.

PBS fish health staff interviewed for this review stated that the goal is to not release sick or infectious fish but they were aware of 3 situations where this goal was not met. They reported;

1. Rare after-the-fact reports of fish demonstrating clinical signs of external parasite infections (*Trichodina* and *Costia* diagnosed on site by hatchery staff) just prior to their scheduled release, subsequently released by hatchery staff on the assumption that exposure to saltwater would be an effective treatment.

   These release situations were not documented in our review of the PBS diagnostic lab records, however they were reflected in our review of on-site hatchery records.
2. Hatchery reared fish are sometimes held for 3-4 weeks in seawater netpens prior to release. There have been verbal reports of fish being released early due to increased rates of death or clinical signs due to suspected Vibriosis.

   We were able to substantiate such a case in the PBS diagnostic records as well as document this practice in a Fish Health Management Plan
3. The historic and current control plan for *Renibacterium salmoniarum* (the cause of bacterial kidney disease – BKD), allows for the rearing of fish known to harbour the pathogen. This is an endemic disease and there are chronically infected populations in federal hatcheries. The DFO has a specific plan for managing this disease that uses broodstock screening and population segregation as foundations for the control program. The control program consist of the following components:
   3.1. If less than 25% of 60 fish opportunistically gathered in their last 2 weeks before release test positive for the disease (using direct fluorescent antibody testing [DFAT]), the population will be deemed okay for release. *We did find two cases in one hatchery's health records describing that fish were released when 45% and 33% were BKD positive*.
   3.2. Not all groups of fish are tested; only those that had (1) disease outbreaks as juveniles, (2) were from broodstock with significantly high levels of the disease or (3) showed poor performance (defined as mortality rate $\geq$5% for any disease in 90 days at least 1 month prior to release).
   3.3. Broodstock are screened for BKD. The BKD management plan allows for eggs from low positive broodstock to be reared to unfed fry stage and released; eggs from moderately infected broodstock to be outplanted in waters downstream from the hatchery inflow and, where conservation concerns are high, to outplant the eggs of broodstock classified as high positive (note – the latter case requires consultation with hatchery, enhancement and fish health staff). The release of eggs is significant as this pathogen can be carried in eggs of infected broodstock and propagated in their offspring. This management approach has been successful in reducing BKD prevalence in hatcheries in the United

States (Munson et al., 2010), but it does allow for fish with low levels of infection to reproduce, and their offspring to be reared and released. It also allows for distribution of potentially infected eggs into streams and rivers. Success of this approach can vary with variations or inconsistencies of other management variables (Munson et al., 2010).

BKD is one of the most studied diseases of Pacific salmon. It has been shown that the progeny of broodstock that are "high positive" do have increased BKD when they get to seawater, are at increased risk of predation and can be more susceptible to other diseases (Munson et al., 2010). The BKD management plan seems designed to first reduce the level of the disease in hatchery fish and second, reduce the risk of transmission to wild fish.

Our review of the PBS diagnostic laboratory records found a small number of additional cases that suggest or show that fish have been released with infections:

- Release was one option given for management of some hatchery coho salmon with myxobacteriosis and myxobacterial gill disease; along with the opinion that predators would kill the infected fish (Capilano, 2000);
- Treatment was not recommended for fish with myxobacteriosis as they were destined for release in 2 months (Spius, 2004);
- Fish that had enteric redmouth (*Yersinia ruckerii*) infections were released or escaped (unclear in the records)from a freshwater pen in a lake in the Okanagan (Oxbow Lake, 2003);
- A report noted that a group that included fish with furuncluosis, dermocystium infestation and myxobacteroisis would be released early (but no details on when the release would occur or interventions before the release were provided) (Spius, 2003);
- A group of fish were transferred to a seawater netpen with myxobacteriosis and minor bacterial gills disease (Quinsam, 2000);
- The reported history of one case lacked detail but indicated that fish were being considered for transfer to a lake. These fish had laboratory findings suggestive of furuncluosis; their fate was not recorded and thus the transfer may not have happened (Puntledge, 2001).

Vibriosis, bacterial kidney disease and furunculosis were classified by Kent (2011) as high risk; myxobacteria (Flavobacteria) as moderate risk; and democystium as low risk. Enteric redmouth was not on his list. It is important to note that there was no data field in the diagnostic records to report the outcome of a disease nor the fate of the fish involved (released or not) and we lacked the data to link the date of diagnosis and release of specific groups of infected fish. Therefore, we cannot evaluate how often fish were infected and treated (or not) within a period close to their release from the diagnostic records. Nor could we substantiate that Fraser River sockeye

salmon were in proximity to the infectious fish when they were released from enhancement facilities. Data for the FFSBC was similar in that they were not set up to record whether or not the fish were close to release when diagnosed.

Our review of the DFO major hatchery, CEDP and PIP hatchery records found 17 reports of fish being placed into fish bearing waters (released into streams/rivers or moved to lake or sea netpens) with known infectious diseases, suspected infections or clinical signs of undiagnosed disease. In some cases, the fish were given a chemical treatment (e.g. formalin or chlormaine T) and released 3-10 days later without records verifying the treatment was effective. In other cases fish were moved directly to lake netpens or released. Most often, these releases involved myxobacterial infections or fish with symptoms consistent with myxobacteriosis. One case involved suspected but unconfirmed furunculosis. One lake netpen of sockeye salmon were diagnosed with IHN but destroyed and not released from the pens.

Myxobacteria-associated diagnoses were common in the review of hazards present in the various diagnostic records as well as being the most common infection associated with release of fish from enhancement facilities. The term Myxobacteria is non-specific and refers to a family of bacteria. The nomenclature of these pathogens has evolved over the past few decades. The Flavobacteria (*F. psychrophilum, F. columnarae* and *F.branchiphilum*) are now recognized as the causes of the myxobacterial diseases, coldwater disease, columnaris disease and bacterial gill disease respectively. These bacteria are presumed to be ubiquitous in freshwater aquatic environments. There is some evidence that *F. psychrophilum* can be transmitted from parent to offspring but the main routes of exposure involve contaminated water and equipment (Starliper, 2011). Sick and dead fish can shed very large numbers of bacteria into the water. Carrier fish and long term (months) shedding suggests that infected fish can contaminate their environment for prolonged periods. The association of overcrowding, poor water quality and horizontal transmission within facilities by personnel and equipment (Starliper, 2011) may be an explanation for motivations to release fish with this infection – with the assumption that release will remove these predisposing factors. Antibiotic treatments, eggs disinfection, rapid recognition and removal of dead fish, improved husbandry and prevention of introduction and transfer of the pathogen are all part of a myxobacterial management strategy.

There remain three critical unknowns regarding risks associated with Myxobacteria; (1) does treatment allow for removal of the bacteria from the population to below or similar to background levels and (2) will fish shedding the bacteria after release increase the exposure of non-enhancement fish to levels greater than background exposure and (3) are ambient environmental and host stressors sufficient to allow a free-ranging fish to develop disease if exposed to these bacteria.

Pathogen movement with fish movement

Fish movements create the risk of pathogen movements (Fenichel et al., 2008). There are many examples of the spread of disease with the movement of wild and domestic animals, including fish (Fevre et al., 2006; Fenichel et al., 2008). There are three types of movements relevant to this project (1) the movement of fish after release from hatcheries or spawning channels; (2) the transportation of fish from a hatchery to a distant receiving water and (3) the transfer of fish between enhancement facilities.

Annual release numbers for sockeye salmon and other salmon species from federally supported facilities are variable but number in the tens of millions per year (Appendix 5).The FFSBC stocks nearly 900 lakes in British Columbia with fish produced from 5 facilities. Between January and May 2011 alone over 1 million salmonids were stocked in lakes in the Lower Mainland, Vancouver Island and Thompson-Nicola regions (http://www.gofishbc.com/r3.htm). The FFSBC has some opportunity to hold the fish until they are apparently healthy as they are not under the same pressures as DFO facilities to time their releases with natural migratory patterns. The FFSBC truck their fish from the hatchery to the release site. FFSBC fish health staff reported that none of their kokanee releases would involve sockeye salmon bearing lakes and that they choose wild broodstock from lakes that do not have sockeye salmon to avoid IHNv. We lacked the time to confer with FFSBC biologists who determine release strategies and locations to compare FFSBC releases against freshwater habitat for Fraser River sockeye salmon.

Carey (2005) reported that 32% of requests to transfer fish in Canada were for fisheries enhancement or habitat compensation purposes. The Federal-Provincial Introductions and Transfers Committee advises agencies on fish movements in BC. It assesses disease, ecological and genetic risks associated with proposed fish movements. The committee has little involvement if fish are moved within the same zone in the province. The Fraser River drainage, although covering a very large area, is considered one zone. The Southern Coast Zone covers Vancouver Island and the Strait of Georgia (Figure 3). A Form A licence is required to move salmonids within a zone. Fish can be moved if they have a satisfactory health status (including lack of clinical signs of disease) and no emergency diseases have been identified in the stock to be transferred. Within zone transfers do not require pathogen screening. Therefore “health status” is not determined through pathogen screening. Much of the movement between enhancement facilities would be considered within zone transfers. The SEP provides a federal enhancement facility with a blanket license issued every 3 years that allows surface disinfected eggs and fish to be moved between facilities given the conditions above plus acceptable mortality rates (note that mortality rates is not a printed stipulation on the permits provided to us) (Personal communication, Mark Higgins, 2011, DFO). The movements can be halted if the

Introductions and Transfers Committee become aware of significant disease issues that could be related to the proposed movement. CEDP and PIP facilities do not require a license to release fish from a community hatchery into their local stream. If the licensed hatchery changes its management plans such that new movements are required, it must submit the new movement plan to the Introductions and Transfers Committee for review.

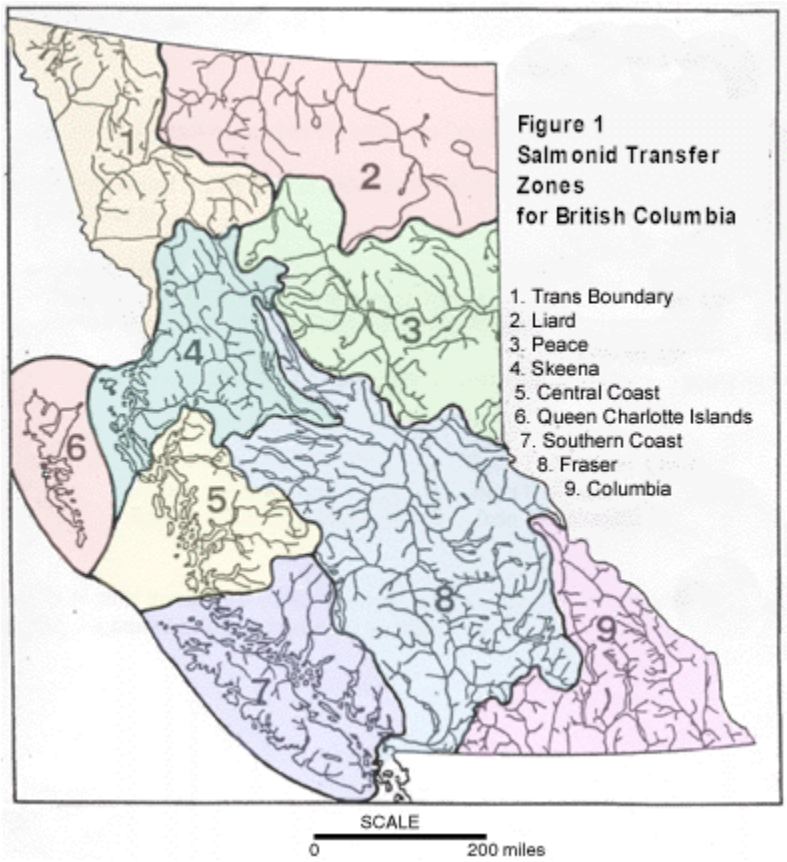

Figure 3: Transfer zones used to assess and licence salmonid movements in British Columbia (http://www.dfo-mpo.gc.ca/aquaculture/regions/pac/application-demande-eng.htm).

DFO has a practice of acclimating salmonids to seawater by short term holding in netpens. DFO records indicated that 6 major facilities and 21 community facilities or sites use seawater or brackish water netpens in BC. We found in the production records sent to us, records from 4 DFO major facilities and 5 CEDP facilities that released salmonids from ocean netpen sites. Records showed that PIP facilities also release from netpens but we could not determine the number of PIPs that use this practice. At least 3 species spend a portion of their lives in seawater netpens. Release data for the study area for this review between 2005-2009 found; 10,531,364 chinook salmon subyearling smolts; 102,419 chum salmon (*Oncorhynchus keta*) fed fry (2009 only); and 5,218,683 pink salmon (*Oncorhynchus gorbuscha*) fed fry were released from

seawater netpens. In 2009, over 4 million chinook and chum salmon were transferred from 7 hatcheries to marine netpens (Figure 4). Some facilities also hold fish in lake pens prior to their release but data were not available to enumerate this practice. Movement to netpens is considered a within zone transfer. In Figure 4, the release waterbody is the site of the netpens. Our findings above demonstrated that fish with known or unknown infections have been moved to netpens. We know of no studies in BC of the frequency of interaction of wild fish (salmonid or non-salmonid) with these netpens and thus the possibility for transfer of infectious agents. The relatively short period of residence in netpens (3-6 few weeks for sea netpens) reduced this exposure time; however, the fish are released from the netpens into fish bearing waters. We found no follow-up studies of the infection status of netpen fish prior to their release.

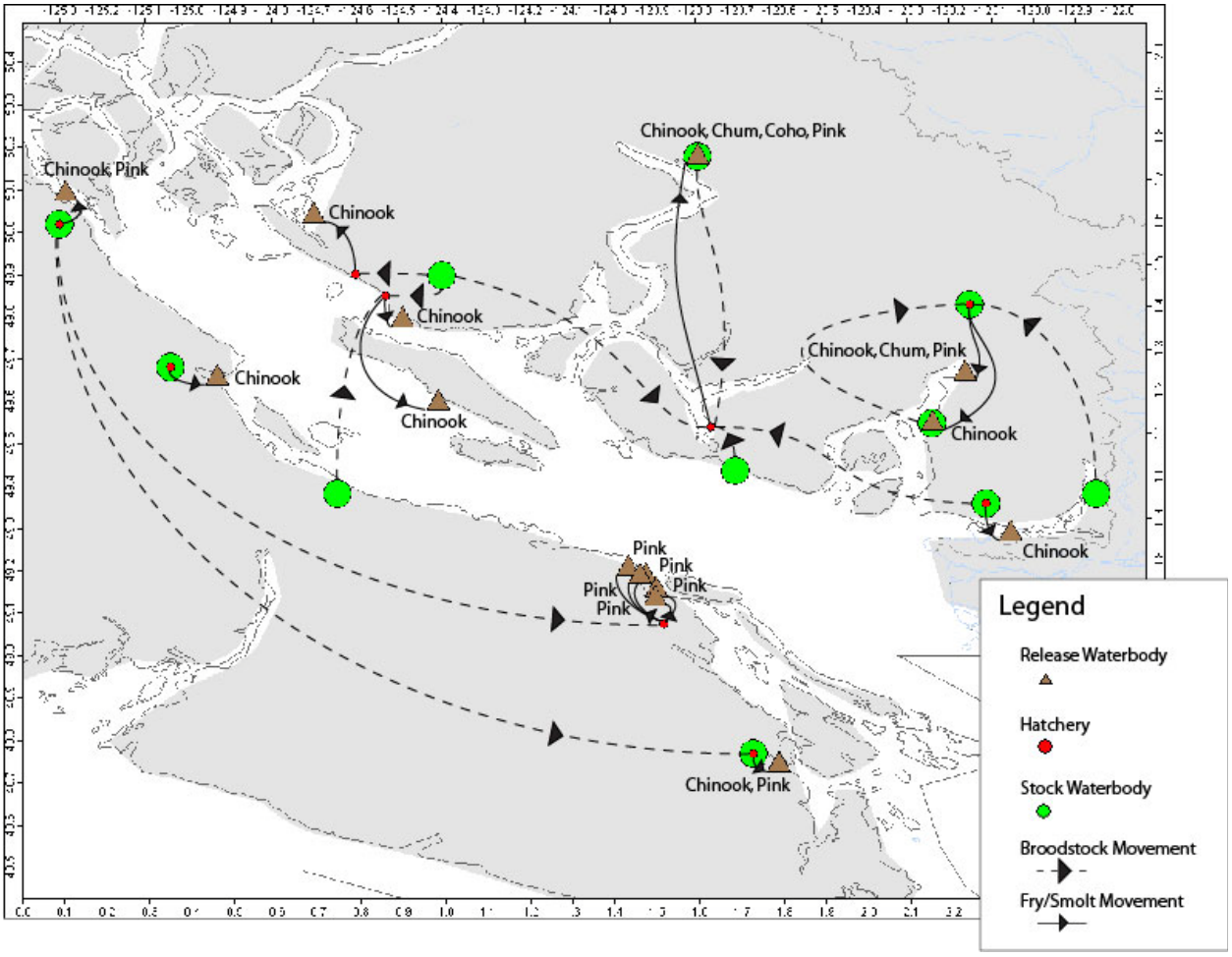

Figure 4: Movement of netpen-released salmonids between stock waterbody, hatchery and release waterbody, 2005-2009 based on production data provided by DFO to the Cohen Commission. (Where a red dot overlays a green waterbody marker, stock are raised at the hatchery associated with the same river from which broodstock originated)

The movement of eggs or fish that are donated from major DFO hatcheries for school projects within their watershed in another example of a within zone transfer done under the hatcheries' movement licence. The disposal of salmon carcasses as part of stream enrichment projects is not under the authority of the Introductions and Transfers Committee as it does not involve live fish but the committee did send advice to federal regulators recommending that carcasses only be deposited in local streams (Personal communication, 2011, Mark Higgins, DFO). If a hatchery becomes involved with fish movements for a research project that is not part of their 'day-to-day' business, they must apply for a new movement license.

If salmonids are to be moved between zones, they must undergo a health check wherein 60 fish are killed and tested for "Schedule II" pathogens as described in the federal Fish Health Protection Regulations. These pathogens include:

> Any filterable replicating agent capable of causing cytopathic effects in the cell lines of fish specified by the Minister including, but not limited to:
>
> 1) Viral Hemorrhagic Septicaemia (Egtved) (Egtved virus, VHS)
> 2) Infectious Hematopoietic Necrosis (IHNV)
> 3) Infectious Pancreatic Necrosis (IPNV)
> 4) Whirling Disease (*Myxobolus cerebralis*)
> 5) Ceratomyxosis (*Ceratomyxa shasta*)
> 6) Furunculosis (*Aeromonas salmonicida*)
> 7) Enteric Redmouth Disease (*Yersinia ruckeri)*

We could not confirm that all of these agents are tested for in all between zone transfers due in part to the lack of details in the diagnostic records. The only routine between zone transfer found was between Cultus Lake and Rosewall hatcheries. Disinfected sockeye salmon eggs from BKD negative broodstock are moved from Cultus Lake Hatchery to Rosewall Creek Hatchery. The eggs are subsequently reared at the Rosewell Hatchery for research, as broodstock or as fry or smolts that are returned to Cultus Lake. Before the fry or smolts are transferred, a sample is supposed to undergo Schedule II testing (Personal communication, 2011, Mark Higgins, DFO). We did not find any mention of tests for whirling disease.

There is no requirement for post transfer follow-up after a Schedule II assessment and thus no ability to determine if they later developed infections or disease. There is some assurance that other agents can be detected by the diagnostic methods used for Schedule II testing. For example, one screening of sockeye salmon moved from Cultus Lake Hatchery to Rosewall Hatchery in 2003 found *Salminicola*, an unknown parasite worm, Myxidium and tapeworm pleuroceroids in a pre-transfer assessment. These fish were recommended for transfer but we had no records of their subsequent movements.

The Introductions and Transfers Committee has records of their decisions and Mr. Higgins indicated that there would be data to recreate the immigration and emigration patterns of between hatchery transfers, but this had not been done. He noted problems in linking the diagnostic and other data at PBS with information on population movements held in Vancouver in the Oceans, Habitat and Enhancement Branch.

Opportunities for interaction between infected and uninfected fish outside of enhancement facilities

Sixty-five percent of DFO hatcheries, 47% of CEDP hatcheries and 75% of PIP operations are located within the Fraser River watershed and Strait of Georgia. Appendix 3 locates DFO hatcheries, DFO spawning channels, community hatcheries, community seapens and enhancement projects within the Fraser River watershed and Strait of Georgia. Salmonid enhancement facilities are found throughout the range of the in-river and coastal Fraser River sockeye salmon, with a concentration in the Lower Mainland, lower Fraser Valley, and east coast of Vancouver Island. Appendix 3 documents that tens-of-millions of enhanced salmonids are released on an annual basis. Nielsen (2003) stated that it is inevitable that released hatchery salmon will interact, in the marine and freshwater environment, with wild fish. We could, however, find no studies that documented how sockeye salmon in the Fraser River and Strait of Georgia interact with fish released from enhancement facilities. The following is an overview of life history and salmonid release data to examine if it is reasonable to assume Fraser River sockeye salmon could be exposed to enhanced salmonids.

The strategy of the SEP program has been to mimic the life history characteristics of each salmonid species to integrate both the naturally-produced and hatchery produced portion of the target wild stock (MacKinlay et al., 2004). To facilitate this, hatcheries preferentially use local broodstock during the entire natural spawning period; smolt releases are timed to coincide with natural migrations; and smolts are released “at a similar weight to the best-surviving naturally-produced migrants, so that they migrate quickly and avoid freshwater interactions" (MacKinlay et al., 2004). DFO release data allowed us to examine the lifestage and timing of fish releases from enhancement operations (Appendix 6). Fish are released as unfed fry, fed-fry, channel fry,

sub-yearling smolts and yearling smolts; release time varies with the species, and whether they are reared in spawning channels, hatcheries and/or netpens.

We were informed by DFO that fish releases for a specific cohort may start prior to the recorded date (from days to weeks), but the recorded date in the production data we were given represented the very last day that the last fish from that cohort were released. We combined the release dates by week and month to generate the total number of releases within a 7 day (or 30 day) time period, and used these data to create Figures 5 and 6. Release timing from DFO hatcheries for the years 2005-2009 showed a distinct bell-curve with the highest releases occurring in April, May and June for DFO facilities (Figure 5). CEDP facilities show a similar

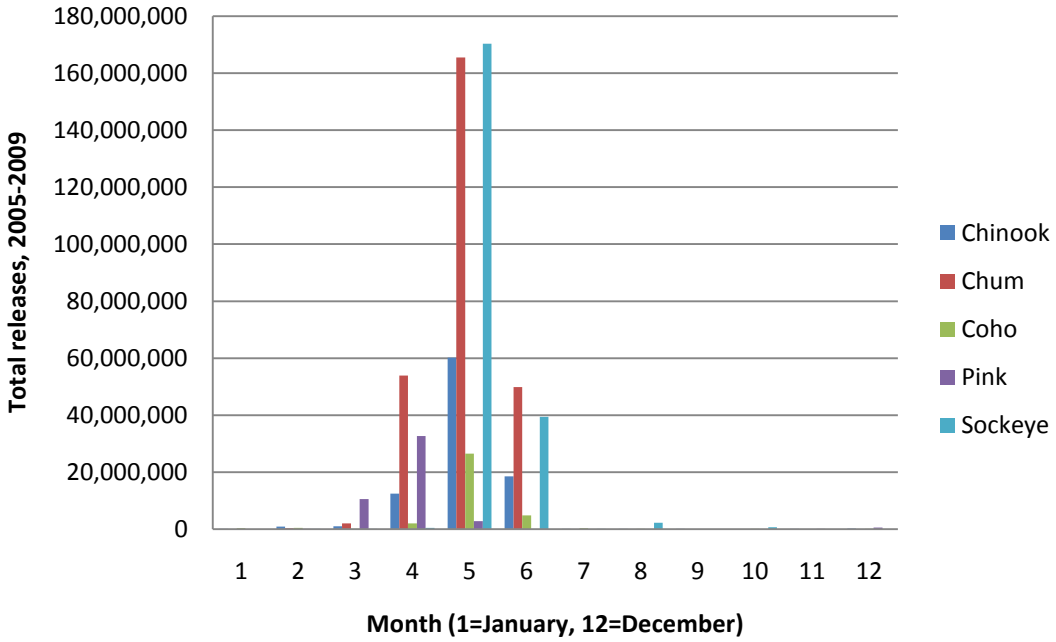

Figure 5: Combined monthly total DFO facility salmonid releases from the Fraser River watershed and Strait of Georgia, 2005-2009 inclusive.

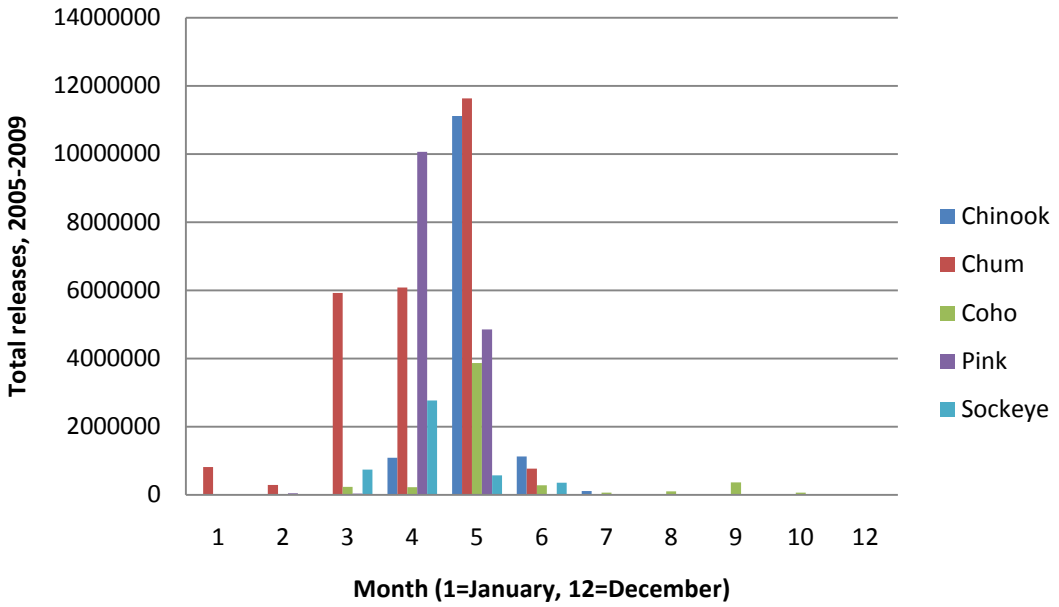

Figure 6: Combined monthly total CEDP facility salmonid releases from the Fraser River watershed and Strait of Georgia, 2005-2009 inclusive.

trend, although with high chum salmon releases in March and fewer releases in June (Figure 6). Much smaller releases do occur throughout the year. Release data for FFSBC hatcheries is presented in Appendix 5.

A comparison of the release data with information on sockeye salmon life history suggest that juvenile sockeye salmon will be in the Fraser River and Strait of Georgia at similar times to when Pacific salmon are released from federal hatcheries. A comprehensive review of sockeye salmon life histories can be found in Burgner (1991); a brief summary specific to the Fraser River population is provided here. The sockeye salmon enter the Fraser River over nearly three months from late June through September. In June and July they travel at rates of up to 51 km/day to Bowron, Quesnel and Stuart Lakes some 630-970 km from the mouth of the Fraser River. A second peak occurs in August, with salmon traveling at speeds of up to 35 km/day for distances of 550-885 km (e.g. to the Chilko and Stellako rivers). In September they travel the 386 km to Adams River at rates of up to 37 km/day. Spawn timing along the Fraser River system is likewise variable, and occurs later in the warmer incubation environments (e.g. Cultus Lake and Harrison Lake). Within the Fraser River watershed, smoltification tends to occur after one year of residence and development in nursery lakes, and downstream migrations from most lakes begin when spring water temperatures rise above 4.4^0C. These migrations are completed before water temperatures reach 10^0C, although year-to-year variation in threshold temperature

for migration does exist (Burgner, 1991). A precise time-frame for the start and duration of the sockeye salmon out-migration was not found, but it occurs in the spring (Burgner, 1991).

In the marine environment, salmon abundance-surveys from Russia, Alaska and BC have caught varying proportions of different salmon species with a single trawl or seine attempt (Karpenko et al., 2005; Orsi et al., 2008). Orsi et al., (2008) reported catch statistics for both hatchery and wild chinook and coho salmon. Unfortunately, because these authors reported these numbers by month and not by seine attempt or location, there is not enough information to place the hatchery and wild coho at the same place and time. It is evident from the publications and reports we have reviewed that life history, migration patterns, and diet and habitat preferences (e.g. near shore, off-shore, depth in water column, salinity preferences etc) can be highly variable between the different salmonid species, and even between life stages within a species (Orsi et al., 2008). However, when the above information is taken together, it is biologically plausible that commingling of enhanced salmonids and Fraser River sockeye salmon in river, estuarine and marine environments could occur, but the extent to which this occurs cannot be commented on at this time.

We found no data on niche or habitat overlaps between sockeye salmon and enhanced salmon; or if the temporal co-occurrence in the same waters is sufficient to result in the exchange of pathogens between sockeye salmon and other enhanced fish. We also lacked the time and data to correlate the seasonal movements of Fraser River sockeye salmon with the dates of diagnosis of diseases within hatchery fish. It is reasonable to assume that there were cases of disease occurring in enhanced salmonids long before their release and/or that pathogens released with waste water occurred at a time when Fraser River sockeye salmon would not have been in the vicinity. Therefore, not all case reports described in our hazard assessment could have resulted in a Fraser River sockeye salmon exposure.

Waste water release

The release of water or wastes contaminated with pathogens forms another plausible exposure route. The scientific literature and standard textbooks indicate that a relatively large number of fish pathogens are transmitted in water (Table 15). There are case studies supporting the conclusion that some fish pathogens can remain viable in the water for relatively long periods outside of a fish host under natural conditions (Stephen et al., 2007; Stephen and Iwama, 1997).

Physical barriers may be insufficient to prevent the movement of waterborne pathogens from salmonid enhancement facilities to fish bearing waters. Case studies described in the literature review have documented the movement of pathogens from river systems into hatcheries. The survival of fish pathogens in water can vary tremendously with environmental conditions; some

can last for hours-days, some for weeks-months and others are common water organisms (Stephen et. al.; 2007; Stephen and Iwama, 1997; Stoskopf, 1993). The presence of organic matter, water pH, salinity, temperature and other abiotic variables are known to affect the survival of a number of important fish pathogens (Stephen and Iwama, 1997). Given that the movement of contaminated water and invertebrate vector species or intermediate hosts has been nominated as the most common mechanisms to spread non-endemic parasites (McIntyre., 1996), water movements cannot be eliminated as a possible exposure pathway. Water movements could include the movements of water with transport of FFSBC fish or the release of water from hatcheries and spawning channels into fish bearing waters like streams and rivers.

Spawning channels are designed to allow ambient water to directly enter fish bearing waters. We did not have access to operating plans that described the sources and discharges of water from the hatcheries in the study area. We did not have access to facility plans for all hatcheries but we did have access to plans submitted for Big Qualicum, Little Qualicum and Rosewall Hatcheries as part of their aquaculture license under the Pacific Aquaculture Regulations. Big Qualicum and Little Qualicum Hatcheries effluent from pond vacuuming is, "discharged directly to the fishway untreated." Little Qualicum Hatchery's "effluent from annual spawning channel cleaning is directed to a settling field and outflow is monitored to maintain particulate levels less than 2.0 NTU's." At Rosewall Hatchery: "effluent from pond vacuuming for 95 % of the site through

Table 15: Examples of transmission pathways for selected pathogens found in salmonids (Stephen et al., 2007).

Pathogen/ Disease	Waterborne	Direct contact	Indirect (fomites)	Vertical	Intermediate host or vector
VHS	X	X	X	Hypothesized	
IHN	X	X		X	X
Salmonid herpesvirus	X	X		Hypothesized	
BKD	X	X		X	
Furunculosis	X	X			
Columnaris	X	X			
PKD	X				X
Gyrodactylosis	X	X			

(X indicates this route of transmissions has been established in the literature)

drum filter prior to release to a settling swamp (sic)." The guidelines do not state what occurs with the remaining 5%, nor does it state what occurs with the sludge from the settling swamp. "Sediments from channel and river cleaning operations are passed down the river system" at the Big Qualicum site. Operating procedures do allow for the discharge of water delivered drugs and chemicals used for fish disease treatment (such as formalin) into surface waters assuming dilution before discharge. The public health and environmental impact of these actions has not been documented for a salmonid enhancement hatchery context.

When examining the risk of fish diseases moving with water from a lake into a river in North Dakota, the US Geological Services concluded that the risks of biota transfers were relatively high if implemented via open conveyance (e.g., open canal) or contained conveyance serving to divert "raw" water (e.g., piped, but untreated water) (Linder et al., 2005). In her talk provided to the State of the Salmon meeting, Bartholomew (2010) noted better outflow management and disinfection as a missing component of hatchery risk reduction actions. This included improved monitoring of outflow and settling ponds for pathogens as well as outflow treatment. There is a variety of federal and provincial legislation that deals with waste discharges into fish bearing water. The review of these regulations was outside the scope of work for this report (Appendix 1). We found no impact assessments of waste discharge or reports of the presence of pathogens in water and discharged wastes from salmonid enhancement hatcheries in British Columbia.

Exposure Assessment Summary

Fraser River sockeye salmon reared in enhancement facilities have the most likely route of exposure to diseases present in hatcheries or spawning channels. Exposure of other Fraser River sockeye to infected enhanced fish, sockeye salmon or otherwise, has not been proven or disproven. Biologically plausible routes of exposure exist, but none have been measured. Generally, there are three variables that affect the probability of exposure; the geographic distribution of the escaped pathogen, the abundance of the pathogen in the receiving environment and the frequency with which the fish are involved in an exposure that results in transmission of the pathogen. As we lack data for these 3 variables, exposure assessment is not possible.

Consequence assessment

This risk analysis was unable to find information that added to the literature review to clarify the consequence of a pathogen of enhancement facility origin for Fraser River sockeye salmon productivity. The risk assessment did establish that known fish pathogens do occur in salmonid hatcheries and spawning channels and that biologically plausible routes of exposure exist, but there was no monitoring or follow-up to establish that pathogens were transferred to sockeye salmon outside of sockeye salmon enhancement facilities and that the transfer impacted the

population. We were left to rely on Kent's (2011) ranking to identify high and medium risk pathogens. There was inadequate time to establish an independent ranking system, however we refer the reader to elsewhere in the report wherein we comment on the limitations of Kent's ranking for assessing the potential for a pathogen to cause population impacts on Fraser River sockeye salmon.

Risk Management

Fish health management plans

Fish health is one of the major management concerns for salmonid enhancement facilities because disease outbreaks can have devastating effects on the fish held in a culture facility. Many management decisions on water quality, nutrition and husbandry are intended to provide the foundation for healthy fish. Historically, these practices were not well standardized or documented across federal and provincial (FFSBC) facilities. Fish Health Management Plans (FHMP) are now an accepted means to develop explicit operating procedures to prevent and control disease.

The intent of FHMP is to set expectations for basic components of a fish health management program for public and private fish culture. FHMP templates and manuals are available through the BC Ministry of Agriculture (BCMA) website (BCMA, 2004). A generic template is provided to help facility operators develop site specific documents that work toward best management practices. The FHMP recognizes the challenge of defining "best management" because of the variation in species and conditions under which fish are reared and due to a lack of evidence-based management programs. However, they do describe the components and goals needed for basic fish health management program as defined by consensus of a working group that involved private and public sector professionals responsible for fish health. The FHMPs are expected to be reviewed and updated regularly.

We obtained FHMP plans from the following federal hatcheries (note – most do not produce sockeye salmon): Big Qualicum, Capilano, Chehalis, Conuma, Cultus Sockeye, Inch Sockeye Satellite, Inch, Kitimat, Little Qualicum, Nitinat, Puntledge, Quinsam, Robertson, Rosewall, Shuswap, Snootli, Spius and Tenderfoot. We also obtained FHMPs from FFSBC hatcheries (Clearwater Trout Hatchery, Vancouver Island Trout Hatchery, and Fraser Valley Trout Hatchery) (see Introduction for names of DFO facilities rearing sockeye salmon). Some of the federal FHMPs were for facilities outside of our study area and therefore were not reviewed. We did not receive FHMP's for the Gates, Horsefly, Nadina and Weaver Creek spawning channels, or for CEDP and PIP facilities. We could not confirm whether these FHMPS do not exist or were not provided. We found 14 files in the documents provided to us by the Cohen Commission

with standard operating procedures (SOPs) for CEDP and PIP facilities within the facility records provided.

The FHMPs included the sections from the BCMA template relevant to infectious disease control, including the responsibilities of the fish health team. They also included operating procedures for quarantine, cleaning and disinfection, movement of fish and fomites, disposal of dead fish, monitoring fish health and disease, record keeping, outbreak management and disease screening in broodstock.

The general goals of the FHMP are to:

1. Prevent introduction of pathogens to a facility
2. Reduce disease levels at a facility
3. Safely and properly administer drugs and chemicals used
4. Minimize the spread of infections
5. Maintain environments that are conducive to good fish health

Neither the federal nor FFSBC programs have evaluated or audited their FHMPs (Personal communications, 2011, Dr. Christine MacWilliams, Ms Sherry Mead). The FFSBC plans we examined had identical and generic operating procedures and therefore are not adapted to specific hatchery conditions. Ms. Sherry Mead of the FFSBC reported that there is a plan to develop site specific plans in the future because of the expectations of the Pacific Aquaculture Regulations. The FFSBC did report an audit of biosecurity practices 3 years ago and there is a plan to repeat this audit in the Fall of 2011. DFO staff reported that a contractor is undertaking a gap analysis to see where FHMPs could be improved for federal facilities. They did anticipate gaps in biosecurity practices to be found. While there was considerable overlap in the content of the federal FHMPs, some differences existed between hatcheries and are highlighted below. PBS fish health staff indicated their goal of reviewing the FHMP for each facility prior to the 18th month license renewal.

Review of the FHMPs can only provide us insight on the intention of management and cannot determine if the practices are regularly applied or if the practices have effectively reduced risk.

Overview of the FHMPs provided for review

The FHMP for Clearwater Trout Hatchery, Vancouver Island Trout Hatchery, and Fraser Valley Trout Hatchery contained identical and generic standard operating procedures under the same headings established by the BC Ministry of Agriculture. The DFO FHMP's followed a standard layout: Sections 1 (Introduction) and 2 (General Principles of Fish Health Management) contained the same components (including subsection titles) as the BCMA Template. These

sections contained generic biosecurity and management principles. Section 3 (A Brief Overview of this Facility) contained a short summary of the specific hatchery, with a description of the enhancement techniques and species involved. In Section 4 (Standard Operating Procedures for the <*name*> Hatchery), specific biosecurity and management procedures are introduced by way of a rational for the procedure, identification of the responsible authority/personnel, and a summary of the general principles (these components were present in all reviewed DFO FHMP's). Each operating procedure is then written out in detail, and in some cases site-specific differences are addressed. The level of detail varied between hatcheries. Disinfection and biosecurity protocols related to general day-to-day operations and are similar for all reviewed DFO FHMP's.

Preventing the release or movement of pathogens outside of the hatchery

Section 2.11 of the FFSBC FHMPs states, "The health and treatment status of fish will be considered when planning intentional fish releases from enhancement/conservation facilities. If there is a health or treatment concern fish shall not be released until risk assessment recommendations are in place". The FFSBC has standard operating procedures for health risk assessment of fish releases (SOP 2.11). Its objective is "to ensure that fish are not released (liberated) until an adequate fish health assessment is conducted by a qualified fish health professional". The operating procedure only deals with "fish that are known to have been exposed to a fish pathogen, have been treated with a drug or chemical or are affected by an unknown cause of death or illness or that require [an Introductions and Transfer Committee] permit for transport". The Fish Health Unit reviews fish stocking requests and determines which stocks of fish require a fish health assessment. The FFSBC FHMP stated that fish cannot be released if a pathogen has been detected and there is a risk of exposing wild aquatic organisms to the pathogen and that fish must be "clean of any bacterial, viral or parasitic infection for 10 days before release." However, it also states that when the Fish Health Unit, senior management and the Hatchery Manager determined that the risk of spreading the pathogen was limited and manageable, some fish transfers would occur, unless the fish are under isolation or quarantine. Fish may not be released if there is an unknown cause of mortality or morbidity.

Ms. Mead of the FFSBC Fish Health Group outlined an informal risk assessment process that considers issues such as; have the fish passed their drug withdrawl time if they have been treated; have 20-30 moribund or fish seeming different than the rest undergone fish disease screening at the lab; and is the release time a reasonably long period after the normal course of infection for the disease in question. The time line between the last detected case and time for allowable release was, admittedly, a matter of professional judgement. However, a window of 2 months from last case to release was generally deemed to be acceptable.

Pre-release risk assessment information was inconsistent in the federal FHMPs provided. Only three of the reviewed DFO FHMP's described formal pre-release screening procedures for an identified disease or transfer event (Capilano, Tenderfoot, and Inch Sockeye Satellite hatcheries). Big Qualicum Hatchery stated in its FHMP, "No formal pre-release risk assessment is performed. Fish are sampled for individual length weights (sic) prior to release and examined for physical condition at this time;" and "no specific health checks precede release." The FHMP for the Inch Creek Sockeye Satellite facility (which deals with endangered stock) had more extensive pre-release operating procedures (described below).

Two tiers of pre-release risk assessments are described (depending on the FHMP, FHMP sections 4.15 through 4.18). Informal risk assessments are permitted between sites "with a long-standing established program involving annual fish transfers," where "appropriate surveillance data" is collected and there is "historical knowledge in endemic disease issues in the two populations." It is suggested that these transfer programs be "reviewed during the facility annual production planning process." In the second tier of pre-release risk assessment, "no sick fish should be transferred between sites or knowingly be released without a disease evaluation." Where disease losses and treatment have occurred, or where a new program is being implemented, "the Veterinarian may request a sample of either healthy or moribund fish for disease prevalence estimation at least 2 weeks prior to transfer/release."

The stated operational procedure at Big Qualicum and Little Qualicum hatcheries is to examine the physical condition of the fish during individual length/weight sampling prior to release. The length of time prior to release was not specified. The FHMP for Capilano Hatchery states that with the exception of BKD screening in the Chehalis coho salmon broodstock and eggs, there is no routine pre-release screening. Pre-release screening is not described in the Chehalis River Hatchery FHMP. Informal risk assessments are permitted at Inch Creek, Shuswap and Spius Creek hatcheries; the Shuswap Hatchery specifies that sockeye salmon ponds will be checked for IHN three weeks prior to release, but does not provide details on how this is to be carried out. At Inch Sockeye Satellite, "all transplant permits must be in place prior to movement of fish or gametes on or offsite." Puntledge River Hatchery is the only FHMP that specifies that samples from unhealthy fish should be forwarded to the lab for diagnostics and that fish releases should not occur before treatment and withdrawal times have elapsed. The Quinsam Hatchery FHMP suggests that the fish be held back and the veterinarian contacted "if there is any question as to the health of the fish to be released." "Fish are not released directly from Rosewall Creek Hatchery as all stocks reared on site are transported from other facilities." Rosewall Creek Hatchery FHMP also states that "fry are not normally screened for disease prior to release." Rosewall Creek Hatchery FHMP contains this single statement: "Fish designated for the watershed should be checked for health

condition prior to release." There is a reference back to the sections on *Pre-release or Transfer Disease Risk Assessment* and *Transporting Fish*, but the specifics of how to check the health condition are not provided. The Rosewall Creek Hatchery FHMP contains the statement: "Juvenile release protocols are governed by the Cultus Lake FHMP". The Cultus Lake Hatchery FHMP states "no sick fish will be transferred between sites or knowingly be released without disease evaluation. Depopulation, treatment and release options will be reviewed on a case by case basis." This section does not contain any further SOP or description as to disease evaluation. Cultus Lake Hatchery FHMP notes that this "sockeye facility is under constant quarantine as are the items they [sic] contained within it." In the case of a suspected or confirmed outbreak of IHN, the population will be destroyed. A brief case definition is given as "high acute mortality, blood in the eye, blood at the base of the fins." Information was not provided on how these fish must be killed or disposed of. Other sections discussed humane euthanasia and carcass disposal, but in generalities and not specifically to fish that fit this case definition.

At the Inch Creek Satellite facility, for each proposed fish transfer, the Fish Health Management Team is directed to consider: species, life stage, disease and treatment records; location of receiving facility or watershed; disease history of the current rearing facility; history of pathogen surveillance within the population being moved; history of pathogen surveillance and prevalence in the feral populations within the receiving waters; and availability of post-release isolation or disease sampling and diagnostics. The operating procedure allows for an informal risk assessment when there is a "long-standing established program involving annual fish transfers between two sites, with appropriate surveillance data collected and historical knowledge in endemic disease issues in the two populations." "In the case of new programs or where pathogen surveillance for either the receiving or rearing populations is lacking or in instances where the rearing population has suffered disease losses and treatment, the Veterinarian may request a sample of either healthy or moribund fish for disease prevalence estimation at least 2 weeks prior to transfer/release". The procedure states that "no sick fish will be transferred between sites or knowingly be released without disease evaluation". As for the FFSBC, there were no set thresholds for acceptable risk. Instead the professional judgements of fish health and hatchery staff seemed critical.

There was some contradiction within an individual hatchery's FHMP with respect to releasing infected fish. The following passage was extracted from the Tenderfoot Hatchery FHMP, Section 4.17:

> "No sick fish will be transferred between sites or knowingly be released without disease evaluation. Depopulation, treatment and release options will be reviewed on a case by case basis.

If there are early stages of disease (e.g. Vibrio) showing in fish that are already in the marine net pens prior to intended net pen release, fish will be released immediately to reduce the impacts of stress associated with continued holding and associated mortalities associated with the disease.

Steelhead smolts being transferred onto the Tenderfoot site have had a historical incidence of myxobacterial infection. Fish are generally treated prior to movement to the Tenderfoot site."

Pre-release screening is at the discretion of the hatchery manager and facility staff, and appears to be performed largely when fish look diseased (e.g. abnormal behaviour, increased mortalities). Dr. Christine MacWillams, DFO veterinarian, outlined for us her procedure for reviewing pre-release risks. Pre-release risk assessments are done for all fish that have received antibiotics and in accordance to their BKD management plan. Risks will also be reviewed for any groups with mortality rates > 0.1%/day for 4 consecutive days near the release window; fish with mortality >5% per day in 90 days prior to release (yearling only); rising morbidity or mortality prior to release or clinical signs of BKD. For fry, pre-smolts and yearlings that have no disease issues or significant losses during rearing or mortality problems have resolved (for fry only), release is at the discretion of the hatchery manager. Lingering losses or presumptive or confirmed disease triggers an examination of hatchery records and submissions of fish for diagnostic examination. Note: these informal guidelines did not specify how close to release these disease events must be to trigger examination. Any sockeye salmon with suspicion of IHN are destroyed and a sample of 20 are submitted to confirm and genotype the virus.

CEDP and PIP operating procedures

The documents we received dealt with chemical treatments or disinfection using chloramine T, Ovadine® and formalin treatments/disinfections with the following exceptions. One document provided a crude case definition for disease in fish. The Cowichan River CEDP facility had a draft FHMP, which did not require a formal pre-release risk assessment. The Seymour facility also had a draft FHMP but we received only 7 pages that dealt with egg disinfection and egg fungus treatment. The Quatse River facility documents included the following: "Always be aware of any signs of concern that become apparent. For example, flashing, lethargy, lesions or boils, haemorrhaging, fin decay, protruding eyes, uneaten feed, and increasing mortality rates may be signs of infection, disease, overcrowding or oxygen concerns. Always inspect morts for any of these signs. Check adult brood daily for fungus. Any signs of concern must be reported to the hatchery manager as soon as they become apparent. The manager or supervisor may send samples of fish for testing." None discussed pre-release disease screening.

It is important to remind the reader that we requested only a random sample of PIP facilities as we were told that DFO was unlikely to be able to produce documents for all relevant PIPs within the timeline of the report. Therefore, there may be PIP facilities with operating procedures that differ from what we described above.

Other risk management measures

All of the reviewed DFO FHMPs contain the following statement: "In the event of a fish health crisis or potential disease outbreak, until the cause of mortality has been confirmed, the site should be managed as though an infectious agent is present" to reduce the pathogen load on-site and to prevent the spread of pathogens on or off the site (depending on the FHMP, FHMP Sections 4.20 through 4.24). When losses are likely to be the result of infectious disease, the DFO veterinarian must be notified immediately and their recommendations for frequency of mortality observation, sample collection for diagnostics, quarantine procedures and treatment protocols followed. Operating procedures for sample collections and quarantine are also outlined in the FHMPs, but it is recommended that the veterinarian's instructions be followed. The protocols described in the reviewed DFO FHMPs are summarized below:

1. Prohibit on-site entry of visitors and non-essential staff unless previously authorized by the Fish Health Management Team. This may include closure of the site to the public
2. Notify surrounding fish rearing facilities of the outbreak
3. Isolate/quarantine the infected population from healthy populations. This may be very difficult to do given water seepage between on-site areas
4. Prohibit handling of the infected fish
5. Immediately halt the movement of fish, vehicles, equipment and personnel from the affected hatchery to fish bearing habitat or other fish rearing facilities
6. If possible, trap effluent and treat it prior to discharge into the environment. It is noted that the treatment of effluent is not always feasible at all locations
7. Healthy fish must be cared for first. Dividing personnel to work in different areas may help reduce any potential cross contamination
8. Increase the frequency of mortality collection as mortalities escalate, ensuring that staff adheres to disinfection protocols between tanks
9. Removal of all sick, slow-moving or moribund fish may be considered depending on the overall mortality rate
10. Thorough disinfection of equipment, surfaces and clothing that come into contact with infected fish or infected material. Frequency of cleaning will increase as mortalities escalate

11. Samples will be collected and forwarded to PBS when an abnormal increase in mortality rates/levels is noted

Of the reviewed DFO FHMPs, only Inch Sockeye Satellite gave specific instructions to destroy any fish population that shows clinical signs consistent with a pathogen (IHN in this instance; a case definition for IHN is provided in the FHMP for this facility). The timely removal of dead fish is intended to decrease predator attraction and pathogen spread, as well as to maintain overall site hygiene. A number of FHMPs state that "if daily mortalities exceed 0.5%, fish health management should be notified and the veterinarian consulted." At the Inch Sockeye Satellite facility, a daily mortality rate of 0.01% was enough to trigger veterinary consultation.

Specific procedural descriptions that relate to the collection and counting of dead fish, disinfection of equipment, and disposal of dead fish varied by hatchery, but overall similar biosecurity principles were described. Surplus broodstock carcasses may be returned to "their natal streams to provide nutrient enrichment" (DFO Carcass Placement Guidelines provides instructions, and the intergovernmental Introductions and Transfers Committee authorizes the permits), be handed over to First Nations for commercial use, or may be frozen for feeding wildlife and for use by wildlife recovery centers; in general however, mortalities from both broodstock collections and rearing facilities are composted, buried or sent to the municipal landfills. In addition to the operating procedures above, the Big Qualicum Hatchery FHMP states that juvenile mortalities in the rearing ponds are generally collected and counted while vacuuming the ponds, and that "these mortalities are pulverized by the pump which discharges the effluent back into the river." A distinction between diseased and non-disease mortalities is not made; however, at Little Qualicum Hatchery, dead juvenile fish, pinheads and moribund fish are disposed of to the enclosure outflow unless a disease problem is known to be occurring, in which case the FHMP stipulates that dead fish be buried in a mort pit.

DFO applies the principles of the Alaska Sockeye Culture Manual (McDaniel et al., 1994) for sockeye salmon culture for IHN control. It relies largely on; (1) segregation of sockeye salmon to their own facility or having them compartmentalized within a multi-species facility; (2) use of virus free water (well water or above barrier-water) and; (3) egg disinfection. DFO no longer does routine broodstock screening for this diseases because of the lack of historic correlation between screening outcomes and subsequent diseases. They will do population screening by testing 60 fish and if IHN is found to be prevalent (level not specified), they will do additional egg disinfection.

Access to low pathogen water sources varies between facilities. FFSBC hatcheries reportedly all use ground water – a water source least likely to have fish pathogens. Most major DFO hatcheries have access to ground water for egg incubation and some have enough for their entire

production. Smaller sites tend more often to use surface water and even larger sites will often need to use surface water for fish culture due to access or cost of using ground water exclusively (personal communication, 2011. Dr Christine McWilliams).

Drug and chemical treatments

Drug and chemical treatments of fish are allowed under the FHMPs. In-house (hatchery-level) treatment records were submitted to us in a variety of formats including: (1) daily husbandry records which documented deaths, handling, and fish health treatments for fish populations held in tanks, tubs, troughs and keeper channels; (2) treatment calculations; (3) hand written treatment records; (4) disease and treatment records; (5) prescriptions; (6) email communication; (7) PBS fish health reports; (8) broodstock BKD prophylactic treatment records; and (9) egg treatment records. All had to be searched and integrated to try to describe treatment practices at hatcheries.

Table 16 summarizes the frequency with which various treatments appeared in treatment records (excluding egg and broodstock treatments). For the chemicals chloramine T and formalin, data in Table 16 are the number of times the treatment was noted in the records. If one tank was treated for 3 days, we would record 3 treatments. This allowed us to get a sense of the number of daily treatments delivered. For drug treatments, the records typically stated when the treatment began, but did not state the duration of the treatment. Antibiotics would have most likely been used under a veterinary prescription that would specify the duration of treatment. For example, in Table 16, we noted 37 treatments with oxytetracycline for CEDP and PIP facilities. If the average duration of treatment was 10 days, this would represent 370 days treatments. We could not readily match the prescription records with the facility treatment records and therefore lacked data to determine duration of drug treatment. The application, and reporting, of chemical and pharmaceutical treatments varied across facilities. For example, 4 CEDP/PIP facilities recorded formalin use and 9 reported Chloramine T use, but only 3 reported use of both Chloramine T and formalin.

Table 16 must not be used to assess if drugs and chemicals were used appropriately as they lacked details on the conditions and duration of use. They should also not be used to estimate volumes of drugs or chemicals used as we could not validate the doses, number of fish treated and duration of uses. The value of Table 16 for this risk assessment is that they show that DFO, CED and PIP facilities do take steps to treat infectious diseases. The drugs and chemicals identified are consistent with the etiologies of infectious diseases identified in the diagnostic and hatchery records. Additional treatment recommendations were provided on husbandry modifications to reduce stressors and in some cases, recommendations were given to not treat fish.

Table 16: Treatments recorded in available CEDP, PIP and DFO hatchery records (not including egg treatments) from March 2000-March 2011.

	CEDP/PIP hatcheries	DFO Major hatcheries
Chemical treatments	**Daily treatments delivered**	
Chloramine T	485	875
Formalin	291	412
Salt	0	45
Drug treatments	**Treatment courses recorded**	
Oxytetracycline	37	59
Tribrissen	5	8
Florfenicol	4	11
Medicated feed (not defined)	15	0
Emamectin	0	8
Romet®	0	1

Records for egg treatments – most of which involved surface disinfection with Ovadine® (iodine based disinfectant) and a small number of reports of using Parasite S® (a formalin based treatment) for fungus - are not included in Table 16. Additional records not in Table 16 covered the use of injectable antibiotics for broodstock infections; a treatment intended to reduce the risk of transfer of *Renibacterium salmoninarum* (causal agent of bacterial kidney disease) from broodstock to eggs.

Treatments records were not always accompanied by a diagnosis. For DFO hatcheries and spawning channels (excluding CEDP and PIP facilities), 202 records for chloramine T and formalin had no stated reason for treatment. Reasons provided for treatment included myxobacteria (n=147); symptoms compatible with external parasites, e.g flashing (n=136); bacterial gill disease (n=110), fungus (n=45); undefined parasites (n=41); trichodina (n=91); increased mortality (n=23); costia (n=12) and other or unknown (n=40).

The treatment record review found that some salmon in the federal system have been released within their drug withdrawal period and in some cases released when fish still showed symptoms or were experiencing ongoing losses (n=3 cases). Nine cases described fish being released while clinical signs were present, 0-10 days after treatment stopped. All involved signs consistent with external parasites or myxobacteriosis but not all cases had accompanying diagnoses in these treatment records. Assuming the fish released are below catchable size, it may be safe to assume

that the fish will undergo drug withdrawal before they enter the human food chain thus presenting little public health risk. It is unknown whether such released salmonids contribute sufficient drug residues to the environment (including predators) to affect the drug resistance patterns or virulence of environmental bacteria or fish pathogens. The impacts of this practice on sockeye salmon disease dynamics or public health have not been evaluated.

Pacific Aquaculture Regulations

The Pacific Aquaculture Regulations came into effect December 2010. Salmonid enhancement facilities are required to comply with the regulations. The information we reviewed, on practices and disease outcomes occurred prior to their implementation.

Among other things, the Regulations deal broadly with measures to control and monitor pathogens and pests in a facility. They require monitoring of the presence of pathogens and pests in wild fish in the waters that might be affected by the aquaculture operation. They require notice to the Fisheries Minister before a substance is used to treat fish for pathogens or pests. Records of diagnoses and treatments and the extent to which fish are affected by a pathogen or pest must be kept. The regulations specify that measures must be taken to minimize the environmental impact of an aquaculture operation. The requirements of the regulations are non-specific and no thresholds for acceptable impact or confidence in monitoring presence of pathogens were evident. The implications of these regulations on enhancement practices described in this document are unknown as we did not find an implementation plan.

We were provided with a draft licence for Big Qualicum Hatchery. In its appendices were FHMPs for Big Qualicum, Little Qualicum and Rosewall hatcheries. These were identical to the versions we described above.

Risk Assessment Summary

Fish in enhancement facilities have been diagnosed with a variety of bacterial, viral and parasitic pathogens. Some of these pathogens can be classified as high risk based on the criteria of Kent (2011). No foreign of exotic fish pathogens were identified. Available data did not allow for the calculation of the rates or frequencies of these hazards in enhanced fish populations. Enhanced Fraser River sockeye salmon have been diagnosed with microbiological hazards.

Several possible routes of release of pathogens from enhancement facilities were found. Some of these routes are standard operating procedures, such as the release into streams of eggs known to come from BKD infected broodstock. Others happen in opposition to the goal of not releasing infected fish; including the release of fish with known infections and/or their transfer to freshwater or saltwater netpens. The proportion of fish that were infected in these cases, their

survival post-release and their interactions with other salmon cannot be established with the data provided. Exposure assessment was not possible.

Risk management plans are in place to reduce the likelihood of infected fish being present or released but there has been no audit or assessment of these procedures to determine their effectiveness. Procedures appear to vary between sites.

The risk assessment can only conclude that the risk of transfer of infectious agents is biologically plausible, but the absence of an acceptable level of risk coupled with the lack of exposure data precludes determination of the magnitude or probability of risk to Fraser River sockeye salmon.

Conclusions

Limitations in scientific understanding, lack of ongoing surveillance of wild and cultured salmonids and non-salmonids, and deficits in the data provided to us precluded qualitative or quantitative assessment of the risk of salmonid enhancement associated infectious diseases to Fraser River sockeye salmon production. The data available for this review could not prove or disprove that diseases associated with salmonid enhancement facilities have been transmitted to Fraser River sockeye salmon and in turn, have impacted their production.

There is a suite of pathogenic hazards present within salmonid enhancement facilities and evidence that pathogens have viable means to escape spawning channels and hatcheries via fish or water and thus enter fish bearing waters occupied by Fraser River sockeye salmon. We can confirm that the portion of the Fraser River sockeye salmon population that is reared in spawning channels or hatcheries has, at times, been exposed to infectious diseases while within the enhancement operations, but we can find no evidence that this exposure had medium-to-long-term population regulating effects. We could not establish if Fraser River sockeye salmon not reared in enhancement facilities had or had not been exposed to infectious agents of enhancement facility origin. Specification of the consequence of a potential exposure to infectious agents on free-ranging fish cannot be determined within the current scope of scientific knowledge.

There is no accepted standard for acceptable exposure of free-ranging fish to pathogens of public fish culture facility origin, apart from preventing the introduction of an exotic disease. A zero risk standard is unachievable with the current system for salmon enhancement because a number of the infectious agents are ubiquitous in aquatic environments or common in cultivated or wild fishes. Our understanding of the dynamics of fish diseases allow us to conclude that a single standard may be impractical as the impacts of diseases in populations will be dependent on environmental and population factors.

The existing operating procedures for risk reduction focus on reducing the prevalence of disease within groups of fish to be released from salmonid enhancement operations as well as pre-release assessments of groups with previous disease or infection histories. There is no ongoing surveillance or assessment of the infections status of groups that either are not showing clinical signs and/or are not progeny of fish with known vertically transmitted infections.

The current programs of fish health units at PBS and the FFSBC are focused on the diagnosis and treatment of disease. There are inadequate resources to have fish health professionals service enhancement facilities to adapt fish health management plans to local conditions, audit practices

and develop ongoing disease prevention programs. This makes their risk reduction steps reactive to disease occurrences rather than proactive and preventive.

Fish health management plans aim to be proactive by creating conditions that are not conducive to the introduction, spread and persistence of infectious diseases. This includes addressing husbandry, biosecurity and disease management risk factors. There is variability and lack of specificity in these plans. The fish health management plans have not been audited or evaluated at the federal or FFSBC enhancement facilities and are lacking at CEDP and PIP facilities for which we had data.

The current system for reporting and recording fish health in enhancement facilities or for documenting the suitability of fish for release lack consistency, quality and accessibility thus limiting external review and public assurance. This situation was most pronounced in the CEDP and PIP facilities for which we had data.

Despite the aforementioned limitations, much can be said for the salmonid enhancement programs and their work in fish health. These facilities have a number of programs or procedures in place intended to reduce risk to wild fish by reducing the amount of disease in their production fish. Fish health staff at DFO and FFSBC are dedicated professionals committed to providing support to staff responsible for enhancing salmonids in British Columbia. They have tremendous responsibility for a very large number of fish and comparatively few resources. Deficits in their capacity to be proactive in risk recognition and reduction, to have more detailed information on determinants of risk and to deliver a fish health service rather than only diagnostic service is a reflection of the historic organizational, infrastructure and capacity issues.

State of the Science

We provided many examples above of the gaps in our understanding of the effects of infectious diseases on the productivity of Fraser River sockeye salmon and the methodological challenges to addressing those gaps through scientific research. The vast majority of fish disease research has investigated the impacts of specific pathogens on individual fish. The research emphasis on the pathophysiology and microbiology of cultured salmonid diseases has been insufficient to answer questions on how infectious disease can affect the distribution and abundance of salmon outside of fish culture settings. The health research paradigm for fish diseases has been one of eradication or control of specific pathogens that limit productivity and survival of fish in fish culture settings and thus has largely defined health as the absence of disease of cultured salmon rather than the capacity for wild salmon to thrive and survive. Little research has been done to define socially and ecologically tolerable levels of disease associated with salmonid enhancement. Fish health research has typically looked at the effects of pathogens in isolation rather than looking at infectious diseases as one of a suite of stressors to salmon populations. Few efforts have been made to take a systems view of health and diseases; a view that attempts to look at health outcomes holistically rather than in a reductionist fashion. The fundamental lack of ability to trace a salmon through its life history is an enormous barrier to characterizing the risks and impacts of diseases on Fraser River sockeye salmon. Evidence-based conclusions on the effects of diseases of enhancement facilities on Fraser River sockeye salmon are not currently possible. We found no research that could document the direction and frequency of pathogen movement between enhancement hatcheries or spawning channels and Fraser River sockeye salmon. Molecular epidemiological studies are absent. The frequency and probability of exposure of Fraser River sockeye salmon to pathogens of enhancement facility origin can, therefore, not be determined.

Risk management at fish culture operations has the advantage of several years of observational studies, some laboratory experiments, analogy with other animal health care settings and personal experiences to develop programs and practices for fish health management. However, few of these practices have been evaluated by methods that would meet expectations for evidence-based medicine. Epidemiological research is rare in this field. Participatory methods have not been used to develop consensus on socially and ecologically acceptable risk management. A fundamental shift in direction is required to mobilize science to generate the information needed to confidently and objectively address the questions within our scope of work and inform management.

Recommendations

The information deficits described above prevented us from concluding if the threshold for applying the precautionary approach of "potential for serious or irreversible harms" (as set out in the Rio Declaration on Environment and Development (UNGA, 1992)), has or has not been reached. The nature of the risk and best means to reduce or prevent risks will not be identified definitively without extensive and innovative research. There will be short, medium and long-term research questions and products, but it can be anticipated that resolving the uncertainties that plagued this review will not be a quick process. There are management recommendations that can be implemented in the short term that would bring the approach to public fish culture in line with expectations for animal health and conservation seen in other settings. The importance of aquatic animal health management in responsible stock enhancement has been recognized by the scientific and international communities (Blankenship and Leber, 1995 from Bartley et al., 2006) and is embodied in articles in the FAO Code of Conduct for Responsible Fisheries. Despite our inability to specify the level of risk, we provide management recommendations that may improve the transparency, efficiency and effectiveness of fish health risk management in BC salmonid enhancement programs.

Management Recommendations

Recommendation 1: Adopt an adaptive management approach that uses systematic monitoring and ongoing evaluation of DFO and FFSBC fish health services and programs to assess the effectiveness, efficiency and acceptability of not only the following recommendations but ongoing program activities.

> Rationale: The lack of systematic evaluations of fish health standards and programs precludes the use of evidence-based definitions of best practices. Ongoing learning through adaptive management will provide a systematic means for progress towards best practices. DFO and FFSBC have access to a number of salmonid enhancement programs both within BC and the US Pacific Northwest to draw on for this experience.

Recommendation 2: Provide the capacity to expand the focus of the fish health units from disease diagnostic services to fish health management support.

> Rationale: The primary means to protect Fraser River sockeye salmon from diseases derived from salmonid enhancement facilities is to prevent disease in enhancement facilities. Fish health units have traditionally been disease diagnostic support for enhancement programs rather than a comprehensive service that works to promote health and prevent and contain disease. This leaves them largely reactive to disease incidents

rather than in a proactive health protection mode. Significant responsibility for health promotion and protection falls onto the hatchery managers and community advisors who have limited training in fish health. A team-based health management program would shift public fish culture to a modern animal health approach.

Sub-recommendation 2a: Make information management and records systems consistent across facilities and accessible to fish health staff to allow for ongoing surveillance of trends in growth, morbidity, mortality, population information and environmental quality.

Sub-recommendation 2b: Enable fish health programs to consistently record, review and assess trends in diagnostic information and hatchery level surveillance of risk factors and population parameters.

Sub-recommendation 2c: Provide personnel in fish health units with continuing education or advanced training in health protection and promotion to allow them to serve as a resource for hatchery staff.

Sub-recommendation 2d: Enable fish health programs to develop extension and training capacity for hatchery staff and community advisors to ensure a common understanding and ability to fulfill fish health recommendations and fish health management plans.

Sub-recommendation 2e: Create the capacity for fish health staff to visit facilities on a regular basis and not just in response to disease outbreaks, urgent issues or for research. Veterinarians responsible for prescriptions must visit sites sufficiently often to ensure appropriate veterinary-patient-client relationships.

Sub-recommendation 2f: Provide regular and continued access for community advisors and hatcheries to local/regional fish health technicians (or equivalent expertise) to assist in planning and implementing fish health programs and to assist in recognition of disease issues.

Sub-recommendation 2g: Provide fish health units with sufficient numbers of highly-qualified human resources to deliver on their diagnostic responsibilities, to record and assess trends in disease, and to work towards the recommendations in this report.

Recommendation 3: Re-organize existing programs so that salmon health is not segregated by ownership or discipline.

Rationale: Current fish health programs separate personnel, infrastructure and capacity by whether or not a salmon is privately owned, is publically owned but cultured or is wild. Understanding the disease relationships of cultured and wild fish will require capacity and expertise to integrate data and efforts across public, private and wild fish sectors. Private sector, DFO and FFSBC each have insights, capacities and methods for monitoring and controlling disease that can be shared for more effective adaptive management. Programs are also separated by discipline in that people dealing with disease outcomes are not linked to people or programs dealing with the determinants of health and disease (e.g. population ecology, environmental quality).

Sub-recommendation 3a: Use the new responsibility for DFO to manage private and public sector fish culture and wild salmon as an opportunity to integrate fish health programs and develop new capacity in wild fish health assessment.

Sub-recommendation 3b: Re-instate and support the federal-provincial fish health management committee which was an advisory body without jurisdictional authority or responsibility that served as a venue for fish health experts to share information, synthesize existing knowledge and provide evidence-informed advice on fish health management.

Rationale: This will be an important mechanism to bring the FFSBC fish health staff into a more integrated program. We foresee this committee as an important non-partisan mechanism to implement the currently vague standards of the Pacific Aquaculture Regulations into equivalent actions across fish culture sectors (federal, provincial, community, private). The committee will need an ongoing budget to support knowledge synthesis and knowledge-to-action activities.

Sub-recommendation 3c: Develop a working group that serves to gather and integrate information on wild and cultured fish health, ecology and management to provide an ecosystem-based view of health risks.

Rationale: As a minimum, this multidisciplinary group should come together regularly to share what they know about fish ecology, habitat, disease and other determinants of fish health. DFO and FFSBC should adapt principles of horizontal management as well as ensure that information technologies are compatible to allow for easy and regular sharing of information across disciplines

(e.g. fish disease, population status, environmental quality) to develop a comprehensive view of fish health. Personnel involved in these activities must be supported by their managers.

Sub-recommendation 3d: Expand the responsibility of fish health units to more than cultured salmonids.

Rationale: Improved understanding of trends in wild fish health cannot be achieved by concentrating efforts on commercially important species (primarily salmonids) held under culture conditions.

Recommendation 4: Develop consistent and transparent processes for assessing the risk of releasing enhanced salmonids into fish bearing waters.

Rationale: Despite the existence of Fish Health Management Plans, there are apparent inconsistencies in how information is gathered and recorded, as well as a lack of transparency and consistency in decisions to release salmonids and wastes from enhancement hatcheries.

Sub-recommendation 4a: Undertake a more detailed audit of fish releases and waste management.

Rationale: Audits should be performed in order to ensure the findings in this report, which were not site specific, are valid across enhancement facilities and to identify priorities and responsibilities for action.

Sub-recommendation 4b: Develop a consensus based decision algorithm for use by hatchery managers to determine if an agreed-to risk threshold has/has not been met prior to fish releases.

Rationale: The rationale for acceptable risk must be made explicit and consistent across facilities and there needs to be supporting evidence that the criteria for acceptable risk have been met. While there are documented standards and procedures for pre-release risk assessment, they did not apply to all groups of fish and did not seem to be consistently applied.

Sub-recommendation 4c: Create capacity for post-release monitoring of enhanced fish and fish in the receiving water to develop evidence of effectiveness and/or need for modified decision standards.

Rationale: Refinement and monitoring of the effectiveness of risk prevention strategies requires data on the impacts of risk management decision on released fish and other fish in the receiving waters.

Recommendation 5: Improve capacity for auditing and oversight of fish health, especially in terms of risks to wild fish (salmonids and non-salmonids).

Rationale: Lack of regular assessment of fish health status in enhanced salmonids not only creates problems in the ability to monitor and adapt programs, but also in standardization of practices and public assurance that health risks are being adequately monitored and managed.

Sub-recommendation 5a: Develop standards for record keeping at enhancement facilities and develop a program for regular review and assessment of fish health records.

Rationale: Fish health is to be viewed comprehensively as not only morbidity, mortality and disease diagnostics, but also tracking risk indicator, risk factors and determinants of health such as water quality, density and growth. Ongoing record assessments and reviews will allow for regular site specific reporting of fish health status and increase transparency of the fish health status of specific locations. This system can be standardized and inputted using web-based technology.

Sub-recommendation 5b: Regularly review, assess and communicate to risk managers trends in diagnostic and screening results to increase surveillance capacity as well as to provide transparency on disease status on hatcheries.

Sub-recommendation 5c: Standardized surveillance case definitions to assist in trend analysis and reporting.

Rationale: Case definitions would assist management decisions such as fish transfers, release decisions, broodstock collection and destruction recommendations.

Sub-recommendation 5d: Develop capacity for surveillance of disease and fish health risk factors that can be used to support assessment of risks to cultured and wild fish.

Rationale: This recommendation includes sub-recommendation 5b plus surveillance of key determinants of risk and increased capacity to track wild fish health. Fish health is not a laboratory subject alone. The determinants of disease and health reside in complex ecological interactions that are further affected by social actions. The typical indices of wild animal health (such as fecundity, distribution and abundance) are also typical measures of population ecology. An integrated health-focused approach is needed to find modifiable risk factors to reduce risks of disease exchange as well as to understand the implications of disease on salmon as a component of ecosystems.

Sub-recommendation 5e: Team fish health staff with hatchery staff for ongoing evaluation and adaptation of fish health management plans for specific facilities.

Rationale: An auditing program (self-audits and periodic external audits) should be developed for the plans. Auditing can be done internally by having teams of fish health staff and members of hatchery staff visit hatcheries other than the ones where the hatchery staff work. This would allow for sharing of lessons across facilities and co-learning. Periodic independent external audits would provide public assurance of quality control on the audit process.

Recommendation 6: Invest in fish health.

Rationale: The amount of fish health resources available is not consistent with the social and ecological responsibility that comes with fish health management decisions. Tens of millions of salmonids in BC, some belonging to threatened and endangered groups, are released every year. The fish health staff are extremely busy and are often left with inadequate time to deal with the tasks expected of a professional animal health program, often leaving them only with the time to service the most pressing needs.

Sub-recommendation 6a: To achieve the necessary auditing and oversight, DFO and the FFSBC must invest in human resources for fish health in BC.

Recommendation 7: Increase the understanding of the specific fish health needs and risks of CEDP and PIP programs.

Rationale: All of these recommendations will be more easily implemented for major DFO and FFSBC hatcheries and the larger CEDP hatcheries. We suspect logistical and human resource challenges will make them more difficult to apply to PIP facilities and spawning channels. We lacked the time and data to determine if the risks related to CEDP and PIP

are systematically different than for major enhancement facilities, but it appeared that the management was not equivalent across all facilities.

Sub-recommendations 7a: Form and support a working group that has the time and resources to examine the practices and potential risks at CEDP facilities, PIP facilities and spawning channels to determine if their risks require the application of the same recommendations above or additional risk reduction steps are needed.

Sub-recommendation 7b: A focused project to assess the fish health management needs for CEDP and PIP hatcheries or incubation systems should be conducted immediately to determine if existing standard operating procedures sufficiently address fish health issues as they relate to the potential to release pathogens from salmonid enhancement facilities.

Research Recommendations

Recommendation 8: Identify the health standard for acceptable risk.

Rationale: Measuring and monitoring risk cannot be achieved without an identifiable target. Since zero risk is not feasible as long as salmonid enhancement facilities exist and there is no social, legal or scientific certainty regarding acceptable risk, participatory research must be undertaken to define acceptable risk. An interdisciplinary project that involves methodologies suited to integrating uncertainty, scientific information and social values may be an important step towards developing advice on criteria for acceptable disease risk for enhancement facilities.

Sub-recommendation 8a: Investment in applied research to define processes, methods and outcomes for setting risk standards for fish health is strongly encouraged.

Recommendation 9: Create capacity for evidence-based decision making to support the management recommendations above plus ongoing fish health activities.

Rationale: Best management practices are currently based largely on experience and expert opinion; both can provide important insights into best practices, but modern standards require evidence-based or evidence-informed approaches to developing best practices. Best practices are also continually evolving and should be revisited and revised regularly once drafted.

Sub-recommendation 9a: Create applied research capacity in the DFO and FFSBC fish health units.

Rationale: There are large deficits in some core research areas that are need to be addressed to shift fish health management to an evidence-based best practices approach including: (1) Clinical trials, observational studies and systematic reviews of existing evidence; (2) Clinical epidemiological studies on the performance characteristics of diagnostic tests and diagnostic protocols coupled with prevalence studies within salmonid enhancement facilities to inform diagnostic testing regimes for surveillance and assurance of absence of disease; (3) Follow-up studies of decision algorithms for pre-release screening to establish the reliability of the algorithms and (4) Development and assessment of methods for surveillance in wild fish

Recommendation 10: Invest in research targeting the core uncertainties preventing assessment of disease risks to free-ranging salmon.

Rationale: Debates regarding the potential impacts of salmonid enhancement on wild fish have been ongoing without resolution for decades in the Pacific Northwest and British Columbia. We found no organized effort to take a system-view of this issue and to facilitate collaborative and integrative studies. In the absence of the required evidence, the need and nature of management decisions to prevent or reduce disease risk reduction will remain contentious and speculative.

Sub-recommendation 10a: Support research to address the 3 outstanding research issues that prevented the assessment of disease associated risks in this case.

Additional details: Outstanding research issues include:

1. How does one accurately account and track the distribution and variation of infectious and parasitic agents in cultured and free-ranging fish?
2. What are the conditions for an effective exposure of a susceptible fish to an infectious fish and how often are those conditions met in the interactions of Fraser River sockeye salmon with enhanced salmonids?
3. What is the proportional role of disease as an independent or additional stressor that influences population health and production of Fraser River sockeye salmon?

Sub-recommendation 10b: Support a working group that has time to consult, review past work and identify candidate teams, methods and processes to define the research agenda.

Additional details: It is beyond the scope of this report to prescribe the research agenda to address these questions. We envision a problem oriented approach that engages a suite of biological, social, and physical sciences necessary to develop a systems-based understanding of the determinants and impacts of Fraser River sockeye salmon health and disease at an individual and population level. These are complex questions that will require novel approaches. We advise against an immediate call for research proposals without due consideration by such a working group. The working group would be tasked not just with thinking about research methods but also in how to develop strategic partnerships and stakeholder engagement to identify those questions and methods that will address the prevailing uncertainties in a feasible and acceptable manner. Their recommendations should include a process with which diverse research outcomes can be integrated into a comprehensive perspective on salmon health. These recommendations for a socio-ecological approach should not come at the cost of laboratory based research that provides critical information on host and pathogens determinants of health outcomes.

Recommendation 11: DFO, the FFSBC and other interested stakeholders lobby national and regional funding agencies such as the National Sciences and Engineering Research Council, foundations and government to secure the required long-term funding to support the necessary research.

Rationale: Ecological questions, such as the role of disease as a population regulation factor, take time. Long term funding has become rare in Canada in recent years. Agencies interested in understanding disease and salmon need to invest in a long term research program and vision.

References

Adler FR and Brunet RC. 1991. The dynamics of simultaneous infections with altered susceptibilities. Theoretical Population Biology 40: 369-410.

Alderstein SA and Dorn MW. 1998. The effect of *Kudoa paniformis* infection on the reproductive effort of female Pacific hake. Canadian Journal of Zoology 76: 2285-2289.

Allendorf FW and Phelps SR. 1980. Loss of genetic variation in a hatchery stock of cutthroat trout. Transactions of the American Fisheries Society 109:537-554.

Altizer S, Harvell D and Friedle E. 2003. Rapid evolutionary dynamics and disease threats to biodiversity. Trends in Ecology and Evolution 18: 589-596.

American Fisheries Society. 2007. Suggested Procedures for the Detection and Identification of Certain Finfish and Shellfish Pathogens. Blue Book 2007 Edition. Fish Health Section, American Fisheries Society.

Amos K, and Thomas J. 2002. Disease interactions between wild and cultured fish: Observations and lessons learned in the Pacific Northwest. Bulletin of the European Association of Fish Pathologists 22:95-102.

Anderson RM. 1991. Populations and infectious diseases: ecology or epidemiology? Journal of Animal Ecology 60: 1-50.

Anderson RM and May RM. 1979. Population biology of infectious diseases. Part 1. Nature 280: 361-367.

Anderson ED, Engelking H, Emmenegger M, Eveline J and Kurath, G. 2000. Molecular epidemiology reveals emergence of a virulent Infectious Hematopoietic Necrosis (IHN) virus strain in wild salmon and its transmission to hatchery fish. Journal of Aquatic Animal Health 12: 85- 99.

Anonymous. 2009. Evaluation of the Salmonid Enhancement Program No. 6B105. Fisheries and Oceans Canada.

Anonymous –DFO. Guidelines for In-Stream Placement of Hatchery Salmon Carcasses for Nutrient Enrichment. Available at : http://www.pac.dfo-mpo.gc.ca/publications/pdfs/carcass-carcasse-guide-eng.pdf. Accessed July 11, 2011.

Apanius V and Schad GA. 1994. Host behaviour and the flow of parasites through host populations. pp. 115-128. In: M.E. Scott & G. Smith (ed.) Parasitic and Infectious Diseases: Epidemiology and Ecology, Academic Press, Toronto.

Arkoosh MR, Clemons E, Kagley AN, Stafford C, Glass AC, Jacobson K, Reno P, Myers MS, Casillas E, Loge F, Johson LL and Collier TK. 2004. Survey of pathogens in juvenile salmon *Oncorhynchus* Spp. migrating through Pacific Northwest estuaries. Journal of Aquatic Animal Health 16: 186-196.

Arkush KD, Giese AR, Mendonca HL, McBride AM, Marty GD and Hedric PW. 2002. Resistance to three pathogens in the endangered winter-run Chinook salmon (*Oncorhynchus tshawytscha*): effects of inbreeding and major histocompatibility complex genotypes. Canadian Journal of Fisheries and Aquatic Sciences 59: 966–975.

Arnott S, Barber I and Huntingford F. 2000. Parasite–associated growth enhancement in a fish–cestode system. Proceedings of the Royal Society of London B 267: 657-663.

Arsan EL. 2006. Potential for Dispersal of the Non-native Parasite *Myxobolus cerebralis*: Qualitative Risk Assessments for the State of Alaska and the Willamette River Basin, Oregon. Oregon State University MSc Abstract on-line. Available at : http://scholarsarchive.library.oregonstate.edu/xmlui/bitstream/handle/1957/3843/Arsan.pdf?sequence=7. Accessed July 11, 2011

Bailey RE and Margolis L. 1987. Comparison of parasite fauna of juvenile sockeye salmon (*Oncorhynchus nerka*) from southern British Columbia and Washington State lakes. Canadian Journal of Zoology 65: 420-431.

Bakke TA and Harris PD. 1998. Diseases and parasites in wild Atlantic salmon (*Salmo salar*) populations. Canadian Journal of Fisheries and Aquatic Science 55(Supp.1): 247-266.

Balfry S, Welch DW, Atkinson J, Lill A and Vincent S. 2011. The effect of hatchery release strategy on marine migratory behaviour and apparent survival of Seymour River steelhead smolts (*Oncorhynchus mykiss)*. PLoS ONE 6(3): e14779. doi:10.1371/journal.pone.0014779.

Bartholomew J. 2010. Reducing disease risks caused by pathogens associated with Columbia River hatcheries (presentation). State of the Salmon.

Bartley DM, Bondad-Reantaso MG and Subasingh RP. 2006. A risk analysis framework for aquatic animal health management in marine stock enhancement programmes. Fisheries Research 80: 28–36.

Berube M and Curtis M. 1986. Transmission of *Diphyllobothrium ditremum* to Arctic char (*Salvelinus alpinus*) in two subarctic Quebec Lakes. Canadian Journal of Fisheries and Aquatic Science 43: 1626-1634.

Bradford MJ, Lovy J, Patterson DA, Speare DJ, Bennett WR, Stobbart AR, Tovey CP. 2010. *Parvicapsula minibicornis* infections in gill and kidney and the premature mortality of adult sockeye salmon (*Oncorhynchus nerka*) from Cultus Lake, British Columbia. Canadian Journal of Fisheries and Aquatic Sciences 67: 673-683.

Epidemiologic Surveillance. Encyclopedia of Public Health. Ed. Lester Breslow and Gale Cengage, 2002. Available at: http://www.enotes.com/public-health-encyclopedia/epidemiologic-surveillance [Accessed June 27, 2011].

Bristow GA and Berland B. 1991. A report on some metazoan parasites of wild marine salmon (*Salmo salar* L) from the west coast of Norway with comments on their interaction with farmed salmon. Aquaculture 98: 311-318.

British Columbia Ministry of Agriculture. 2004. Fish Health Management Plan. Available at: http://www.al.gov.bc.ca/ahc/fish_health/fish_health_management_plan.htm [Accessed June 27, 2011].

British Columbia Salmon Farmers Association. Fish Health Database Quarterly Reports. Available at: http://www.agf.gov.bc.ca/ahc/fish_health/BCSFA/BCSFA_Fish_Health_Quarterly_Report_Review.pdf [Accessed June 27, 2011].

Brown R. 2003. Oroville Facilities Relicensing (FERC Project No. 2100). SP-F9 Evaluation of Project Effects on Natural Salmonid populations. Phase 1 Interim Literature Review.

Burgner RL. 1991. Life history of sockeye salmon *(Oncorhynchus nerka).* In: Pacific Salmon Life Histories. C. Groot and L. Margolis (Eds.). UBC Press, Vancouver.

Canadian Science Advisory Secretariat. 2010. Assessment of Cultus Lake sockeye salmon in BC in 2009 and evaluation of recent recovery activities. Science Advisory Report 2010/056.

Carey T. 2005. Introductions and transfers of aquatic animals – a survey of procedures and activities in Canada (2002-2005). Report to Aquaculture Management Directorate. Fisheries and Oceans Canada.

Centre for Disease Control. 2001. Updated guidelines for evaluating public health surveillance systems. US Centres for Disease Control . MMWR. 50(RR13):1-35.

Committee on the Status of Endangered Wildlife in Canada. 2003. COSEWIC assessment and status report on the sockeye salmon *Oncorhynchus nerka*

(Cultus population) in Canada. Committee on the Status of Endangered Wildlife in Canada.Ottawa. ix + 57 pp.

Cone DK, Beverley-Burton M, Wiles M and McDonald TE. 1983. The taxonomy of *Gyrodactylus* (Monogenea) parasitizing certain salmonid fishes of North America, with a description of *Gyrodactylus nerkae* n. sp. Canadian Journal of Zoology 61: 2587-2597.

Cvitanich JD, Garate O and Smith CE. 1991. The isolation of a rickettsia-like organism causing disease and mortality in Chilean salmonids and its confirmation by Koch's postulate. Journal of Fish Disease 14: 121-145.

Davis K., Drey N and Gould D. 2009. What are scoping studies? A review of the nursing literature. International Journal of Nursing Studies 46: 1386-1400.

Dobson, A. P., May, R.M., 1987. The effects of parasites on fish populations-theoretical aspects. Int. J.Parasitol. 17, 363-370.

Dohoo I, Martin W and Stryhn H. 2003. Veterinary Epidemiological Research. AVC Inc. Charlottetown.

Donaldson MR, Hinch SG, Patterson DA, Farrell AP, Shrimpton JM and Miller-Saunders KM. 2010. Physiological condition differentially affects the behaviour and survival of two populations of sockeye salmon during their freshwater spawning migration. Physiological and Biochemical Zoology 83:446–458.

Donas B, Newman N. 2008. Sockeye parasite sampling program 2008. At Fulton, Pinkut and Nadina spawning channels and the Babine River fence. Oceans Habitat and Enhancement, Northcoast B.C.

Dowling D, McMahon T, Kerans B and Vincent R. 2002. Relation of spawning and rearing life history of rainbow trout and susceptibility to *Myxobolus cerebralis* infection in the Madison River, Montana. Journal of Aquatic Animal Health 14:191–203.

Eaton W and Kent M. 1992. A retrovirus in Chinook salmon (*Onchorynchus tschwytscha*) with evidence of plasmacytoid leukemia and evidence for the etiology of the disease. Cancer Research 52: 6496-6500.

Evans AS. 1976. Causation and Disease: The Henle-Koch Postulates Revisited, Ⅱ. Yale Journal of Biology and Medicine 49: 175-95.

Faisal M and Winters A. 2011. Detection of Viral Hemorrhagic Septicemia virus (VHSV) from *Diporeia* spp. (Pontoporeiidae, Amphipoda) in the Laurentian Great Lakes, USA. Parasites and Vectors 4:2.

Fenichel E, Tsao J, Jones J and Hockling G. 2008. Fish pathogen screening and its influence on the likelihood of accidental pathogen introduction during fish translocations. Journal of Aquatic Animal Health 20:19–28.

Fevre EM, de C Bronsvorrt BM, Hamilton K and Cleaveland S. 2006. Animal movements and the spread of infectious disease. Trends in Microbiology 14: 125-131.

Firestone J. 2006. Dilemmas and dimensions of non-indigenous organisms and pathogens in the marine environment: A sea change**.** Journal of International Wildlife Law and Policy 9:123-132.

Fisheries and Oceans Canada. 1984 (revised 2004). Fish Health Protection Regulations: Manual of Compliance. Fisheries and Marine Service Miscellaneous Special Publication 31 (revised) pp.50.

Flagg TA, Berejikian BA, Colt JE, Dickhoff WW, Harrell LW, Maynard DJ, Nash CE, Strom MS, Iwamoto RN, Mahnken CVW. 2000. Ecological and behavioural impacts of artificial production strategies on the abundance of wild salmon populations. U.S. Department of commerce, NOAA Technical Memorandum NMFS-NWFSC-41, 92pp.

Foott JS, Free D, McDowellT, Arkush KD and Hedrick RP. 2006. Infectious Hematopoietic Necrosis virus transmission and disease among juvenile Chinook salmon exposed in culture compared to environmentally relevant conditions. San Francisco Estuary and Watershed Science, 4(1) Art 2. Available at: http://repositories.cdlib.org/jmie/sfews/vol4/iss1/art2 [Accessed June 27, 2011].

Gardner J, Peterson DL, Wood A and Maloney V. 2004. Making sense of the debate about hatchery impacts: Interactions between enhanced and wild salmon on Canada's Pacific coast. Report to the Pacific Fisheries Resource Conservation Council.

Garver K. 2010. Hypothesis: Diseases in freshwater and marine systems are important contributors to the Fraser sockeye situation. Submitted to the Cohen Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River. DFO-420606[01-02]. CAN319632_0001

Glover KA, Hamre LA, Skaala Ø and Nilsen F. 2004. A comparison of sea louse (*Lepeophtheirus salmonis*) infection levels in farmed and wild Atlantic salmon (*Salmo salar* L.) stocks. Aquaculture 232: 41-52.

Gochfeld M and Burger J. 1993. Evolutionary consequences for ecological risk assessment and management. Environmental Monitoring and Assessment 28:161-168.

Gonzalez L, Juan Carvajal J and George-Nascimento M. 2000. Differential infectivity of *Caligus flexispina* (Copepoda, Caligidae) in three farmed salmonids in Chile. Aquaculture 183: 13-23.

Good C.M, Thorburn MA and Stevenson RMW. 2001. Host factors associated with the detection of *Aeromonas salmonicida* and *Yersinia ruckeri* in Ontario, Canada government fish hatcheries. Preventive Veterinary Medicine 49:165-173.

Gozlan RE, Peelerb EJ, Longshaw M, St-Hilaire S and Feist SW. 2006. Effect of microbial pathogens on the diversity of aquatic populations, notably in Europe. Microbes and Infection 8:1358-1364.

Greiner M and Gardner IA. 2000. Epidemiologic issues in the validation of veterinary diagnostic tests. Preventive Veterinary Medicine 45: 3-22.

Halpenny C and Gross M. 2008. Macro- and microparasite infection profiles of hatchery fish before and after release from a conservation hatchery (steelhead trout, *Oncorhynchus mykiss*). Ecoscience 15:417-422.

Hammell L, Stephen C, Bricknell I, and Evensen O. 2009. *Salmon Aquaculture Dialogue Working Group Report on Salmon Disease* commissioned by the Salmon Aquaculture Dialogue, World Wildlife Fund. Available at: http://wwf.worldwildlife.org/site/PageNavigator/SalmonSOIForm [Accessed June 27, 2011].

Hänninen ML, Ridell J and Hirvela-Koski V. 1995. Phenotypic and molecular characteristics of *Aeromonas salmonicida* subsp.*salmonicida* isolated in Southern and Northern Finland. Journal of Applied Microbiology 79: 12-21.

Hanson RP. 1988. Koch is dead. Journal of Wildlife Disease 24:193-200.

Heard WR, Shevlyakov E, Zikunova OV and McNicol RE. 2007. Chinook salmon – trends in abundance and biological characteristics. North Pacific Anadromous Fish Commission 4: 77–91.

Hedrik R. 1998. Relationship of the host, pathogen and environment: Implications for diseases of cultured and wild fish populations. Journal of Aquatic Animal Health 10:107-111.

Hilborn R and Hare SR. 1992. Hatchery and wild fish production of anadromous salmon in the Columbia River basin. Report of the Fisheries Research Institute, School of Fisheries, University of Washington. FRI-UW-9107. September. https://digital.lib.washington.edu/researchworks/bitstream/handle/1773/4173/9207.pdf?sequence=1 [Accessed June 27, 2011].

Hinch S, Larsson S, Crossin G, Mathes T, Wagner G, Cooke S, Farrell T, English K, Welch D, Jones S and Patterson D. In: Scott G. Hinch and Julie Gardner (Eds.), Proceedings of the conference on early migration and premature mortality in Fraser River late-run sockeye salmon. Vancouver June, 2008.

Hinch S and Martins EG. 2011. A review of the potential climate change effects on survival of Fraser River sockeye salmon and an analysis of interannual trends on en route loss and pre-spawning mortality. Technical Report 9. Cohen Commission on the Inquiry into Decline of Sockeye Salmon in the Fraser River. Available at: http://www.cohencommission.ca/en/pdf/TR/Project9-Report.pdf#zoom=100 [Accessed June 27, 2011].

Holmes JC. 1995. Population regulation: a dynamic complex of interactions. Wildlife Research 22: 11-19.

Johnsen BO and Jensen AJ. 1991. The *Gyrodactylus* story in Norway. Aquaculture 98: 289-302.

Jones S, Prosperi-Porta G, Dawe S and Barnes D. 2003. Distribution, prevalence and severity of *Parvicapsula minibicornis* infections among anadromous salmonids in the Fraser River, British Columbia, Canada. Diseases of Aquatic Organisms 54:49-54.

Jones S, Prosperi-Porta G, Kim E, Callow P and Hargreaves BN. 2006. The occurrence of *Lepeophteirus salmonis* and *Caligus clemensi* (Copepoda:Caligidae) on three-spine stickleback *Gasterosteus aculeatus* in coastal British Columbia. Journal of Parasitology 92: 473–480.

Karpenko VI, Kovalenko MN, Erokhin VG, Adamov AA., Dekshtein AB and Subbotin SI. 2005. Abundance estimates of juvenile pacific salmon in the Eastern Okhotsk Sea and Western Bering Sea. North Pacific Anadromous Fish Commission Technical Report #6.

Kaufman JR. 2010. Studies on Prevention and control of Infectious Diseases of Salmonids. Report from Oregon Department of Fish and Wildlife. PROJECT NUMBER: F-104-R-30

Kelso JRM, Steedman RJ and Stoddart S. 1996. Historical causes of changes in Great Lakes fish stocks and the implications for ecosystem rehabilitation. Canadian Journal of Fisheries and Aquatic Sciences 53(Supp. 1): 10-19.

Kent ML, Whitaker DJ, Dawe SC. 1997. *Parvicapsula minibicornis* n. sp. (Myxozoa,

Myxosporea) from the kidney of sockeye salmon (*Oncorhynchus nerka*) from British Columbia. Journal of Parasitology. 83:1153-1156.

Kent ML, Traxler GS, Kieser D, Dawe SC, Shaw RW, Prosperi-Porta G, Ketcheson J and Evelyn TPT. 1998. Survey of salmonid pathogens in ocean-caught fishes in British Columbia, Canada. Journal of Aquatic Animal Health 10: 211-219.

Kent M. 2011. Infectious diseases and potential impacts on survival of Fraser River sockeye salmon. Cohen Commission Tech. Rept. 1: 58p. Vancouver, B.C. Available at: www.cohencommission.ca [Accessed June 27, 2011].

Kitron U. 2000. Risk maps: Transmission and burden of vector-borne diseases. Parasitology Today 16:324-325.

Kohler SL and Hoiland WK. 2001. Population regulation in an aquatic insect: The role of disease. Ecology 82:2294–2305.

Kohler SL and Wiley MJ. 1997. Pathogen outbreaks reveal large-scale effects of competition in stream communities. Ecology 78: 2164-2176.

Koopman JS and Lynch JW. 1999. Individual causal models and population systems models in epidemiology. American Journal of Public Health 89: 1170-1174.

Leighton FA. 2002. Health risk assessments of the translocation of wild animals. Revue Scientifique et Technique-Office International des Epizooties 21: 187-195.

Levin PS, Zabel RW and Williams JG. 2001. The road to extinction is paved with good intentions: Negative association of fish hatcheries with threatened salmon. Proceedings: Biological Sciences 1472: 1153-1158.

Linder G, Little E, Peacock B, Goeddeke H, Johnson L and Vishy C. 2005. Risk and Consequence Analysis Focused on Biota Transfers Potentially Associated with Surface Water Diversions Between the Missouri River and Red River Basins [CD]. U.S. Geological Survey, Columbia, Missouri.

MacKinlay DD, Lehman S, Bateman J, and Cook R. 2004. Pacific Salmon Hatcheries in Canada. In: M. J. Nickum, P. M. Mazik, J. G. Nickum & D. D. MacKinla (Eds.), American Fisheries Society Symposium 44, pp. 57-75.

Marina CF, Fernandez-Salas I, Ibarra JE, Arredondo-Jimenez JI, Valle J and Williams T. 2005 .Transmission dynamics of an iridescent virus in an experimental mosquito population: the role of host density. Ecological Entomology 30: 376-382.

Marcogliese DJ. 2002. Food webs and the transmission of parasites in marine fish. Parasitology 124: 83-99.

Marty GD, Quinn TJ, Carpenter G, Meyers TR and Willits NH. 2003. Role of disease in abundance of a Pacific herring (*Clupea pallasi*) population. Canadian Journal of Fisheries and Aquatic Science 60:1258–126.

Mazur C, Iwama G. 1993. Handling and crowding stress reduces number of plaque-forming cells in Atlantic salmon. Journal of Aquatic Animal Health 5: 98-101.

Maule AG, Rondorf DW, Beeman J and Haner P. 1996. Incidence of *Renibacterium salmoninarum* infections in juvenile hatchery spring Chinook salmon in the Columbia and Snake Rivers. Journal of Aquatic Animal Health 8: 37-46.

MacDonald TE and Margolis L. 1995. Synopsis of the parasites of fishes of Canada: Supplement (1978-1993). Canadian Special Publication of Fisheries and Aquatic Sciences 122.

McDaniel T, Pratt K, Meyers T, Ellison T, Follett J and Burke J. 1994. Alaska Sockeye Salmon Culture Manual. Alaska Department of Fish and Game. Special Publication No. 6.

McMahon T, Robison Cox J, Rotella J, Horton T, Kearans B. 2010. Toru population response to whirling disease epizootics in Montana rivers. Wild Trout X Symposium – Conserving Wild Trout. Available from: http://www.montana.edu/~wwwbi/staff/mcmahon/Wild%20Trout%20pdf.pdf. Accessed July 11, 2011

McIntyre PB. 1996. Environmental stress and colonization time as predictors of the susceptibility of fish communities to introduced parasites. Journal of Undergraduate Science 3:75-77. Available at: http://www.hcs.harvard.edu/~jus/0302/mcintyre.pdf. [Accessed June 27, 2011].

McMicheal GA and Pearson TN. 1998. Effects of wild juvenile spring Chinook salmon on growth and abundance of wild rainbow trout. Transactions of the American Fisheries Society 127: 261-274.

McVicar AH, Sharp LA, Walker AF and Pike AW. 1993. Diseases of wild seatrout in Scotland in relation to fish population decline. Fisheries research 17: 175-185.

McVicar AH. 1997. Disease and parasite implications of the coexistence of wild and cultured Atlantic salmon populations. ICES Journal of Marine Science 54:1093-1103.

Mesa MG, Poe TP, Maule AG and Schrek CB. 1998. Vulnerability to predation and physiological stress response in juvenile Chinook salmon (*Oncorhynchus tshawytscha*) experimentally infected with *Renibacterium salmoninarum*. Canadian Journal of Fisheries and Aquatic Science 55:1599-1606.

Michael J. 2003. Nutrients in salmon hatchery wastewater and its removal through the use of a wetland constructed to treat off-line settling pond effluent. Aquaculture 226: 213-225.

Mitchell AJ. 2001. Finfish health in the United States (1609-1969): historical perspective, pioneering researchers and fish health workers, and annotated bibliography. Aquaculture 196:347-43.

Moles A and Heifetz J. 1998. Effects of the brain parasite *Myxobolus arcticus* on sockeye salmon. Journal of Fish Biology 52: 146-151.

Moller H and Anders K. 1986. Diseases and Parasites of Marine Fishes, Verlag Moller, Kiel. 365 pp.

Moore J. 1995. The behavior of parasitized animals. BioScience 45: 89-96.

Morabia A. 1991. On the origins of Hill's Causal Criteria. Epidemiology 2: 367-369.

Mosquera J, de Castro M and Gomes-Gesteria M. 2003. Parasites and biological tags of fish populations: Advantages and limitations. Comments on Theoretical Biology 8:69-91.

Munson DA, Elliott DG and Johnson K. 2010. Management of bacterial kidney disease in Chinook salmon hatcheries based on broodstock testing by enzyme-linked immunosorbent assay. A multi-year study. North American Journal of Fisheries Management 30:940-955.

Myers RA, Levin SA, Lande R, James FC, Murdoch WW and Paine RT. 2004. Ecology. hatcheries and endangered salmon. Science 303(5666): 1980.

Naish KA, Taylor JE, Levin PS, Quinn TP, Winton JR, Huppert D and Hilborn R. 2008. An evaluation of the effects of conservation and fishery enhancement hatcheries on wild populations of salmon. Advances in Marine Biology 53: 61-194.

Nelitz M, Porter M, Parkinson E, Wieckowski K, Marmorek D, Bryan K, Hall A and Abraham D. 2011. Evaluating the status of Fraser River sockeye salmon and role of freshwater ecology in their decline. Cohen Commission Technical Report 3. Vancouver, B.C. Available at: www.cohencommission.ca [Accessed June 27, 2011].

Nielsen J, 2003. History and effects of hatchery salmon in the Pacific. Proceedings World Summit on Salmon, Chapter 15. Available at: http://www.sfu.ca/cstudies/science/resources/summit/pdf/Groupings/III_Threats.pdf [Accessed June 13, 2011]

Noakes DJ, Beamish RJ and Kent ML. 2000. On the decline of Pacific salmon and speculative links to salmon farming in British Columbia. Aquaculture 183: 363-386.

Noga EJ. 1996. Fish Disease. Diagnosis and Treatment. Mosby. Toronto.

Nylund A, Plarre H, Karlsen M, Fridell F, Ottern KF, Bratland A and Saether PA. 2007. Transmission of Infectious Salmon Anaemia virus (ISAV) in farmed populations of Atlantic salmon (*Salmo salar*). Archives of Virology 152: 151-179.

OIE. World Organization for Animal Health. 2006. Aquatic Animal Health Code. Available at: http://www.oie.int/eng/normes/fcode/en_sommaire.htm [Accessed June 27, 2011].

Orsi JA, Fergusson EA, Sturdevant MV, Wing BL, Wertheimer AC and Heard WR. 2008. *Annual Survey of Juvenile Salmon and Ecologically Related Species and Environmental Factors in the Marine Waters of Southeastern Alaska, May–August 2007* No. NPAFC Doc. 1110). 17109 Point Lena Loop Road, Juneau, AK 99801 USA: Auke Bay Laboratories, Alaska Fisheries Science Center, NOAA Fisheries, United States Department of Commerce, Ted Stevens Marine Research Institute.

Ostfeld RS and Holt RD. 2004. Are predators good for your health? Evaluating evidence for top-down regulation of zoonotic disease reservoirs. Frontiers in Ecology and the Environment 2: 13–20.

Patterson D, Covy J, McKay D, Bradford M and Bennett B. 2009. Disease and pre-spawning mortality for late-run sockeye. In: Scott G. Hinch and Julie Gardner (Eds.), Proceedings of the

conference on early migration and premature mortality in Fraser River late-run sockeye salmon. Vancouver June, 2008.

Rand P.S. 2008. *Oncorhynchus nerka*. In: IUCN 2011. IUCN Red List of Threatened Species. Version 2011.1. Available at: www.iucnredlist.org [Accessed June 27, 2011].

Reno P. 1998. Factors involved in the dissemination of disease in fish populations. Journal of Aquatic Animal Health 10: 160-171.

Rhodes L, Durkin C, Nance S and Rice C. 2006 Prevalence and analysis of *Renibacterium salmoninarum* infection among juvenile Chinook salmon *Oncorhynchus tshawytscha* in North Puget Sound. Disease of Aquatic Organisms 71: 179-190.

Rintamäki-Kinnunen P, Valtonen ET. 1996. Finnish salmon resistant to *Gyrodactylus salaris*: a long-term study at fish farms. International Jpournal for Parasitology. 26(7): 723-732

Rodger HD, Drinan EM, Murphy TM and Lunder T. 1991. Observations on erythrocytic inclusion body syndrome in Ireland. Bulletin of the European Association of Fish Pathologists 11: 108-111.

Rodgers LJ and Burke JB. 1981. Seasonal variation in the prevalence of 'red spot' disease in estuarine fish with particular reference to the sea mullet, *Mugil cephalus* L. Journal of Fish Diseases 4: 297-307.

Sackett DL, Haynes RB, Guyatt GH and Tugwell R. 1991. Clinical Epidemiology: A Basic Science for Clinical Medicine. 2nd ed. Little, Brown and Company. Toronto

Sargeant, J. M., Amezcua, M. D. R., Rajic, A., & Waddell, L. (2005). *A guide to conducting systematic review in agri-food public health.*

Scott MC and Hall LW. 1997. Fish assemblages as indicators of environmental degradation in Maryland coastal plain streams. Transactions of the American Fisheries Society 126:349-360.

Sindermann, C.J., 1987. Effect of parasites on fish populations: practical considerations. Int. J. Parasitol. 17, 371-382.

Snieszko, SF. 1974. The effects of environmental stress on outbreaks of infectious diseases of fishes. Journal of Fish Biology 6: 197–208.

Starliper CE. 2011. Bacterial coldwater disease of fishes caused by *Flavobacterium psychrophilum*. Journal of Advanced Research 2:97-108.

St-Hilaire S, Boichuck M, Barnes D, Higgins M, Devlin RH, Withler RE, Khattra J, Jones, S, Kieser D. 2002. Epizootiology of *Parvicapsula minibicornis* in Fraser River sockeye salmon, *Oncorhynchus nerka* (Walbaum). Journal of Fish Disease. 25: 107–120.

St-Hilaire S, Ribble CS, Stephen C, Anderson E, Kurath G, Kent M. 2002. Epidemiological investigation of infectious hematopoietic necrosis virus in salt water net-pen reared Atlantic salmon in British Columbia, Canada. Aquaculture 212: 49-67.

Stephen C, Dawson-Coates J and DiCicco E. 2007. Pathogen Risks Associated with the Diversion of Water from Devil's Lake into the Red River Drainage. Report to the International Joint Commission

Stephen C and Thorburn M. 2002. Formulating a vision for fish health research in the Great Lakes. Available at: http://www.glfc.org/research/reports/stephen_fish_health.pdf [Accessed June 26, 2011].

Stephen RC and Ribble CS. 1995. The effects of changing demographics on the distribution of marine anaemia in farmed salmon in British Columbia. Canadian Veterinary Journal 36:557-562.

Stephen, C and Iwama G. 1997. Report to the B.C. Environmental Assessment Office Salmon Aquaculture Review. Health Issues.

Strak K and Salman M. 2002. Relationships between animal health monitoring and the risk assessment process. Acta Veterinaria Scandanavica 2001, Suppl. 94, 71-77.

Strom M, Harrell L and Johnson K. 2002. Infectious diseases in fresh and saltwater-reared salmon captive broodstock programs. In: Proceeding of the Workshop on Captive Broodstock recovery of Imperiled Salmonid Populations. Available at: http://www.krisweb.com/biblio/battle_xxxx_berejikianetal_2002_capbroodconfproc.pdf#page=16 [Accessed June 26, 2011].

Stoskopf MK. 1993. Fish Medicine. WB Saunders Company. Toronto.

Szalai AJ and Dick TA. 1991. Role of predation and parasitism in growth and mortality of yellow perch in Dauphin Lake, Manitoba. Transactions of the American Fisheries Society 120: 739-751.

Thorburn MA. 1996. Apparent prevalence of fish pathogens in asymptomatic salmonid populations and its effect on misclassifying population infection status. Journal of Aquatic Animal Health 10:143-151.

Thorburn MA. 1992. The randomness of samples collected by dip-net methods from rainbow trout in tanks. Aquaculture 101:385-390.

Tierney KB and Farrell AP. 2004. The relationships between fish health, metabolic rate, swimming performance and recovery in return-run sockeye salmon, *Oncorhynchus nerka* (Walbaum). Journal of Fish Diseases 27: 633-671.

Traxler G and Rankin J. 1989. An Infectious Hematopoetic Necrosis epizootic in sockeye salmon *Oncorhynchus nerka* in Weaver Creek spawning channel, Fraser River system, British Columbia. Diseases of Aquatic Organisms 6:221-226.

Traxler GS, Richard J and McDonald TE. 1998. *Ichthyophthirius multifiliis* (Ich) epizootics in spawning sockeye salmon in British Columbia, Canada. Journal of Aquatic Animal Health 10: 143-151.

Troyer RM and Kurtah G. 2003. Molecular epidemiology of Infectious Necrosis virus reveals complex virus traffic and evolution within southern Idaho aquaculture. Diseases of Aquatic Organisms 55: 175-185.

United Nations General Assembly. 1992. Report of the United Nations Conference on Environment and Development. Annex I Rio Declaration on Environment and Development. Available at: http://www.un.org/documents/ga/conf151/aconf15126-1annex1.htm [Accessed June 26, 2011].

United States Fish and Wildlife Service. 2003. Aquatic Animal Health Policy Overview. Available at: http://www.fws.gov/policy/713fw1.html [Accessed June 26, 2011].

van Dobben WH. 1952. The food of cormorants in the Netherlands. Ardea 40: 1-63.

Wagner GN, Hinch SG, Kuchel LJ, Lotto A, Jones SRM and Patterson DA. 2005.

Metabolic rates and swimming performance of adult Fraser River sockeye salmon (*Oncorhynchus nerka*) after a controlled infection with *Parvicapsula minibicornis*. Canadian Journal of Fisheries and Aquatic Sciences 62: 2124-2133.

Ward JR and Lafferty KD. 2004. The elusive baseline of marine disease: Are diseases in ocean ecosystems increasing? PLoS Biology 2: 0542-0547.

Winton J. Disease Risks Posed by Hatchery Salmon (presentation). US Geological Survey, Western Fisheries Research Centre, Seattle, Washington. Available at:

http://www.stateofthesalmon.org/conference2010/downloads/Wed_presentations/Session_1a/Winton%20-%20State%20of%20the%20Salmon%202010.pdf [Accessed June 26, 2011].

Appendix 1: Statement of Work

Scope of Work as outlined in the contract between the Centre for Coastal Health and Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River.

Statement of Work

Consulting and Professional Services

SW1 Background

1.1 The Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River

(www.cohencommission.ca) was established to investigate and report on the reasons for the decline and the long term prospects for Fraser River sockeye salmon stocks and to determine whether changes need to be made to fisheries management policies, practices and procedures.

1.2 An evaluation of the impacts of hatchery and spawning channel disease occurrence and frequency is required to determine their role in the reductions in Fraser sockeye productivity.

SW2 Objective

2.1 To review disease data and reports from salmon enhancement facilities operated by Canada and BC and evaluate the potential for a qualitative and/or quantitative assessment of the potential effect of diseases present in enhancement facilities on Fraser River sockeye salmon. The scientist will analyze fish disease frequency and mortality rate at, or adjacent to, hatcheries, spawning channels and aquatic ecosystems where hatchery fish are released. Diseases to be evaluated include communicable diseases like those due to parasites, bacteria and viruses but not non-infectious diseases.

2.2 Subject to a decision by the Commission regarding the feasibility of a qualitative and/or quantitative assessment related to the potential effect of diseases present in enhancement facilities on Fraser River sockeye salmon as described in 2.1, to assess the documented and potential effects of diseases present in enhancement facilities on Fraser River sockeye salmon. This will include the role of hatchery diseases in the 2009 run failure as well as the longer term decline in Fraser sockeye productivity over the past 20 years.

SW3 Scope of Work

3.1 The Contractor shall provide the services of Craig Stephen, Tyler Stitt and Jennifer Dawson-Coates [with later amendment to add Anne McCarthy] to review data, reports and other

information provided by the Commission. This will include information that the Commission receives from Canada and the Province of BC, relating to fish health, mortality and the occurrence of, monitoring of and response (including treatment, enforcement and authorizations) to pathogens (in particular, infectious hematopoietic necrosis virus, bacterial kidney disease, infectious salmon anaemia and furunculosis) in finfish hatchery and spawning channel facilities.

3.2 The Contractor will evaluate the potential for a qualitative and/or quantitative assessment of the potential effect of diseases present in enhancement facilities on Fraser River sockeye salmon. This evaluation will include an analysis of fish disease frequency and mortality rate at, or adjacent to, hatcheries, spawning channels and aquatic ecosystems where hatchery fish are released.

3.3 Subject to the outcome of the analyses in 3.1 and 3.2, the Contractor will evaluate, qualitatively and/or quantitatively, the disease risks posed by the operation of salmonid enhancement facilities on the production of Fraser River sockeye salmon.

SW4 Deliverables

4.1 The Contractor will organize a Project Inception meeting to be held within 2 weeks of the contract date in the Commission office. The meeting agenda will be set by the Contractor and will include a work plan for project implementation.

4.2 The main deliverables of the contract are: 1) a feasibility report that addresses Objective 2.1; and 2) contingent on the commission requesting the Contractor complete Objective 2.2 and section 3.3, a final report.

4.3 The feasibility report will be provided to the Cohen Commission in pdf and Word formats by May 1, 2011. A draft Final Report will be provided to the Cohen Commission in pdf and Word formats by June 30, 2011. The draft Final Report should contain an expanded Executive Summary of 1-2 pages in length as well as a 1-page summary of the “State of the Science”. Comments on the draft Final Report will be returned to the contractor by July 8, 2011 with revisions due by July 15, 2011.

4.4 Dr. Stephen will make himself available to Commission Counsel during hearing preparation and may be called as a witness.

Appendix 2: Report Peer Reviews and Responses

This section has been organized so that our responses are embedded in each section of the 3 reviewer's comments. The reviews have been kept in their original format. *Responses to the reviewers are presented in italics.*

REVIEWER 1
Report Title: Assessment of the potential effects of diseases present in salmonid enhancement facilities on Fraser River sockeye salmon
Reviewer Name: Sonja Saksida
Date: July 8, 2011

1. Identify the strengths and weaknesses of this report.

General comments

Strengths -

The methodology used by the authors to tackle the objectives was well laid out.

The authors provide a good discussion the significant problems with the quality/quantity of data, as well as the lack of baseline health data from the wild populations.

The authors give a very good description of the large variation in the operational standards and resources at these facilities as well as the diagnostic/screening capacity provided to these facilities. It is very apparent from this report that the resources and financial support available are suboptimal for these operations to function properly.

Weakness –

There were a lot of basic principles of epidemiology presented in this report, much of this material, although interesting, isn't essential and could be either removed or added as appendices.

I*t was our view that the readership of this report would include people with a lack of general knowledge of epidemiology and principles of infectious disease. We note below that Dr. Saksida appreciated the background on disease in the report and we suspect others might require the epidemiological background to understand the reasoning behind our interpretations and*

conclusions. We have therefore, opted to leave this information in the body of the text.

I would have liked to see more 'expert opinion' in the document with regard to health management at the different facility types and perhaps discussion of how health management in enhancement facilities in BC compares to other regions (i.e. various US states) as well as perhaps a comparison with management practices at private commercial facilities.

The evaluation of specific management practices was outside of the scope of work and timeframe for this project. The fish health management plans provided insufficient details of existing practices and there has been no systematic and controlled evaluation of their effectiveness (See Risk Assessment section – Fish Health Management Plans). We would be unable to comment on how these practices are applied and thus could only examine their intended use (as done in our section on Fish Health Management Plans). We did note how the BC fish health community is well connected to the fish health community in the Pacific Northwest and participate in continuing education. We also note that the fish health management plans are the current accepted standard (a broader opinion that just ours) but further note on the same page that "best practice" will need to be defined by site specific risks and production circumstances. Our Recommendation #1 is entirely consistent with Dr. Saksida's suggestion in that we recommend adaptive management based on ongoing learning from experiences and evaluation of management plans. We have modified this recommendation to indicate that this experience can be from within as well as outside of BC. Recommendation #10 also emphasizes the need for regional cooperation on research

I would have liked to see a critical evaluation as to the resources (human, lab and financial) provided to these facilities for fish health management. Again perhaps a comparison with other jurisdictions with enhancement facilities as well as private commercial facilities. Also, a discussion of how this may have changed over time.

All these elements are really important to put context to the data quality/quantity.

Data on financial support for the DFO or FFSBC was not provide nor requested and therefore cannot be added to revisions of this report. We do summarize the capacities of the labs in our risk assessment section (see sections: Data sources for hazard assessment and Validity of diagnostic tests). Data on program support were not requested originally and were not able to be retrieved during this review period

Our findings demonstrate that fish health staff at these labs are resource and time limited to do much more than respond to diagnostic cases. Therefore, regardless of their resources, there are not the resources to do more than the current work, including evolving to undertake a larger role

in health management. Sub-recommendation 6a reflects our opinion that more investment is required.

I also would have liked to see a breakdown of health issues presented and interpreted by year and possibly by region. Emphasis could have been the diseases Dr Kent suggested as important to sockeye.

A new appendix has been added to the report (Appendix 7) which presents the diagnostic data by region, source and year for all enhanced salmonids and for sockeye only. The reader will see from this that the number of facilities providing samples was relatively low and was inconsistent each year; and the average number of submissions per year in the data we received was small. We provided this appendix as the reviewers were all interested in looking for spatial and temporal trends. However, we caution the reader that these data are insufficient for trend analysis as we cannot differentiate variation in decisions to submit cases from differences in disease patterns, we cannot distinguish changes in disease patterns with changes in patterns of the population at risk, and we have not been able to confirm that all cases are included in the data we received as we did not have access to the original database.

For example

BKD is a very important disease in Pacific salmonids, including sockeye salmon. However not all broodstock are screened for BKD. I believe that screening is based on health information collected in the past. Acknowledging that disease occurrences are not constant, historical information to inform current management practices should be considered unreliable without continued monitoring and examine for change. I believe it would be important to describe and discuss more about BKD management at these facilities.

Evaluation of the effectiveness of various fish health programs was not within our statement of work. The DFO BKD management plan is summarized in the Exposure Assessment section (Release/escape of pathogens from enhancement facilities) and includes a reference to Munson et al, 2010 which provides some research results suggesting the DFO approach can be successful in reducing prevalence of BKD in salmonid hatcheries in the USA. The diagnostic database yielded only 97positive BKD results over the past decade from DFO facilities (see Table 14). We did not have information on how the BKD plan was implemented (which hatcheries, when and for how long). Moreover, we found no evidence that a controlled evaluation of this management program has been undertaken (ex. clinical trial). Often, with disease eradication and control, it is easy to reduce the disease from high to moderate or low levels. But removing the final cases can be costly and sometimes not possible. As there is no accepted standard of how much risk reduction is required to conclude the program effective,

there is no point of reference for evaluation of the effectiveness of the program, especially from a wild salmonid perspective. However, our recommendation 1, sub-recommendation 3b, recommendation 4 (and its sub-recommendations) and recommendation 9 all support the approach advocated by Dr Saksida.

Another known disease considered important to sockeye salmon is IHN. Other salmonid species may also be affected or act as carriers but sockeye are the most heavily impacted.

Here the authors did comment that they were surprised with the lack of data from the enhancement facilities. However, most sockeye are not reared in traditional hatchery settings but rather at spawning channels. And there has been a tremendous amount of work that has been done by Garth Traxler and others on IHN and diseases/infections in sockeye salmon in spawning channels. For example; G. Traxler of PBS had collected IHNv information from spawning channels (Weaver, Nadina, Fulton) for many years, which was not presented in this report. This data should be included in the report.

We undertook a secondary inventory of the files provide to us to reconfirm the nature of the data to us. Of the 3153 PDF files presented to us, 2590 involved information on diagnostic tests, screening or health management for DFO and 486 for FFSBC (this information has been added to the methods section) Only 82 sockeye salmon submissions were represented in the diagnostic data files and 2/3 came from Rosewell and Cultus Lake facilities. This information has now been presented after table 14.

We were reluctant to present data based on region or time because of the unequal "sampling effort" that resulted from unequal numbers of submissions from various facilities. For example, one hatchery (Spius) was responsible for the majority of submissions to the PBS lab for facilities in the Fraser Valley (table 12 and Appendix 7). We were unable to confirm if this reflected differences in disease prevalence, the care and attention staff placed on fish health, special topics/issues being investigated or other reasons for submissions. If issues of unequal sampling exist, any spatial or temporal trends would be biased.

We have created Appendix 7 which summarizes the number of submissions and fish for data available to us to illustrate the distribution of submissions in the data provided to us. This appendix shows the relatively low number of samples per year/facility (submissions being the unit of analysis for facility level diagnostics). Appendix 7 breaks the diagnostic data down by region, year and number of hatcheries or spawning channels submitting. This demonstrates the lack of information for spawning channels in the diagnostic files provided to us. We have included a new section in the Hazard Assessment specific to spawning channels. We note that

Traxler's studies are discussed in the literature review.

More specific comments

Background /Introduction

The authors provide a very good background on the types and number of facilities that are considered as enhancement facilities in British Columbia.

The authors may want to provided more background on the FFSBC (eg. used to be operated by the provincial government but now operates as a not for profit)

Some additional background is provided on page 12

The authors provide a good introduction to disease, types of disease, the role of disease and possible outcomes. The authors also clearly differentiate terminologies (i.e. disease versus infection). I would suggest that it would be valuable for the authors to discuss disease patterns in this section (cyclical, seasonal, erratic, etc) as well as stress that if nothing else, disease is not predictable and that baseline data collected in the past may not have relevance to current situations.

We believe that the first section in the Literature review (Linking effects with cause: how do we define effects) introduces the challenges in predicting disease patterns as do the reference from Koopman and Lynch (1999) and our section on impacts of environmental change. Most importantly, we have a section titled "Ecological and epidemiological variability prevent consistent prediction" that emphasizes Dr Saskida's concern. We therefore did do repeat this information in the introduction.

Literature Review

In their literature review the authors provide a good discussion on the limited amount of disease studies in free-ranging fish and the problem with using analogies since diseases/infections vary between species.

They provide a good discussion of possible ways enhancement facilities may influence the health of wild populations. There however does not appear to be much discussion regarding stream seeding practices (placing salmon carcasses in streams for nutrient enrichment).

Note in our Methodology section (Risk assessment Objective 3) that a request for information on carcass use in stream nutrient enrichment was made. The only information we received was one policy document and information from discussions with Mr Mark Higgins (see section in Risk

Assessment – Pathogen movement with fish movement). We have added this possible route of exposure in the section "How might salmonid enhancement facilities affect Fraser River sockeye salmon disease status".

Experimental Evidence Studies (pg 40-45)

This section is interesting but could be condensed or alternatively added to the appendix.

Experimental evidence provides the cornerstone for most discussion about impacts of salmon diseases. As we point out, it is experimental effects and the effects in fish culture facilities that generate most information informing attempted risk assessments. We are concerned that relegating this information to an appendix would provide an unbalanced understanding of the information available for risk assessment for readers unfamiliar with this topic and have chosen to leave it in its original location.

The authors provide details on the source of data.

With regard to BC Salmon Farmers database, as the authors indicated, the original intent of the database was to have enhancement facilities participate. However, even though the quarterly summary reports rolled up commercial and enhancement facility data, individual enhancement facility data was entered into the database and could have been accessed.

We based our interpretation of data availability on the information provided on the website describing this data source and thus were unaware that it could have been disaggregated and assessed on a site specific basis. We did not pursue these data as we assumed that the diagnostic data provided to us from DFO and FFSBC would be the basis of these reports and we had access to those data.

On pg 58, the authors speak of the lab accreditation program (ISO-17025); however, this has only been in place in the last 2 or less years. The impression provided in the document is that this has been in place for a much longer period of time.

This has been clarified

The section on pg 59 which discusses estimating true prevalence is based on random sampling in a population and would provide an overestimation if fish were being submitted for diagnostic purposes (i.e. fish submitted have clinical signs). Again sections such as this may be more appropriate as appendices.

Again, we feel this context is required for the reader unfamiliar with the statistical aspects of

how prevalence is estimated. Providing numerical examples, we hope, will more clearly illustrate the impacts of sample sizes and thus should not be left to an appendix.

There is a very good summary provided on screening versus diagnostic submissions between FFSBC and DFO (Tables 11,12).

The authors provide tables (13,14) summarizing infectious agents identified by the diagnostic laboratories as well as indicate which were isolated from sockeye salmon. I would be interested in seeing a table showing the breakdown by year.

See appendix 7 plus additional information on sources of sockeye samples for table 14

Release/escape of pathogens from enhancement facilities.

One of the methods not included is the possible release of pathogens when stream seeding with broodstock carcasses. It would be interesting to know the level at which this occurs and whether carcasses are screened or tested prior to seeding the streams.

As noted above, data were not provided by the Cohen Commission to us for such an analysis

Consequence assessment

Pathogens can transfer between salmon species and between salmonid and non salmonid species (not just between enhanced and wild sockeye)

Indeed this is true. Our statement of work was, however, restricted to assessing the possibility to assess the risks to Fraser River sockeye salmon and thus the consequence assessment was restricted to this population of interest. We do note in our literature review that diseases can affected ecologically important species such as prey species for sockeye salmon. In a number of our recommendations, we purposively use the term 'wild fish" rather than the more restricted Fraser River sockeye salmon in recognition that other fish are part of the disease risk setting (See sub-recommendation 3d for a specific recommendation to consider non-salmonids and sub-recommendation 4c as another example).

Fish Health Management Plans

The authors provide a good review of the FHMP, presenting problems with completeness, and consistency between facilities.

2. Evaluate the interpretation of the available data, and the validity of any derived conclusions. Overall, does the report represent the best scientific interpretation of the available data?

The apparent poor quality and quantity of the data available to the authors did not allow them to provide an interpretation of the level of risk. However, it certainly provided them with ample information to discuss the gaps.

There was IHNv data missing from the document that should be included.

As mentioned elsewhere, sparse IHNv data was present in the diagnostic records provided to us. DFO reported that routine screening is no longer done. This was mentioned in the report. We have added some information on IHN in the new section on spawning channels in the Hazard Assessment.

3. Are there additional quantitative or qualitative ways to evaluate the subject area not considered in this report? How could the analysis be improved?

I would be interested to see if there was annual variation in the diseases that were documented at the facilities (i.e. BKD, IHN). For example -were specific diseases/infections in 2007 higher or lower than in other years (annual variation). Is there enough data to suggest temporal variation?

See responses below to Dr. St. Hilaire's recommendation for type of analysis.

There should be inclusion of the spawning channel work done by G Traxler.

See comments above on new section on spawning channels and data provided for spawning channels. Note however, that diagnostic data on spawning grounds was sparse in the information provided to us and Traxler's publications mostly involved laboratory work on IHN, a report of an outbreak in Weaver Creek (included) and a report of an outbreak in non-enhanced sockeye salmon on Vancouver Island (not included)

I would also be interested in seeing what percentage of facilities incorporated preventative medicine practices (i.e. Broodstock and Pre-release screening (%/species), vaccination, monitoring).

We note in the report that we had neither the time nor the data to do site specific reviews of risks or risk prevention/mitigation practices but we do have some recommendations on the need for site specific work to assess the needs for local adaption of fish health management (ex.

Recommendation 7 and sub-recommendation 4a). Given that neither the FFSBC nor DFO have evaluated their fish health management plans, we suspect such data are unavailable.

I would like some description on the funding structure for enhancement facilities and how these have changed (or not) over time.

See comments in Dr. Kent's review. These data would inform future budget planning and help with development of our recommendation but they were not available for our review.

4. Are the recommendations provided in this report supportable? Do you have any further recommendations to add?

I agree with Recommendations 1 and 2

I agree that there should be one standard for fish health management between facilities and all facilities should be similar. This must also include PIP and CEDP facilities.

However I am not a fan of committees/working groups as these often take people away from their core objectives (which should be the fish). I would be more in favour of smaller grass roots project ideas that come from those familiar with the facilities rather than large scale research projects.

I personally am in favour of enhancement facilities however currently they are totally under supported and under resourced. Fisheries and Oceans Canada, and the public must reconcile the objectives and the role of these facilities and if they are deemed important then they must be provided the resources needed to operate successfully. The program should also assess what species will be enhanced.

Recommendations as I see them

1 - Appropriate support (financial, human resources, training) for programs and facilities to function properly. Enhancement programs have been under resourced for too many years. It may be useful to look at some of the models used in some states in the USA.

2- All enhancement facilities need to operate the same or equal standard that ensures that fish can be raised in a healthy environment. Currently standards vary highly between facilities. Standards should be comparable to the private commercial standards.

3 - Emphasis should be placed on preventative medicine (with standardization of screening

programs, vaccination programs, biosecurity). We cannot or should not want to produce 'sterile' fish however enhanced fish should not have significantly higher prevalence levels of infection or disease levels than the wild populations.

4 - Resources and lab capacity are needed to ensure that diagnostic/screening testing is done in a timely manner so that appropriate actions can be taken by the hatchery. Even at the current level of screening, the lab at PBS is the bottleneck with samples being analyzed weeks or months after being received.

5 - Better record keeping is needed for disease/screening results. This database should also include results from samples evaluated outside the standard diagnostic labs (i.e., G. Traxler IHNv work on sockeye salmon in the spawning channels).

This data must be analyzed, interpreted and published on a regular basis (i.e. every 5-10 years).

6 - Test results and recommendations should to be sent in electronic or paper form to be kept on record. It has not been uncommon to simply receive a telephone call.

7- Training must be provided to bring all the facilities to an acceptable standard of operation. Operations that cannot achieve the minimum standard should be closed.

8- All hatchery fish must be marked/tagged so that they can be readily identified from wild. This is essential if we are to be able to determine survival or evaluate the effects of certain changes in the program and compare disease/infection levels between wild and enhanced populations. Currently only 10% (or less) of the fish are marked or tagged with overall survival of some species at 1% or less - this makes any identification of fish almost impossible. In the past there was a head recovery program in place for commercial and sport fisherman to send heads in for identification - this is no longer in place,- this program could be revisited.

9 - Disease and infection prevalence vary over time making baseline data collected in the past not necessary appropriate to make management decisions. Regular health surveys in populations in the wild to determined prevalence of diseases and infection need to be conducted. To do this properly it is necessary to be able to identify wild from enhanced populations.

10- As a consequence of the similarity of migration of both wild and enhanced populations - comingling during their entire lifecycle is inevitable. However a reduction in comingling during the critical early marine stage could be achieved by modifying release time or holding enhanced population in net pens over summer period and release after most of the juvenile fish have left the area.

11 - Carcass in stream placement for enrichment should be discontinued or modified to prevent introduction of disease into the waterway (i.e., autoclave carcass before seeding or other appropriate method).

Upon cross-referencing Dr. Saksida's recommendations with our, we concluded that our recommendations include and accommodate Dr. Saksida's with a few exceptions. Her recommendation #8 is a methodological detail for future research. We are not advocating specific research methodologies until time is invested in identifying the critical and achievable questions to be answered. We can foresee the need to somehow tag fish to track their movements, allow for re-examination and monitor survival but cannot yet conclude that the costs of tagging all fish would generate research outcomes equal to the investment in people, resources and funds to tag all enhanced fish. In addition, there is a need for better data to determine if/how tagging affects survival and productivity to account for their effects in analysis of disease impacts. Recommendation 10 is a hypothesis rather than a specific recommendation. We were unable to make a recommendation regarding her point 11 (Carcass placement for enrichment) due to the lack of data to determine if it is or is not a risk. DFO does have guidelines in place to reduce disease risks but their effectiveness, to our knowledge, has not been assessed and found to be (in)sufficient.

5. What information, if any, should be collected in the future to improve our understanding of this subject area?

Need to know disease/infection levels in wild stocks and enhanced stocks.

Need to have better records of the disease/infection levels in enhanced stocks.

6. Please provide any specific comments for the authors.

Pg 18 para 4 - 1st sentence doesn't make sense - should it be "While a population can be healthy.... (omit therefore?)

Para 4 - missing word in first sentence?

Pg 51 1.1 - 2nd line should read health not heath

Corrected

Pg 52 3.2 - incomplete sentence

Revised

Pg 54 2nd line from bottom - a legible month ?? Not sure of meaning

Legible means – it could be read on the records. We have modified this sentence to indicate this meant records where the dates were recorded or recorded but not legible.

Pg 61 bottom paragraph - 2nd sentence - not sure of the meaning of the sentence (reword?)

Sentence modified

Table 13 - is the date correct in the caption?

Yes

Table 14 - date in caption not complete

Corrected

Pg 69 - 1st para, 2nd last sentence - Ichthyophonus not Ichthophtheirus?

Both organisms were found and the sentence is correct as stated

Pg 71 - 2nd paragraph - remove (error! Reference source not found.)

Corrected

Pg 76 - 2nd paragraph - Emerging instead of Emergency disease?

Emergency is correct (see http://www.dfo-mpo.gc.ca/aquaculture/regions/pac/application-demande-eng.htm)

Figure 5 and 6 - may be easier if use month names instead of numbers

Affected formatting too much to allow it to fit nicely on one page

Figure 6 - add ',' to numbers on y axis

Table 16 - may want to provide the number of hatcheries that treated

Some more information is provided. The numbers of hatcheries using treatments varied with

treatment and our data were not recorded in a manner that allowed for re-analysis for all treatments within the timeframe for the review.

REVIEWER 2

Reviewer Name: Michael Kent
Date: 8 July 2010

1. Identify the strengths and weaknesses of this report.

Strengths: This report is a thorough and detailed description of the fish health and diagnostic programs within enhancement facilities in British Columbia. It is well-written and contains very few typographical and grammatical errors. It provides a very useful summary of data on infectious diseases in SEP and other facilities and provides a comprehensive review of the status of fish health programs relating to these facilities in the Province.

Weakness. The main weakness was the lack of available data for the authors to conduct their assigned tasks in the Statement of Work. As the authors meticulously demonstrate, the records, publications, etc. do not provide the data to conduct a meaningful assessment of the potential effects of diseases. The authors correctly justify this because of the lack of data to conduct these analyses.

2. Evaluate the interpretation of the available data, and the validity of any derived conclusions. Overall, does the report represent the best scientific interpretation of the available data?

The report does provide the best analytical interpretation of the work. See comment in item 3.

3. Are there additional quantitative or qualitative ways to evaluate the subject area not considered in this report? How could the analysis be improved?

However, I believe it would be useful to the Commission to provide the authors opinions on what would be the pathogens of most concern for transmission from captive fish to wild sockeye.

For example, it appears that *Flavobacterium* spp., particularly *F. pyschrophlium,* would top the list as 1) it is pathogenic, 2) it occurs in hatcheries but apparently not wild fish, 3) it is apparently transmissible directly from fish to fish.

I agree that there is no evidence at present that this pathogen posses a significant risk, but in my opinion it would be worthy of investigation. Perhaps Dr. Stephen and co-authors disagree, but still it would be useful to provide their ranking based on the considerable amount of knowledge they have on the subject.

In other words, Dr. Stephen is a recognized expert in fish epidemiology, with particular experience with salmonids in British Columbia. Therefore, it would be appropriate for him and his colleagues to provide their qualitative opinions on which pathogens they conclude (yes, based on very limited data) might be of most concern relating to transfer from hatcheries, etc. to wild sockeye salmon.

We appreciate Dr. Kent`s confidence in our opinions, but in his review and in his report to the Cohen Commission, Dr. Kent noted the lack of evidence to make conclusions on risk, particularly at a population level. In our definition of risk, there must be knowledge of not only of the potential magnitude of harm of a pathogen, but also on the probability that the population at risk (Fraser River sockeye salmon) is exposed to the hazard. We document in Risk Assessment section (Exposure Assessment) that exposure assessment can only determine that exposure is plausible but cannot allow us to specify the range of probabilities for exposure to specific agents. Throughout the report we note that there are no data with which to determine (1) that salmonid enhancement increase environmental exposures of Fraser River sockeye salmon to levels beyond background; (2) that all relevant hazards are identified; (3) how sampling and diagnostic biases affect our ability to accurately enumerate all potential infectious hazards and (4) the nature of impact of pathogens on Fraser River sockeye salmon population. Our opinion in this matter is that it is better to focus our recommendations on improvements to the overall knowledge base, and on general risk management and oversight practices that may reduce the risk from multiple pathogens rather than to speculate on which specific agent will be of greatest risk. Our concern with doing the latter is that responses to those opinions may drive follow-up actions towards a single pathogen rather than systematic changes to reduce risks from known and unknown infections.

We do discuss in some depth in the section "Release/escape of pathogens from enhancement facilities" aspects of Myxobacteria (including Flavobacterium spp) relevant to risk and outline critical unknowns that would not allow for determination of the level of risk to wild fish. In his review, Dr. Kent highlights these as critical unknowns that should be emphasieed.

4. Are the recommendations provided in this report supportable? Do you have any further recommendations to add?

The recommendations provided are correct and supportable. However, I would also recommend development of a list of pathogens of most concern based on the limited information. I realize that this is rather subjective, but it still would be a worthy endeavour (see comment above). This would allow for prioritization of specific research projects to fill important information gaps.

See section 3 above, particularly our view that the research recommendations should be systems-focussed and not pathogen-focussed.

5. What information, if any, should be collected in the future to improve our understanding of this subject area?

Dr. Stephen and colleagues articulate this in depth in their report. In brief, some examples of key data that are needed include data on prevalence of pathogens in fish that are released from facilities and background levels in wild sockeye are needed. Also, information on infectious dose and viability of specific pathogens is needed.

6. Please provide any specific comments for the authors.

Did the authors see in patterns in the disease data from "facilities" that would suggest a possible correlation with the exceptionally poor returns in 2009. I think the answer will be "no", but it would be useful to provide their opinions or conclusions on this subject.

We refer the reader to our replies to Dr. St.Hilaire below for a more detailed answer regarding the inappropriateness of analysis of temporal and spatial correlations between patterns of wild salmon disease and enhanced salmon diseases because of critical gaps in available data

We refer the reader to our replies to Dr. Saksida's review for an explanation of data features by time and place that are presented in a new Appendix 7.

References need to be provided for several general statements as indicated in the marked text.

Marked text was not provided

I recommend inserting a brief discussion on the fact that "facility" fish are deliberately released into the wild and certainly co-mingle with wild fish, and thus potentially posse a higher risk of transmission to infectious agents to the latter. This concept is woven into the document, but I recommend this point be better emphasized.

We have the following statements in the report: ` Salmonid enhancement facilities typically aim to release their fish timed with the movement of outmigrating wild fish (MacKinley et al., 2004). This could allow for commingling of wild and enhanced fish and thus present possible opportunities for transmission of infections between these groups (Rhodes et al., 2006)." We feel these are clear and definitive statements. These references can be found at the start of the section: "How might salmonid enhancement facilities affect Fraser River sockeye salmon disease status? Establishing direct transmission of pathogens as a route of exposure". This concept is re-enforced later in the risk assessment and thus we feel does not need further repetition.

Somewhere in the text it would useful to provide a succinct summary of the functions of the diagnostic and health support provided to facilities. Number and location of diagnostic labs, number of staff, financial support, overview of types of diagnostic tests. For example, PCR and tissue culture for major viruses, histolopathology?, etc. This would help the readers and Commission better understand the capabilities of support to carry out the proposed recommendations.

An overview of the diagnostic tests is provided on in the Section ` Data source for hazard assessment`. We have added in that there is only 1 diagnostic lab at each of PBS and the FFSBC. Additional information is provided in the section on data validity (Validity of the diagnostic tests). We did not request information on human and financial resources at the outset of the project and it was not possible to obtain these data within the timeframe for response to reviewers (1 week) as all requests for information had to go through legal channels rather than through direct request by our team to DFO or FFSBC.

Other Specific Comment

Table 2 (page 13). It would be helpful to provide data on the number of these listed facilities that have or have had fish health and diagnostic support

Tables 9, 11, 12 provide information on facilities from which we received diagnostic data. The second paragraph in the Risk Management: Fish Health Management Plans section, list

facilities from which we received plans. Our new Appendix 7 includes the number of facilities providing diagnostic submissions per year

Page 17. 2nd paragraph. Remove ";" after Rhodes e al.

Our referencing style has a `;` between multiple authors throughout the report

Literature Review. It would be useful to provide a review on transmission of pathogens – e.g., viability in water, infectious dose, etc. for the major pathogens of concern. I realize these data are lacking for many pathogens, but I believe such studies have been done with the major salmonid pathogens, such as IHN and *Aeromonas salmonicida*.

There are two ways to approach this comment. First, is to consider the possibility of answering the question, "would the conditions of the Fraser River sockeye salmon environment(s) allow for pathogen viability and exposure to an infectious dose so as to result in an effective transmission?" We did note in the exposure assessment section(Exposure of Fraser River sockeye salmon in enhancement facilities), that we can conclude that Fraser River sockeye salmon within an enhancement facility have acquired infections. To examine the possibility of other forms of transmissions we would need (1) data on concentrations of pathogens in fish bearing waters associated with enhancement activities; (2) data on viability and infectious doses and how those vary with differing environmental conditions and (3) data on environmental conditions associated with enhancement activities. We found no published literature on the last point and as Dr. Kent points out, limited information for the second point and less for the first. We did not request environmental monitoring data from PBS or FFSBC but it is our understanding that such data are not routinely collected. Therefore, such an analysis was not possible.

The second approach was to try to convey to the reader that pathogen transmission requirements vary with species, pathogen and environmental setting. Table 15 (Examples of transmission pathways for selected pathogens found in salmonids) is an illustration of the importance of waterborne transmission as well as to demonstrate that key salmonid pathogens can be transmitted in more than one way. We also pointed to general reference and other research that supports the conclusion that effective transmission of a pathogen in aquatic environments would differ with different environmental settings due to the effects of environmental conditions. Our section on susceptibility in the literature review also describes how environmental conditions and host susceptibility can affect transmission. Because the available literature deals largely with settings other than the ones specific to this report, a further literature review would only provide some data not specific to pathogen viability and transmission throughout the Fraser River sockeye salmon ecosystem. For this reason, we did not explore these concepts further than

the aforementioned introductions to the concept. We did, however, note in our exposure assessment that lack of information on the distribution of pathogens in the environment prevents exposure assessment. Additional information on concentrations and viability would be essential adjuncts to distribution data. Time constraints prevented a more detailed review and summary of research on pathogen viability and transmission under natural conditions.

Page 23. The authors cite Stephen and Thorburn (2004) and state that the as of 2001 no articles in fish health journals dealt with the effects of disease [in fish] on populations or ecocystems. This statement is somewhat misleading. This lack of published research might be true for the fish health journals they investigated, but there are several descriptions of pathogens (parasites) affecting wild fish in the parasitology and ecology literature (e.g., Jacobson et al. 2008). This example is after 2001, but there are several similar studies going back several decades – e.g., studies on the impact of *Ichthyophonus* in herring. Dobson and May (1987) and Sinderman (1987) provided 2 reviews of this in an issue of Intl. J. Parasitol.

Dobson, A. P., May, R.M., 1987. The effects of parasites on fish populations-theoretical aspects. Int. J.Parasitol. 17, 363-370.

Jacobson, K.C., Teel, D., Van Doornik, D.M., Castillas, E., 2008. Parasite-associated mortality of juvenile Pacific salmon caused by the trematode *Nanophyetus salmincola* during early marine residence. Mar. Ecol. Prog. Ser., 235-244.

Sindermann, C.J., 1987. Effect of parasites on fish populations: practical considerations. Int. J. Parasitol. 17, 371-382.

I do agree, however, that there are very few such studies with salmonids. But in addition to Jacobson et al. (2008), there are a few others. For example, Kocan and colleagues have been studying the impacts of *Ichthyophonus* on Yukon Chinook on at the population level. Also, there are a few papers by Norwegian scientists on impacts of worms in char at the population level. See references below

Kocan R, Hershberger P, Winton J (2004) Ichthyophoniasis: an emerging disease of Chinook salmon. *Oncorhynchus tshawytscha* in the Yukon River. J Aquat Anim Health 16: 58–72

Halvorsen, O. and Andersen, K. 1984. The ecological interaction between artic char Salvelinus alpines (L.), and the plerocercoid state of *Diphylobothrium ditremum*. J. Fish Biol. 25: 305-316.

The Stephen and Thorburn reference was made specifically for the fish health literature. We do agree with Dr. Kent that there are a variety of papers that look at impacts of parasites and pathogens in the parasitology and ecology literature and that this literature is sparse for

salmonids. We did include some examples of results of parasitology or ecology studies of fish parasites and pathogens, although they are not placed right next to the Stephen and Thorburn statement. We referred to Kent's (2011) general comment on these types of data followed by a reference from Arkoosk stating these data are minimal. (see section Potential mechanisms for impact on Fraser River sockeye salmon by diseases associated with salmonid enhancement*). We also provided examples of these types of data in the section "Experimental evidence other than death and disease" and in the section on "Cross sectional studies and surveys."We do make reference to the Ichythophonus case as example in our section "Potential mechanisms for impact on Fraser River sockeye salmon by diseases associated with salmonid enhancement." We have, however, modified this section to recognize the contributions of these areas of research to our understanding of the possible impacts of fish diseases and parasites: Sindermann 1987 and Dobson and May (12987) have now been cited.*

Kocan et al (2004) is a description of clinical signs, descriptive epidemiological features of the outbreak and changes in prevalence and not an assessment of population impacts. Jacobson et al (2008) was not accessible via the University of Calgary on-line library but it's abstract was available and suggested that it too was a study of variation in prevalence and not population regulation. Halversen and Andersen (1984) concluded that their "results are not believed to represent the true pattern of uptake of copepods by the fish population" and that they could only conclude that "fish with a higher number of plerocercoids could be either longer (heavier) o r shorter (lighter), but in no single group was there a statistically significant trend in either direction with increasing number of plerocercoids." Finally, these authors hypothesized that "plerocercoids of D . ditremum alone <u>may</u> cause mortality in the fish intermediate host population". We conclude that this paper is a good example of studies that help to describe the ecology of the host-parasite relationship but did little to document impacts on the host population.

Page 24. Last sentence. "Long term studies of salmon diseases were rare" Please provide the citations for these "rare" studies.

Examples and references are now provided

Page 28. Perhaps here would be the appropriate spot to insert a table of the documented freshwater pathogens and infectious diseases found in sockeye in the wild, and contrast this with those found in "facilities". A partial list is found in Table 13 (page 66) for pathogens of fish from facilities. I realize an exhaustive list of the parasites found in wild sockeye would be too much, but a representative list would be very useful to the Commission and those reading the document. Why ? Because this would clearly set the stage for comparing wild vs captive

diseases

We understood that the assembly of a list of wild sockeye salmon infectious diseases was under the scope of work for Technical report 1 which stated; "The veterinary scientist will take a broad view of sockeye diseases and parasites that span the life cycle from egg to adult. The scientist will evaluate the full spectrum of diseases that occur at all life history stages."

We originally highlighted in Table 14 which pathogens are from sockeye salmon and have now indicated which found in sockeye salmon classified as "wild or semi-wild" in the PBS diagnostic data provided to us. Because of the lack of clarity of what constitutes wild, semi-wild and semi-cultured in the diagnostic records, a comparison of wild vs cultured could be unclear.

Page 29. continuation of Table 4. Effects of genetics alterations in hatchery fish leading to increased susceptibility to disease is mentioned. I provided a report to the Commission and Dr. Stephen by Kaufman et al. (2010) documenting studies along these lines that are ongoing in Oregon. This work should be cited in the appropriate location in the report.

This reference has been reviewed and results incorporated into the text.

Kaufman, J.R. (2010). Studies on Prevention and control of Infectious Diseases of Salmonids. Report from Ore. Dept. Fish and Wildlife. PROJECT NUMBER: F-104-R-30

Page 31. Line 10. Provide the conclusions from the Hanninen et al. study.

Done

Page 39. Line 13. I believe this is Hedrick, not Hendrick. Correct in literature cited as well.

Done

Page 44. Another point to address regarding problems with surveys – often titles state "Parasites of XXXX from XXX region", when actually they do not include protozoans parasites in their investiagtion. This is thus misleading on the distribution of these microparasites. For example, see

J. R. Arthur, E. Albert. A survey of the parasites of Greenland halibut (*Reinhardtius hippoglossoides*) caught off Atlantic Canada, with notes on their zoogeography in this fish*Canadian Journal of Zoology*, 1994, 72:(4) 765-778,

We have added in this concern more generally by saying that surveys are often focussed on one

pathogen or group of pathogens at the exclusion of others.

Page 45. 3rd from last line. Please provide citations for these "past reviews".

These reviews were mentioned elsewhere in the text but are citations are now repeated at this point in the text as well.

Page 46. Line 10, 11. Please provide the citations or more information regarding "historic cases"

Citations provided

Page 46, 47 Table 6. Tables are should be "stand alone" documents. Hence, the authors must indicate what X and the check stand for. Better yet, how about stating "not fulfilled" (I assume the X) and "Fulfilled" (for the check) in the table.

Recommended change has been made

Page. 52. Bullet 3.2. This is an incomplete statement.

Modified

Page 74. Please provide date and location for the six bullet items.

Done

Page 75. The authors make very good points in the statement "There remain three critical unknowns….". This is rather lost in this big paragraph. In my opinion, these statements are very important and should be presented as stand alone bullets.

Done as a separate paragraph

Page 79. 2nd to last line. *Myxidium* is not spelled correctly.

Corrected

Page 83. Add a subtitle for the last paragraph on release of water or wastes as this is a well-defined separate section.

Done

Page 84. "The survival of fish can very tremendously…" provide references and examples to back up this statement. Also, Table 15. What does "X" mean.

Footnote added to Table 15. References added

Page 90. Here one paragraph takes almost the entire page. Thus this section should be divided into 2 or 3 paragraphs.

Unchanged as the content of this paragraph is all related

Page 92. "Most documents…" Please give us an idea what "most" means. That is, how many documents fall into this category.

This has been clarified

Page 101. Recommendations. I agree with most of these recommendations. Please provide statements on feasibilities for each. For example, better record keeping and compliance is certainly achievable with limited extra funds. In contrast, amount of effort and funds to support the needed research to fill the knowledge gaps on transmission of pathogens from facilities, with subsequent significant cause of disease, in wild sockeye salmon would likely be very great.

Feasibility is an important consideration. We tried to only recommend steps that we felt were feasible but this is only opinion based and did not account for current federal or provincial budgets, other recommendations arising from the Cohen Commission or an scan of opportunities for synergies with ongoing work. A program feasibility assessment is beyond our current capacity within the time and information available for this review.

REVIEWER 3

Report Title: **Assessment of the potential effects of diseases present in salmonid enhancement facilities on Fraser River sockeye salmon**

Reviewer Name: **Sophie St-Hilaire**
Date: **July 4, 2011**

1. Identify the strengths and weaknesses of this report.

Strengths:

The review of the health records of the enhancement facilities by Dr. Stevens and his team identified several possible routes of pathogen transfer from enhancement facilities to wild fish populations that should be addressed. They also identified several disease control measures that need to be re-assessed. In most cases, DFO makes the right decision when they release enhancement fish but, as the research team pointed out, a more systematic evaluation would be beneficial to ensure the goals of the enhancement program are being met. The pathogen transmission and disease control issues identified in this report are not specific to Fraser River sockeye salmon and the recommendations are of potential benefit to all wild salmonids.

Weaknesses:

The primary weakness of this report is that the authors did not assess whether the current decline in the Fraser River sockeye salmon stocks is partially due to pathogens originating from enhancement facilities. The primary reason given for this deficiency was the lack of high quality data. If the researchers had looked at the wild fish population recruitment and escapement data and the wild fish disease data, in conjunction with the enhancement facility disease data over time, they could have made more conclusive statements on the potential role (or not) that enhancement facilities play in the decline of wild sockeye salmon. It is unlikely they would have been able to make definitive conclusions, but if they had seen temporal and spatial trends in disease outbreaks that corresponded with population declines, such observations would have been useful for formulating strong hypotheses.

The task of associating pathogens with the decline of Fraser River sockeye was not in our scope of work, but rather was in the scope of work for technical report 1: specifically that report was tasked with "evaluating the documented and potential effects of parasites and diseases on Fraser River sockeye salmon and their role in the 2009 run failure". We were tasked with assessing the capacity of conducting a risk assessment and only if deemed possible, to undertake an assessment of the risk of infectious diseases from enhancement facilities. The major conclusion of our report was that a reliable risk assessment cannot be undertaken.

Dr. St. Hilaire found the lack of spatial and temporal analysis to be the primary weakness of our report. Dr. St. Hilaire recognized that it is "unlikely [this analysis] would have been able to make definitive conclusions" but instead it might generate a "strong hypothesis." Our statement of work was focussed on assessing the ability to assess if a risk assessment could be done (we concluded it could not) and not on hypothesis development.

We suggest that Dr. St. Hilaire has failed to accommodate for the many problems in quantity and quality of data available to us when making her recommendation. We note the following quote from the Journal of Epidemiology and Community Health. 2007 February; 61(2): 98–102., "The

danger of ignoring data-quality issues is that, because of missing cases or inaccurate baseline population data, one might arrive at a misleading (invalid) high or low estimated risk".
Quataert et al (1999) provide detailed critique of spatial and temporal cluster analysis for disease that informed our consideration of whether or not the data would allow for reliable modelling or analysis. Their conditions required for reliable spatial and temporal trends analysis are noted below, with comments on the ability of the data provided to us to fulfill the criterion:

1. *Sufficient geographic and temporal variation in exposure exists*
    a. *Exposure characteristics are unknown for this case*
2. *Information about the mobility and trends in the population at risk is provided*
    a. *We lacked data on the movement of Fraser River sockeye salmon with respect to their exposure opportunities near hatcheries or while comingling with enhanced fish*
3. *Ability to distinguish exposure from the source of concern from alternative sources*
    a. *The pathogens involved in this case are endemic and could be from 'wild' or enhanced sources*
    b. *Directionality of pathogen movement cannot be established with historical data*
    c. *Molecular epidemiological data are lacking to determine if the pathogens in wild and enhanced salmon in BC are from the same source.*
4. *Classification of cases need to be reliable and consistent*
    a. *No case definitions were available and we detected variation in how cases were recorded in the records*
    b. *The lack of systematic surveillance suggests some cases may be under-reported while other cases may be over-reported.*
    c. *DFO itself comments in its carcass placement guidelines that data on wild fish diseases is lacking.*
5. *Understanding of normal spatial and temporal clustering patterns for diseases*
    a. *Lack of background information would prevent us from distinguishing normal clusters from abnormal clusters*
    b. *Spatial autocorrelation could occur if wild and enhanced fish are exposed to a shared source unrelated to the enhancement facilities or fish*
6. *Information on exposure status of individuals is required for causal conclusions*
    a. *We could not determine how a salmon in a river or ocean is exposed to an endemic pathogens which has more than 1 possible source.*
7. *Sampling programs should not introduce bias in case distribution*
    a. *Within the data provided to us, there was unequally numbers of submissions from hatcheries and spawning channels such that the intensity of oversight was*

unequal across facilities. The likelihood that a pathogen would be detected would, therefore, differ across facilities due to differences in sampling, thus creating a sampling bias.

Typically, the selection of analytical method and calculation of the power of the analysis for spatial and temporal analysis requires knowledge of ; (1) the sampling design of the surveys; (2) the spatial distribution and movement of the underlying populations at risk; (3) the scales of geographic space used to locate animals; (4) the variability expected in the data; (5) how many years the data was collected; (6) the intervals between observations; (7) the required significance level of the tests; (8) the number of counts per areas surveyed and (9) the number of surveys. Most of these data were not available to use. The diagnostic data provided to us were not from systematic surveys. Some publications of periodic surveys were found. We are confident that there would be variations between methods, power and frequency of surveys within and between facilities and between enhanced and wild fish, making collapse of these various data sets into one analytical framework a threat to the spatial-temporal cluster analyses methods with which we are familiar. For all of these reasons, plus the delays in receiving the first set of data and thus no time to request additional data, the recommended analyses were not undertaken. We recommend serious consideration of each of the threats to the validity of such an analysis before they are undertaken or used to inform decisions.

Quataert PKM, Armstrong B, et al. 1996. Methodological problems and the role of statistics in cluster response studies: A framework. European Journal of Epidemiology 15: 821-831

A more comprehensive summary of diseases found in wild fish would have also been beneficial. The literature review did not identify all disease outbreaks in the Fraser River and the researchers did not request diagnostic information from DFO. The DFO diagnostic laboratory investigates several wild fish "die offs" a year (at least they used to) and, had these been requested, the researchers could have compared the list of pathogens in enhancement facilities to those in wild fish. It is true that the wild fish disease investigations are limited, but if the pathogens causing mass mortality in wild sockeye salmon are never found in enhancement facilities then it is unlikely that these facilities are the source. This comparison, along with the temporal and spatial trend in disease outbreaks mentioned below in point #2, could have provided information to support strong hypotheses on the role enhancement facilities have played (if any) in the decline of sockeye salmon.

We did request diagnostic data from DFO. In our section on representativeness of the data we did state; "There were reports of wild fish disease investigations each year in the PBS diagnostic data base. Because wild fish diseases were outside of our scope of work and were a small proportion of the diagnostic submissions and because the timeline for data review prevent a full

assessment of hatchery results as well as wild fish results, wild fish records were not included in the hazard assessment. The opportunistic nature of wild fish investigations (often initiated by field staff or the public), the comparatively small number of cases of wild fish disease investigations and the limited spectrum of pathogens sought in broodstock screening restricts the capacity of these data sets to reflect the patterns of diseases in wild fishes." We refer again to our terms of reference that focussed on an assessment of the capacity to undertake a risk assessment and compare it to the scope of work for technical report #1 as described elsewhere.

Finally, there was very little information specific to spawning channels, which is the primary method of enhancing sockeye salmon and, therefore, these types of facilities have the most potential for affecting wild sockeye populations. This informational gap was most likely due to the fact that these facilities did not provide data.

The relevant published literature on spawning channel diseases is presented throughout the report, specifically to work on IHN and "Ich." We re-searched Google Scholar and PubMed in response to this concern, searching with the key worlds "sockeye spawning channel disease British Columbia" as well as using the surnames of two prominent BC fish virus researchers (Traxler and Garver) and found no additional information. We re-searched the diagnostic data and found submissions from Weaver Creek (5), 3 from Nadina River, and 7 from Inch Creek.

In recognition of the importance of spawning channels, we created a new section in the Hazard Assessment titled; Spawning channels". This does not affect the conclusions of the hazard assessment but does provide some focussed summaries of past work including some cases on previous surveys done in spawning channels. Most work in spawning channels has been limited to 3 pathogens. Garver's summary report is cited. It indicated that there has been no noticeable trend in Ich or IHN in 2 spawning channels (Weaver Creek and Nadia River) and thus he concludes these are unlikely to have impacts on Fraser River sockeye. Parvicapsula remains a possible concern. We discuss this disease elsewhere in the report and note that it is not thought to be associated with enhancement operations. Additional survey data from spawning channels might reveal additional pathogens but we think the likelihood of this is low because (1) interviews with DFO staff did not suggest there were unusual problems or problems unique to spawning channels that could not be found elsewhere; (2) the literature and diagnostic data review did not yield unexpected pathogens and (3) most of the survey work was limited to studies of specific pathogens.

2. Evaluate the interpretation of the available data, and the validity of any derived conclusions. Overall, does the report represent the best scientific interpretation of the

available data?

The researchers claimed they did not have sufficient data on disease occurrence and prevalence in enhancement facilities to asses the transfer or risk to wild fish, but they could have used available data to look for evidence (or not) of movement of pathogens between enhancement facilities and, possibly, to wild fish populations.

We do not know what data Dr St. Hilaire is aware of that shows direction of movement of pathogens between enhanced and wild salmonids. Our interviews with PBS and FFSBC staff and review of the literature failed to find research outcomes or methods that have been used to document directionality of pathogen traffic between wild and enhanced sockeye salmon. This is discussed in our literature review.

The researchers claim throughout the document there is no disease information available on Fraser River sockeye salmon, but this is not true. While there may not be many diagnostic work-ups on early fresh water life stages, over the last 10 years there have been numerous investigations on returning adult sockeye in the Fraser. The diagnostic work-ups may not be written up each year, but they are still available through the DFO diagnostic laboratory.

We cannot comment on data that were not provided to us or published in the literature.

Also, the researchers claim that subclinical effects are not measured in wild fish and so cannot be assessed. This is true, but I don't think the problem with the Fraser River sockeye salmon is a subclinical problem with subtle effects.

Dr. St. Hilaire comment stands in contrast to the bulk of the literature we found on the population regulating effects of disease in wild species, which is presented in our literature review. Indeed the 2 largest disease outbreaks in spawning channels (IHN and Ich) failed to have predictable effects or long term or medium term impacts on returns in subsequent years

The lack of monitoring of subclinical disease at facilities is not relevant for assessing the spread of pathogens at this point in time, when we still do not have a good understanding of how much transfer occurs during clinical disease outbreaks. I would argue that if you cannot see evidence of pathogen transmission in the face of a disease outbreak then you are very unlikely to see it when you only have subclinical disease.

Given the lack of data we received on monitoring of wild fish adjacent to enhancement operations, the lack of capacity to follow infected wild fish after they transit by enhancement facilities or comingle with wild fish (these challenges are detailed in the report), we cannot

conclude that there has or has not been pathogen transmission.

The DFO diagnostic lab maintains effective surveillance of disease outbreaks at enhancement facilities and, to some extent, in wild fish, so the researchers should have been able to analyse these data for trends.

The effectiveness of DFO surveillance has not been documented. DFO has had some active surveillance for specific pathogens that take the form of some broodstock screening and spawning channel screening. There were no reports of systematic and standardized wild fish surveillance, although period investigations of die-offs has been done or surveys for specific pathogens. These surveys have not taken into account the trends in the populations providing the data. Analyzing disease trends in the absence of data on population trends is a well known and serious error in surveillance analysis.

It also appears no effort was made to look at the effects of outbreaks at enhancement facilities on the escapement numbers of the affected stocks. There are several examples of pathogens in enhancement facilities (spawning channels) where a large proportion of the enhanced and/or wild stock was negatively affected (i.e. *Ichthyophthirius multifiliis*, IHNV).

Both of these outbreaks are described in our literature review. Our new section on spawning ground hazards supplements the current information in the report. The remaining data we were provided with was not recorded in a way that allowed us to determine if cases submitted were part of an outbreak or were part of investigations of endemic background infections. The data also did not allow us to estimate prevalence of infections but rather were suited only to determine presence or absence of pathogens at the time samples were submitted and thus are unsuited to looking for correlations with escapement. Finally, we did not have data on confounding or interacting variable and believe a univariate analysis of presence of an infection in diagnostic submissions with escapement would be overly simplistic and potentially erroneous approach. It would result in a reasonable likelihood of encountering the "ecological fallacy": an epidemiological concern when general data are interpreted too particularly or minutely (characteristics of the group are assumed to be characteristics of the individual)

The researchers even identify a few of these outbreaks, but do not make full use of the information. They could have requested population survival information on the affected stocks (smolt estimates and adult escapements), as well as diagnostic information on any wild fish in the area at the time of the problem or facilities downstream from the affected enhancement facilities.

Indeed we agree that these additional data would have been welcome, but delays in receiving the

data and the months it took to re-assemble the hundred of pdf files into an analyzable database consumed all of the time for this project and did not allow for the request of additional data. It was our understanding from interviews with staff that wild fish surveillance was not systematic and not common practice when diseases occurred in enhancement facilities. We were therefore under the impression that such data were rare or absent in most cases. This impression was supported by comments about the lack of wild fish disease data in the DFO Carcass Placement guidelines

Further, the researchers could have assessed the data they had from the BKD and IHNV screening programs to determine if these programs are affecting the prevalence of these pathogens in the wild fish populations over time. Here are two programs that are trying to reduce the potential effect that enhancement has on disease occurrence in wild fish, but they were not evaluated. Are the programs working? Are facilities that have never had these pathogens but are near affected sites gradually starting to see problems with these diseases? Claiming that there are false positive results is no reason not to analyse the data for trends. False positives will occur with all tests and are simply a limitation when interpreting the results.

When we searched the key words 'false positive' in our report, we only found references to test performance as it relates to detection of disease and estimations of prevalence and not to assessing BKD program effectiveness. We do describe the one study we found that examined the effects of a BKD management program like that used by DFO (see comments in Dr Saksida' s review and reference Munson et al, 2010). We were not provided data with site specific BKD prevalence, degree of site specific BKD control, screening results or other confounding factors required to assess the effectiveness of a program and thus relied on Munson et al's work to give some indication that the DFO BKD management plan is likely to reduce BKD prevalence on site. The reliance on a case series rather than a controlled study creates some problems in drawing cause-effect conclusions about the effects of an intervention. The lack of wild fish monitoring and studies or surveillance capable of linking pathogens of enhancement origin versus from elsewhere also makes it hard, if not impossible, to attribute any reduction in risk from trend analysis at a hatchery We do provide one paper in our literature review that suggests the probability of BKD infection is the same for wild and hatchery Chinook salmon, suggesting a non-hatchery source is possible (Rhodes et al, 2006).

Although it is clear that there are limitations to the data available from the diagnostic laboratories, the researchers did not evaluate the data as thoroughly as they could have for spatial and temporal disease patterns, which may have provided evidence for or against pathogen spread from hatcheries and spawning channels. Perhaps it was outside the scope of this project, but this is one of the analyses required to assess the role of enhancement facilities on the decline of

Fraser River sockeye salmon and, as such, it should be included as a research recommendation.

As noted elsewhere, an analysis of the role of infectious diseases in the decline of Fraser River sockeye salmon was within the scope of work for technical report 1 while our assigned objective was to review disease data and reports from salmon enhancement facilities operated by Canada and BC and evaluate the potential for a qualitative and/or quantitative assessment of the potential effect of diseases present in enhancement facilities on Fraser River sockeye salmon.

Despite the lack of data analysis the researchers identified some very important potential routes of pathogen transfer (i.e. release of infected fish and lack of proper effluent water treatment) and other disease management issues for enhancement facilities that, even without definitive "evidence," are biologically plausible routes of pathogen transfer and should be addressed, especially given the objectives of enhancement facilities. In other words, despite not demonstrating evidence of pathogen transfer, the conclusions and recommendations made in the report are consistent with the principals of infectious disease epidemiology and are therefore valid. The route of transmission and other disease management issues identified in the report expose a potential risk of enhancement facilities to all wild salmonids.

3. Are there additional quantitative or qualitative ways to evaluate the subject area not considered in this report? How could the analysis be improved?

As mentioned above in point #2 there were no analyses done. There were good summary statistics and the critical points for pathogen transmission were clearly identified, but it would have been useful to see analytical discussion of the diagnostics on wild fish. Descriptive information on wild sockeye salmon disease outbreaks (based on the DFO laboratory diagnostics and supplemented with the peer-reviewed journal articles) would have been useful for comparison with enhancement facilities. Some causes of mortality could be similar to what is diagnosed in enhancement facilities, but other types of pathogens may not have been identified in enhancement facilities (i.e. *Parvicapsula minibicornis*, *Dermocystidium*, *Cryptobia* sp, etc...) so cultured fish are unlikely to be the source of these. It is still possible enhancement facilities indirectly impact the occurrence of these pathogens, but targeted studies are required to answer those specific questions.

Another analysis that would improve the report is evaluating the temporal and spatial patterns to the disease outbreaks in enhancement facilities and wild fish stocks. This would provide evidence for or against pathogen transmission. In other words, when there were hatcheries or

spawning channels that had disease outbreaks, did wild fish or other facilities in the surrounding area also experience disease events? If so, based on the time of diagnosis, which disease outbreak appears first? A temporal and spatial cluster analysis (using GIS) would be an effective tool with which to assess the spread of pathogens within a geographic region. It is possible that the results of this type of analysis are inconclusive, but until it is performed we will not know.

Lastly, information on which life stage(s) of the sockeye salmon is first affected by the decline would help the investigation into the factors driving these declines. For example, is the problem with sockeye salmon first observed during the early fresh water life stages or during the salt water life stages? If the decline is acute and severe, as it appears to be, it should be detectable using the stock assessment data collected by DFO (I am certain they already evaluate these data). Pathogens that originate in the spawning channels and are acute in nature (i.e. viral diseases) would be expected to have an effect before the fish migrate to salt water, given the extended period of time that sockeye spend in fresh water. Pathogens that cause chronic problems may manifest in salt water, even if they originate in the spawning channel. The more definitively we can identify the time when the problem begins the easier it will be to identify the reason(s) for the decline. Understanding the timing of the problem requires expanding the data search to include production and stock assessment information. It may also require increasing surveillance of some key fish populations.

Each of these questions is relevant to the issue of the role of disease in Fraser River sockeye salmon declines as well as to the general causes of the decline. Each, as we outlined in our report and in our explanation above, are beyond the scope of our work, unable to be answered within the assumptions and needs for spatial-temporal cluster analysis or beyond the available data to answer. Significant work in accounting for the biases of the data and the collection of additional data on confounding and modifying factors would be required before more than simple correlations could be attempted. Our recommendations # 10, 10a and 10b on future research are not prescriptive.

4. Are the recommendations provided in this report supportable? Do you have any further recommendations to add?

Although the researchers did not assess the risk of pathogen transfer from enhancement facilities to wild fish, they did identify some management practices that are problematic for a number of wild fish species. The four critical issues identified, that need to be addressed immediately, are the release of diseased fish, the lack of effluent water treatment, the improper disposal of dead fish at some facilities, and the unauthorized use of chemical bath treatments at some facilities

(i.e. particularly spawning channels). The researchers also identified some deficiencies in the disease surveillance program, although in my experience the diagnostic laboratory at DFO has a very good database for the types of pathogens that have caused mortality events at hatcheries and spawning channels. Certainly, there could be improvements to any system, but the deficiencies with detection of pathogens causing high mortality in enhancement facilities seem less significant than controlling the fate of diseased animals and infected water at the enhancement facilities.

We were not provided the DFO disease database. We were provided with specific files from the Cohen Commission that represented DFO's response to our request for data as outlined in our methodology section. We did not request data from the science branch on all work on sockeye pathogens, assuming this was within the scope of technical report 1. Time limits did not allow us to ask for supplementary data.

Dr. St. Hilaire's last sentence in this section is important and is in accordance with our recommendations. Rather than focussing on a specific pathogen(s), we are advocating for changes to the fish health management system that might provide general improvements in oversight, management and risk detection and prevention. This, we hope, will provide for a broader level of protection rather than steps that are targeting single pathogens.

General recommendations:

Recommendations 1 through 7 in the report are all consistent with good management practices. Many are in place already at some facilities, and should be applied to all remaining facilities. However, I know it will be difficult to achieve the recommended high standards at the smaller community enhancement projects.

Research recommendations:

The research recommendations were good though broad, and some may be difficult to achieve using a rigorous scientific approach in a realistic time frame that is required for the management of Fraser River sockeye salmon stocks.

We agree that there will be significant time delays in achieving the required research outcomes. Because of this, our recommendations are weighed more towards management changes that could happen in the immediate term; changes that will help evolve the fish health programs to a more health protection rather than diagnostic approach.

The one research recommendation that was not explicitly stated was to identify whether hatcheries are having short and/or long-term effects on fish populations. Given the existing 25+

years of fish disease data that DFO has on both wild fish and enhancement facilities, it is currently possible to do a preliminary assessment of pathogen transfer between and within these enhancement facilities and wild stocks. The basic methodology for doing this is outlined in the above sections but, briefly, future studies should include targeted short-term studies (to see the movement of pathogens when hatcheries and/or spawning channels are having disease issues) as well as longer-term studies to assess effects on genetics and abundance of fish.

Please see sub-recommendation 10a, additional point 3 where this question was addressed as one of the 3 critical research questions.

There was also no mention of research to address why the Fraser River sockeye salmon stocks are declining. If you do not know why the stocks are declining it is difficult to ensure that you have addressed the problem. To better understand what is causing the decline see point #5 below.

Our recommendations were informed by our scope of work which was focussed on the ability to undertake a risk assessment regarding infectious diseases from enhancement facilities. However, sub-recommendation 10a, point 3 specifically addressed this concern: namely "What is the proportional role of disease as an independent or additional stressor that influences population health and production of Fraser River sockeye salmon?"

5. What information, if any, should be collected in the future to improve our understanding of this subject area?

In order to understand whether enhancement facilities play a role in the decline of the Fraser River sockeye salmon it is necessary to know why the populations are declining. A thorough investigation of the problem, which identifies the key life stage where the decline is occurring, would help target the time-frame when samples should be collected. It should be possible to identify the most likely time when fish are dying by examining the DFO fisheries information with the expertise of the stock assessment branch of DFO. Although information is not available for all Fraser River sockeye stocks, the department monitors a significant number of them at the egg, smolt, and returning (escape) adult stages. It should be possible to identify whether the problem is in fresh and/or salt water using these monitored populations. Once the stage where the largest effect is occurring is identified, it may then be possible to identify specific pathogens and/or environmental factors that are driving the declines.

As mentioned above, to determine whether pathogens are transferred from enhanced stocks to

"wild" sockeye salmon, a targeted surveillance program should to be implemented that investigates disease outbreaks as they occur in either enhancement facilities or in wild fish populations. These outbreak investigations should establish the temporal and spatial spread of pathogens. Each investigation needs to be tailored to the situation, but sampling should occur during the course of the outbreak and should establish the distribution of the pathogen in time and space (using surrounding facilities and wild fish). The DFO diagnostic laboratory does this to some extent, but they do not have the personnel or resources to conduct thorough outbreak investigations with extensive targeted sampling.

6. Please provide any specific comments for the authors.

The report identifies some critical points that could potentially result in pathogen transfer between enhancement facilities and wild fish populations. Regardless of whether or not there is evidence of pathogen transfer via these transmission routes, they should be addressed. I think it is unfortunate that the report provided very little evidence on whether or not enhancement facilities are potentially contributing to the decline of the Fraser River sockeye salmon populations. I realize this is difficult to do, but I wonder why the research team didn't conduct a preliminary spatial and temporal analysis of disease outbreaks in wild sockeye salmon and enhancement facilities associated with the Fraser River. I realise it would not have been conclusive, but it may have helped prioritize some of the issues.

Please see comments above on the cautions about undertaking spatial or temporal analysis

Appendix 3: Overview of salmonid enhancement in British Columbia

The following is a brief overview of salmon enhancement hatcheries operated by Fisheries and Oceans Canada (DFO) and the Freshwater Fisheries Society of BC (FFSBC) in the Fraser River drainage basin and Strait of Georgia. Efforts to enhance all seven species of Pacific salmon including coho salmon (*Oncorhychus kisutch*), chinook salmon (*Oncorhynchus tswatcha*), sockeye salmon (*Oncorhynchus nerka*), pink salmon (*Oncorhynchus gorbuscha*), chum salmon (*Oncorhynchus keta*), steelhead and rainbow trout (*Oncorhynchus mykiss*) and cutthroat trout (*Oncorhynchus clarki)* will be introduced.

The term enhancement in this scope of work means any human intervention that aims to increase the survival or production of salmonids. The term hatchery refers to an enhancement facility that combines captive incubation and rearing (feeding/husbandry) of those fish. The term spawning channel refers to an artificial channel created adjacent to rivers to provide additional area in which salmon can spawn. Spawning channels can be controlled through manipulation of water flow and consequently the number of fish allowed to spawn in the channel.

Introduction

In the late 1970's, DFO launched the Salmonid Enhancement Program (SEP). The goal of the SEP is to ensure that the public has access to harvest opportunities and that the public supports the protection, stewardship and rebuilding of salmon and their habitat. To accomplish this goal, SEP undertakes projects that produce salmon; support vulnerable salmon populations; and enable First Nations, local communities and external parties to participate in cooperative fisheries and watershed stewardship activities.

There are 3 "branches" of the SEP. First, major hatcheries (larger hatcheries operated by DFO staff) and spawning channels have been constructed on some salmonid-producing rivers in order to increase the survival of Pacific salmon during the freshwater life history phase. Second, the Community Economic Development Program (CEDP) works in partnership with First Nations and other community groups to operate local enhancement hatcheries / projects. Third, SEP provides financial and technical assistance to numerous Public Involvement Projects (PIP) to operate small scale hatcheries and pursue related stewardship endeavours.

The BC Ministry of Environment (MOE) is responsible for steelhead and cutthroat trout population management. MOE provincial regional biologists determine the stocking levels, types and sizes to be released into BC's lakes. The Freshwater Fisheries Society of BC (FFSBC) is a non-profit organization that provides the rearing and stocking activities to meet MOE's management goals. The Society's mandate to conserve, restore and enhance the freshwater fish

resources of BC involves enhancing freshwater recreational fisheries, supporting the recovery of endangered fish populations, and promoting a conservation ethic and interest in recreational fishing. Several federal hatcheries have cooperative arrangements with FFSBC to raise steelhead and cutthroat trout.

Enhancement Facilities

Although original enhancement activities began early in the century and were mostly focused on sockeye salmon, the SEP began in the late seventies and has evolved considerably to encompass the enhancement all seven Pacific salmon species. A list of these facilities along with what species are cultured for enhancement is provided in Table A1 while the locations are shown in Figure A1.

Table A1. Summary of federal and provincial salmonid enhancement facilities examined in the Fraser River drainage basin and Strait of Georgia.

Agency / Program	Facility Name	Species produced (common name)	Hatchery (H); Spawning Channel (C)
DFO / CEDP	Deadman River Project	Coho, Chinook	H
DFO / CEDP	Seymour Hatchery	Coho, Chinook , Chum, Pink, Steelhead	H
DFO / CEDP	Sechelt (AKA MacLean Bay Hatchery)	Coho, Chinook, Chum, Pink	H
DFO / CEDP	Powell River Project	Coho, Chinook, Chum	H + C
DFO / CEDP	Sliammon Project	Chum	H + C
DFO / CEDP	Port Hardy / Quatse Hatchery	Steelhead, Coho, Chum, Pink	H
DFO / CEDP	Gwa'ni (aka Namgis)	Coho, Chinook, Chum, Sockeye	H
DFO / CEDP	Nanaimo River Hatchery	Coho, Chinook, Chum, Pink	H
DFO / CEDP	Cowichan River Hatchery	Coho, Chinook	H
DFO / CEDP	Chemainus River Hatchery	Chinook, Coho	H
DFO	Nadina Spawning Channel	Sockeye	C

DFO	Horsefly Spawning Channel	Sockeye	C
DFO	Shushwap River Hatchery	Coho, Chinook	H
DFO	Spius Creek	Coho, Chinook	H
DFO	Gates Spawning Channel	Sockeye	C
DFO	Tenderfoot Creek Hatchery	Coho, Chinook , Chum, Pink, Steelhead	H
DFO	Pitt River Upper	Sockeye	H
DFO	Weaver Creek Spawning Channel	Chum, Pink, Sockeye	C
DFO	Chehalis River	Steelhead, Coho, Chinook, Cutthroat	H
DFO	Chilliwack River	Steelhead, Coho, Chinook, Chum	H
DFO	Inch Creek Hatchery	Steelhead, Coho, Chinook, Chum	H
DFO	Capilano Hatchery	Steelhead, Coho, Chinook	H
DFO	Quinsam River Hatchery	Steelhead, Coho, Chinook, Pink, Cutthroat	H
DFO	Puntledge Hatchery	Coho, Chinook, Chum, Pink	H
DFO	Big Qualicum Hatchery	Steelhead, Coho, Chinook, Chum, Cutthroat	H
DFO	Little Qualicum Hatchery	Steelhead, Coho, Chinook, Chum, Cutthroat	H
FFSBC	Clearwater Trout Hatchery	Rainbow, Cutthroat, Kokanee	H
FFSBC	Fraser Valley Trout Hatchery	Steelhead, Rainbow, Cutthroat	H
FFSBC	Vancouver Island Trout Hatchery	Steelhead, Rainbow, Cutthroat	H

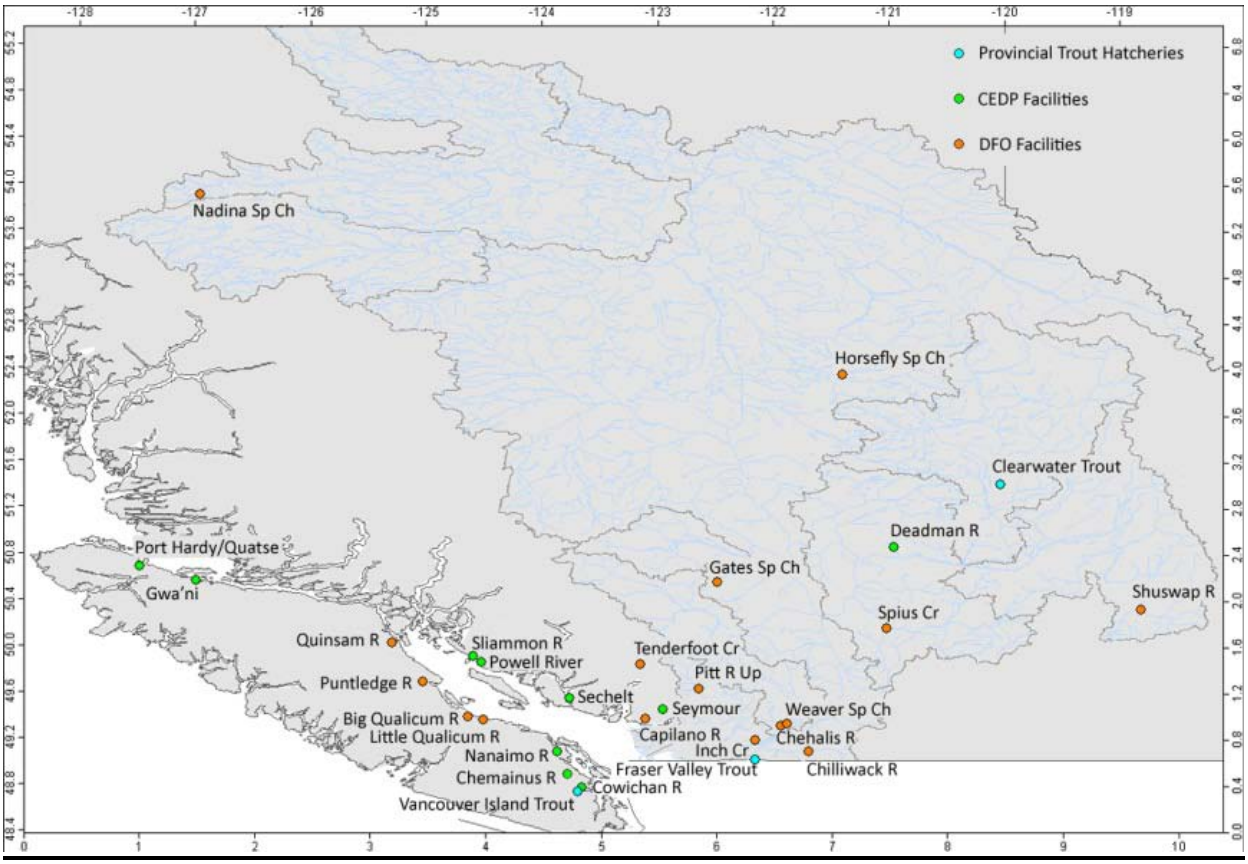

Figure A1: Location of DFO and FFSBC hatcheries (by project name) included in this review. GIS information from DFO Spatial Data Holdings; map generated in SAGA: System for Automated Geoscientific Analysis.

Enhancement Methods

The federal and provincial facilities included in this review used spawning channels and hatcheries independently or in combination. Other methods not employed include various techniques for improving freshwater habitats. Hatcheries predominate both the FFSBC and DFO methodology for enhancement and vary greatly in approach. Depending on the facility and the species of fish, a variety of hatchery methods are used to collect broodstock, incubate eggs and rear offspring to either the fry (fed or unfed) or smolt stage.

Spawning Channels

Man-made spawning and rearing channels vary in size but all provide additional habitat in which salmon survival is higher than in natural areas of the same watershed. Spawning channels provide clean, uniform spawning gravel and often controlled water flow to optimize temperature and reduce the negative impacts from flood / drought events. By using a weir or fish fence, the

number of returning salmon “loaded” into the channel can be controlled. Sockeye salmon spawning channels are often closely associated with sockeye salmon lake habitat.

Hatcheries

Broodstock collection

Pacific salmon broodstock are collected from September to December from rivers during the migration of fish upstream to spawning grounds. Steelhead trout have 2 distinct runs (winter and summer) with adults returning during their respective season. Usually, hatcheries will focus broodstock collection on their associated rivers alone, with a few facilities functioning as central locations for collections for spawning and rearing from a number of local streams. Broodstock capture at the DFO hatcheries is accomplished by a variety of means including; beach seining in river pools, angling, using fish fences and weirs to aggregate returning salmon and the construction of attraction channels. An attraction channel connects the hatchery to the river. It allows mature salmon to swim directly into holding raceways or ponds.

FFSBC hatcheries maintain a few different strains of rainbow and cutthroat trout as captive broodstock. There are also wild broodstock lakes in which adults are trapped with nets and transported to the hatchery where the eggs are incubated and subsequent offspring are then stocked into various lakes in the province. Steelhead and cutthroat trout broodstock are angled by fisherman and sent to a hatchery for spawning, incubation and rearing to pre-smolt size.

Egg collection

After a period of holding in the hatchery fish become ready to spawn. Removing eggs from Pacific salmon (with the exception of steelhead / rainbow trout and cutthroat trout) is a fatal process. Females are killed with a swift blow to the top of the head. The fish is hung upside down by the caudal fin and the gills are slit to bleed the fish and prevent blood from contaminating the eggs upon collection. The abdomen of the fish is carefully opened up and the eggs scooped into a bowl by hand. Milt (semen) is collected by gently squeezing the male’s belly in a sweeping motion anterior to posterior. Typically, eggs from one female are fertilized with the milt from 2 males.

Steelhead / rainbow and cutthroat trout are live spawned which means both eggs and milt can be gently squeezed from the adults body into a bowl. Adults can be spawned more than once in a lifetime. Egg takes occur March – May.

Incubation

Incubation of fertilized eggs can occur in a variety of ways. Most hatcheries incubate and hatch indoors in a controlled hatchery environment in Heath trays or similar stack incubators. These methods can keep large volumes of eggs in a cold, dark environment protected from predators and the public. A simple form of incubation is the use of in-stream incubators, in which incubation occurs in "cassettes" or "bam boxes". When fry cultured in in-stream incubators reach the swim-up stage, they migrate directly into the stream (also called volitional release). The FFSBC commonly shocks eggs during incubation to produce sterile offspring (or triploids) for introductions.

Rearing

Salmon normally deposit their eggs in gravel of streambeds and develop there from eggs stage to alevins (newly hatched fish with the yolk sac still attached) and then into free swimming juveniles (fry) in the gravel of the streambed. This is identical to life in a spawning channel. In both the provincial and federal hatcheries, alevins develop into fry in containers in the hatchery, or in semi-natural or artificial channels. The fry may be kept and fed in enhancement facilities, or they may be released without ever being fed. If they are kept, they are later released as fed fry or smolts (young salmon physiologically ready to go to sea). Early rearing can be accomplished in a variety of systems, usually flow-through. This includes the use of aluminum or fibreglass troughs, concrete or fibreglass raceways, earthen ponds, and lake net pens. Occasionally, fish will be fed in seawater net pens for safe rearing and feeding before being released. Commercial trout and salmon feeds are widely used.

Release

Release of smolts occurs in spring for coho salmon, chinook salmon, chum salmon, steelhead and cutthroat trout. Chum and pink salmon are released either unfed after emergence from channels or incubation boxes, or as fed fry from hatcheries or sea pens after few weeks of feeding. Coho salmon are released as fry, either at emergence or after three to five months of rearing, or as smolts after one year of rearing. The majority of sockeye salmon are released as unfed fry, although a small number are incubated and reared in a hatchery. In the case of chinook salmon, coastal stocks are released after three to four months of rearing, while interior stocks are frequently reared to the yearling stage before release.

The fry or smolts released from the hatchery are called "hatchery releases" or "hatchery production." Fish may be directly released into receiving waters or transported. Live haul tank

truck are used to transport fish directly to the targeted water body. For remote locations FFSBC has released fish via helicopter.

The FFSBC rears juveniles to be released at different ages (or sizes) (Table A2). To some extent, the age for release is determined by the method of release (e.g. remote access), the season for planned releases and hatchery rearing facility limitations. However, the most appropriate age/size for release will also be determined by a number of factors including the likelihood of winterkill, presence of predators/competition, priority of the system, and proximity to urban centers.

Table A2. Categories of juvenile salmonids commonly released by FFSBC.

Juvenile catergory	Size at release	Suitability	Timing of release Vancouver Island	Timing of release Lower Mainland
Fall fry	<5 g	• appropriate for remote access (most fish per volume) in a productive monoculture environment (i.e. no competition or predation) • cost-efficiency (lowest cost per fish) • reduce the incidence of early maturation under some conditions	Cutthroat: mid June Rainbow fry: late August	Cutthroat: September – October Rainbow: mid August
Yearling	10-25 g	• better survival especially where other species occur competition/predation pressure • some cost-efficiency (compared to catchables) • slight adaptive advantage over catchables.	Cutthroat: mid March – mid April Rainbow: May 1-30th	Cutthroat: March – April Rainbow: mid April – late May
Smolts	Approx 100 g	• increased survival in freshwater • shorter residence time in freshwater	Steelhead smolts: mid April – mid May Anadromous	Steelhead smolts: late April – late May

			cutthroat: early April	Anadromous cutthroat: early April – mid May
Catchables	12” +	• appropriate in urban setting or high creel areas • immediately available to fishery • most expensive to raise	Rainbow: early March, mid May, mid September, late October	Rainbow: March – May, September - November

Appendix 4: Literature search key terms

Key terms and search phrases used individually or in combination as part of the literature review.

A (Anadromous and (salmon or salmonid)) or coho or Chinook or chum or "pink salmon" or sockeye or steelhead or "rainbow trout" or "cutthroat trout" or (oncorhynchus and (kisutch or tshawytscha or keta or gorbuscha or nerka or mykiss or clarkii))

B Hatchery or hatcheries or "spawning channel" or fishway or "incubation box" or "incubation boxes" or "side-channel" or "fry release" or enhancement

C Release or survival or returns or viable or “salmon run” or “in-migration” or “out-migration” or transplant or “stock restoration” or stocking or smolt or smoltification

D Canada or "British Columbia" or "Fraser Valley" or "Fraser River" or "Georgia Strait" or "Strait of Georgia" or "Gulf of Georgia"

E Pathogen or pathogens or pathogenic or parasite or parasites or parasitic or viral or virus or bacteria or bacterial or fungus or fungal or protozoa or protozoan or myxozoa or myxozoan or helminth or infection or infectious or disease

F Subclinical or “pre-symptomatic” or “chronic disease” or “incidental findings” or morbidity

G Infectious disease terms including: IHN or “Infectious Hematopoietic Necrosis Virus” or “*Vibrio anguillarum”* or *“Aeromonas salmonicida”* or *“Renibacterium salmoninarum” or “Ichthyophthirius multifiliis”* or Ich or “*Parvicapsula minibicornis” or* “*Flavobacterium* spp” or “Coldwater disease” or “*Saprolegnia* spp” or “*Ichthyophonus hoferi”* or *“Cryptobia salmositica”* or *“Tetracapusolides bryosalmonae”* or *“Eubothrium* spp” or “*Lepeophtheirus salmonis”*

H Guidelines or program or procedure or permits or plans

I Monitoring or surveillance or survey or “best practices” or tracking or “risk assessment” or “pre-release risk assessment” or “health oversight” or diagnostics or screening or mortality or “population effects” or “population impacts”

J “Captive brood stock” or introductions or transfers or transplant or “Carcass placement” or “Coho fry planting” or “Sockeye culture” or “small scale enhancement” or incubation or rearing or release or “Fish health management” or Fish health Standard operating”

K Waste or wastewater or effluent or sewage or “out-flow” or discharge

L Carcass or mortalities or “stream enrichment” or “in-stream placement” or “carcass placement

Appendix 5: Averaged release data from DFO, CEDP, PIP and FFSBC facilities

Data source as in appendix 6

Table A2: Annual salmonid release numbers for all DFO facilities (hatcheries and spawning channels) within the Fraser River drainage and Strait of Georgia, 2005-2009.

Release Year	Chinook	Chum	Coho	Cutthroat	Pink	Sockeye	Steelhead	Total
2005	21,181,525	68,057,944	8,738,382	32,938	7,884,846	49,216,595	407,073	155,519,303
2006	18,011,884	39,866,727	6,252,921	31,790	11,107,740	50,939,139	263,960	126,474,161
2007	19,639,824	72,641,026	5,757,622	28,764	7,556,741	63,122,558	184,060	168,930,595
2008	18,307,718	51,724,795	6,758,089	35,787	11,805,293	37,805,563	252,173	126,689,418
2009	16,677,322	39,368,003	7,243,073	24,310	8,450,041	12,329,001	218,750	84,310,500
Total	93,818,273	271,658,495	34,750,087	153,589	46,804,661	213,412,856	1,326,016	661,923,977

Table A3: Annual salmonid release numbers for all CEDP facilities within the Fraser River drainage and Strait of Georgia, 2005-2009.

Release Year	Chinook	Chum	Coho	Cutthroat	Pink	Sockeye	Steelhead	Total
2005	1,559,835	3,738,075	876,489	976	2,226,592	737,541	110,401	9,249,909
2006	3,918,155	8,161,480	920,749	882	3,229,944	1,247,959	86,043	17,565,212
2007	2,700,557	7,058,271	898,895		2,891,570	1,148,573	70,943	14,768,809
2008	1,807,350	2,190,287	1,063,290		4,691,741	609,141	115,812	10,477,621
2009	3,452,574	4,356,856	1,465,674		1,950,060	688,290	113,819	12,027,273
Total	13,438,471	25,504,969	5,225,097	1,858	14,989,907	4,431,504	497,018	64,088,824

Table A4: Annual salmonid numbers for all PIP facilities within the Fraser River drainage and Strait of Georgia, 2005-2009.

Release Year	Chinook	Chum	Coho	Pink	Sockeye	Total	Average
2005	1,143,437	2,772,616	1,937,360	1,609,518	25,927	7,488,858	1,497,772
2006	1,396,058	2,109,816	1,659,614	6,125,709	94,510	11,385,707	2,277,141
2007	1,625,165	2,302,130	1,209,601	1,300,000	86,061	6,522,957	1,304,591
2008	1,312,658	2,358,639	1,025,769	6,402,741	1,101,564	12,201,371	2,440,274
2009	1,567,283	2,923,251	1,519,141	4,749,143		10,758,818	2,689,705
Total	7,044,601	12,466,452	7,351,485	20,187,111	1,308,062	48,357,711	

Table A5: Annual salmonid release numbers for all Provincial facilities within the Fraser River drainage and Strait of Georgia, 2005-2009.

Release Year	Coastal cutthroat trout	Eastern brook trout	Kokanee	Rainbow trout	Steelhead	Total
2005	152,486	275,480	1,319,463	3,073,013	8,139	4,828,581
2006	122,320	258,000	834,495	2,994,269	6,000	4,215,084
2007	78,201	187,202	610,541	2,563,864	51,786	3,491,594
2008	63,718	205,547	1,330,026	3,909,753	17,751	5,526,795
2009	60,161	201,224	1,007,632	3,530,791	23,440	4,823,248
Total	476,886	1,127,453	5,102,157	16,071,690	107,116	22,885,302

Appendix 6: Annual patterns of fish releases including data from FFSBC and DFO hatcheries

Salmonid release data obtained from Ryan Galbraith via a request to the Cohen Commission contained release end dates in the format yyyymmdd for the major DFO facilities and CEDP hatcheries within the geographic scope of this review. Provincial hatchery historical releases (ddmmyy format) were also provided through a request to the Cohen Commission (source unknown); however, as there was no indication whether or not these were release start or release end dates, we made the assumption that they were release end dates. Using Microsoft Excel, dates were converted into a month number (1-12) and week number (1-53); DFO and CEDP release statistics by week and species for the period 2005-2009 were then summed to create Figure A2. A separate figure, A6, was created to show total DFO and CEDP Cutthroat and Steelhead releases by month for the time period 2005-2009. Provincial release statistics by hatchery, month and species for the period 2005-2009 were summed to create Figures A3-A5.

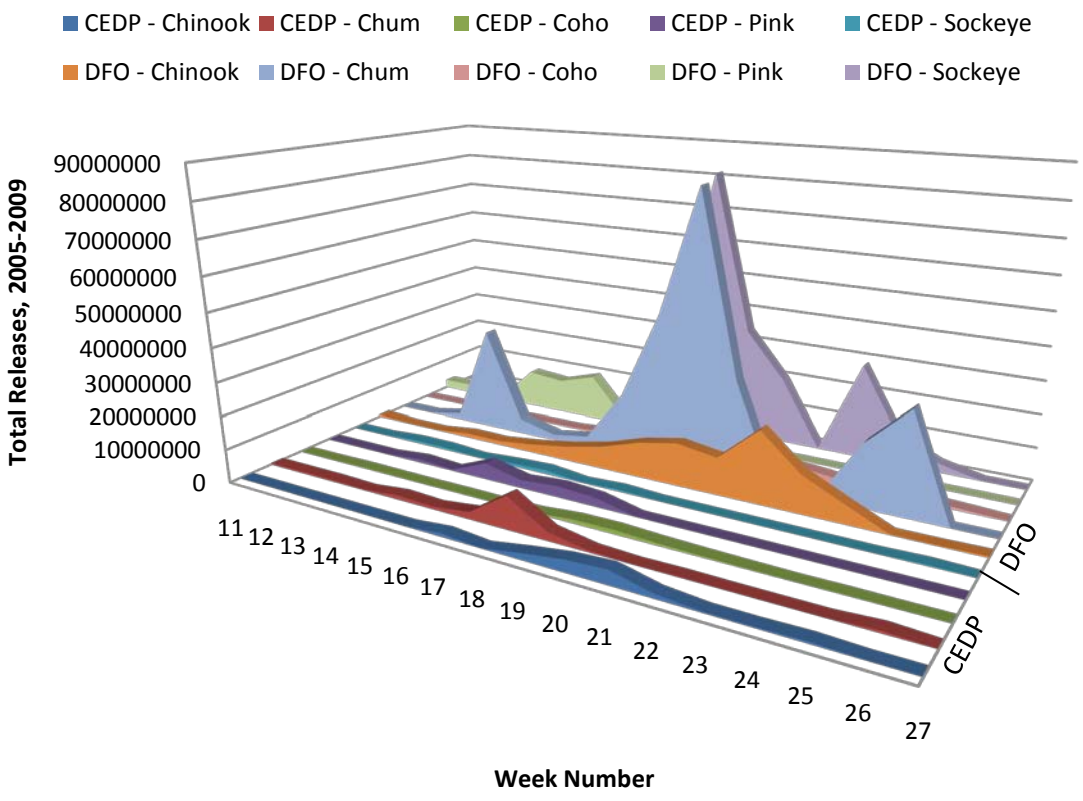

Figure A2: Total DFO and CEDP release numbers into the Fraser River Watershed and Strait of Georgia by week in the period March to June, 2005-2009.

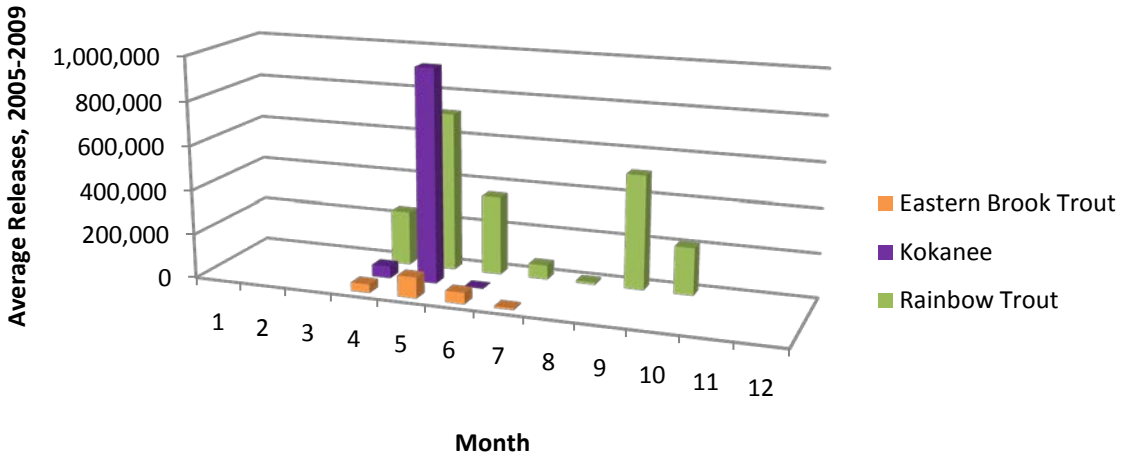

Figure A3: Average monthly releases from Clearwater Hatchery, 2005-2009.

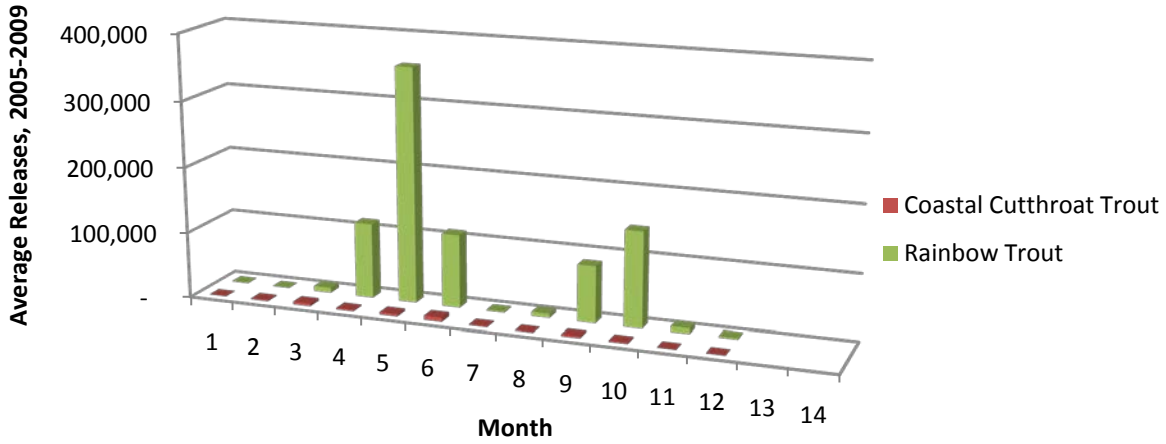

Figure A4: Average monthly releases from Fraser Valley Hatchery, 2005-2009.

Fraser Valley Hatchery also raised steelhead trout in the years assessed. Steelhead is not represented in this figure because release numbers ranged from 2,169 to 11,724 per year with no releases in 2009. In 2007, steelhead were released 3 times throughout the year (in February,

April and November); releases in the remaining years only occurred in September (2005), December (2006) and April (2008).

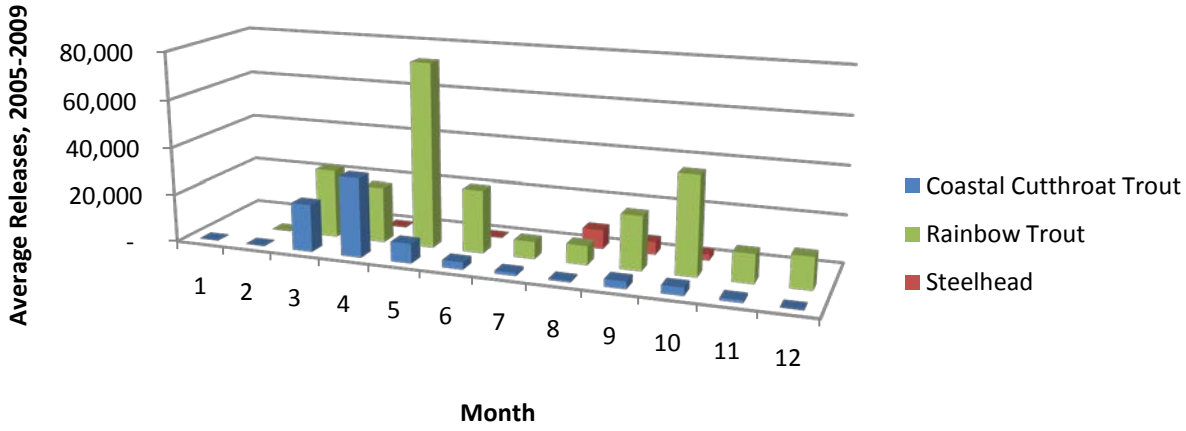

Figure A5: Average monthly releases from Vancouver Island Hatchery, 2005-2009.

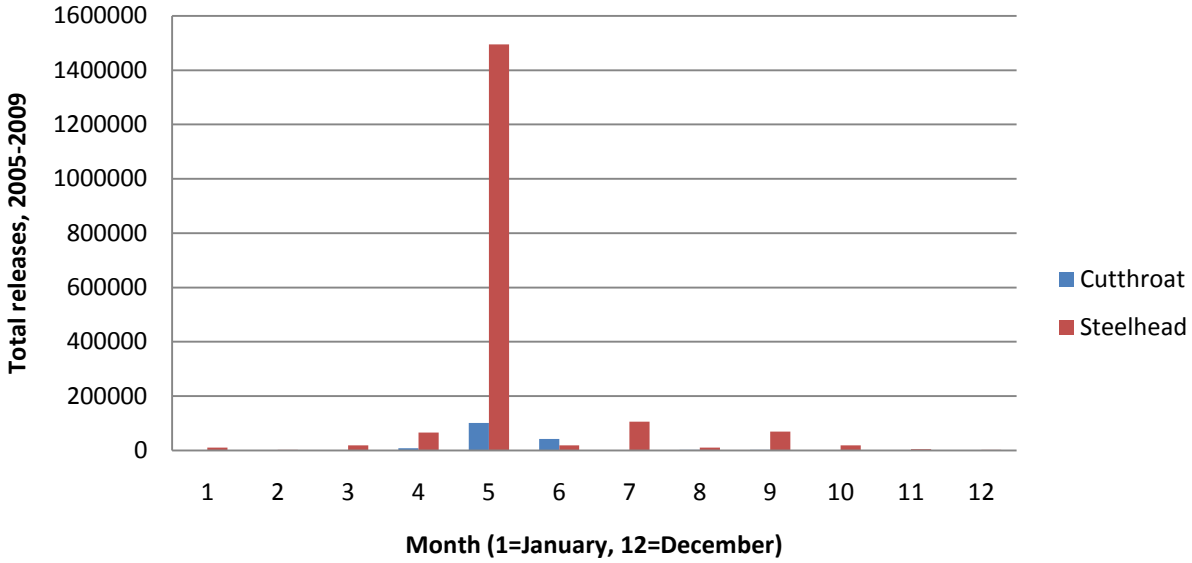

Figure A6: Total releases of cutthroat trout and steelhead from CEDP and DFO facilities in the Fraser River waterbasin and Strait of Georgia by month, 2005-2009.

Appendix 7: Annual patterns of fish releases including data from FFSBC and DFO hatcheries

DFO Hatchery and Spawning Channels (2000-2010)

For sockeye salmon specific data, numbers in parentheses refer to fish classified as wild or semi-wild.

Major DFO Facilities Diagnostic Records from PBS (2000-2010)				All Salmonids		Sockeye Salmon		
Zone	Facility Type	Year	Hatchery N =	Number of Lab Submissions	Number of Fish Submitted to Lab	Hatchery N =	Number of Lab Submissions	Number of Fish Submitted to Lab
Fraser River	Hatchery	2000	5	25	568	0 (1)	0 (1)	0 (2)
		2001	4	23	601	1	4	91
		2002	5	45	1467	2 (2)	13 (5)	511 (132)
		2003	4	44	876	2 (2)	4 (2)	163 (152)
		2004	3	32	705	2 (1)	2 (1)	54 (12)
		2005	3	17	549	-	-	-
		2006	3	19	328	-	-	-
		2007	3	7	140	2	No Data	No Data
		2008	4	13	283	2 (1)	2 (1)	11 (4)
		2009	4	30	953	1	1	92
		2010	3	4	191	-	-	-
				259	**6661**		**27 (10)**	**924 (303)**
		Total with Spius removed			**90**	**2051**		
	Spawning Channel	2000	1	3	135	0 (1)	0 (2)	0 (129)
		2008	1	1	30	0 (1)	0 (1)	0 (30)
		2009	2	2	27	2 (1)	2 (1)	27 (20)
				6	**192**		**5 (4)**	**186 (179)**
	Fraser River Total			**265**	**6853**		**32 (14)**	**1110 (482)**

		Total with Spius removed		**96**	**2243**			
Southern Coast	Hatchery	2000	4	28	862	-	-	-
		2001	3	5	185	-	-	-
		2002	3	12	252	0 (1)	0 (5)	0 (26)
		2003	4	25	664	1	4	158
		2004	4	22	620	1 (1)	17 (1)	556 (3)
		2005	3	8	198	-	-	-
		2006	3	5	176	1	1	120
		2007	4	6	124	1	1	21
		2008	4	8	165	1	2	8
		2009	4	19	583	1	9	320
		2010	1	1	60	-	-	-
				139	**3889**		**39 (6)**	**1209 (29)**
	Combined (Hatchery / Spawning Channel)	2000	1	1	8	-	-	-
		2001	1	1	3	-	-	-
		2004	2	2	26	-	-	-
		2006	1	4	112	-	-	-
		2007	1	1	60	-	-	-
		2010	1	1	60	-	-	-
				10	**269**		**-**	**-**
	Southern Coast Total			**149**	**4158**		**39**	**1209**
DFO Facilities Total				**414**	**11011**		**71 (20)**	**2319 (511)**

FFSBC Hatcheries 2000-2010

Provincial Diagnostic Records (2000-2010)

			All Salmonids			Kokanee Salmon		
Zone	Facility Name	Year	Hatchery N =	Number of Lab Submissions	Number of Fish Submitted to Lab	Hatchery N =	Number of Lab Submissions	Number of Fish Submitted to Lab
Fraser River	Clearwater Trout Hatchery	2006	1	3	110	-	-	-
		2007	1	4	117	-	-	-
		2008	1	5	115	-	-	-
		2009	1	3	150	1	2	120
		2010	1	3	130	1	1	40
				18	**622**		**3**	**160**
	Fraser Valley Trout Hatchery	2000	1	10	214	-	-	-
		2001	1	9	148	-	-	-
		2002	1	7	179	-	-	-
		2003	1	7	112	-	-	-
		2004	1	9	157	-	-	-
		2006	1	10	302	-	-	-
		2007	1	35	830	-	-	-
		2008	1	38	712	-	-	-
		2009	1	20	327	-	-	-
		2010	1	28	646	-	-	-
				173	**3627**		**-**	**-**
	Fraser River Total			**191**	**4249**		**3**	**160**
Southern Coast	Vancouver Island Trout Hatchery	2000	1	13	225	-	-	-
		2001	1	11	294	-	-	-
		2002	1	10	243	-	-	-

	2003	1	15	381	-	-	-
	2004	1	12	259	-	-	-
	2005	1	2	61	-	-	-
	2006	1	1	3	-	-	-
	2007	1	5	162	-	-	-
	2008	1	6	215	-	-	-
	2009	1	7	261	-	-	-
	2010	1	13	433	-	-	-
			95	**2537**		**-**	**-**
Southern Coast Total			**95**	**2537**		**-**	**-**
Provincial Facilities Total			**286**	**6786**		**3**	**160**

CEDP and PIP Diagnostic Data 2000-2010

Numbers of fish submitted to the lab was not available in records provided

CEDP and PIP Diagnostic Records from PBS (2000-2010)				All Salmonids			Sockeye Salmon		
	Zone	Facility Name	Year	Hatchery N =	Number of Lab Submissions	Number of Fish Submitted to Lab	Hatchery N =	Number of Lab Submissions	Number of Fish Submitted to Lab
	Fraser River	CEDP	2004	1	1	-	-	-	-
			2005	1	1	-	-	-	-
			2006	1	1	-	-	-	-
			2007	1	1	-	-	-	-
			2009	1	3	-	-	-	-
			2010	1	1	-	-	-	-
					8	-		-	-
		PIP	2005	1	1	-	-	-	-
			2009	1	1	-	-	-	-
					2	-		-	-
	Fraser River Total				**10**	-		-	-
	Southern Coast	CEDP	2000	1	1	-	-	-	-
			2001	1	4	-	1	3	-
			2002	2	3	-	-	-	-
			2003	1	1	-	-	-	-
			2004	5	14	-	-	-	-
			2005	4	13	-	-	-	-
			2006	4	7	-	-	-	-
			2007	2	6	-	1	3	-

		2008	4	4	-	1	1	-
		2009	5	12	-	1	1	-
		2010	2	2	-	-	-	-
				67	-		**8**	-
	PIP	2004	1	1	-	-	-	-
		2005	2	4	-	-	-	-
		2006	2	3	-	-	-	-
		2008	2	4	-	-	-	-
		2009	2	4	-	-	-	-
		2010	1	1	-	-	-	-
				17	-		-	-
	Southern Coast Total			**84**	-	-	**8**	-
CEDP and PIP Total				**94**	-	-	**8**	-