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

February 2011

**TECHNICAL REPORT 1**

# Infectious Diseases and Potential Impacts on Survival of 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

Numerous pathogens have been reported in sockeye salmon and a few of them have been documented to be, or are, potential causes of significant mortality in this salmon species in the Fraser River system. At present, there are no direct links between a specific pathogen and sockeye salmon survival at a population level in British Columbia. This report reviews 5 viral, 6 bacterial, 4 fungal, and 19 parasitic pathogens that are known to or could potentially infect sockeye salmon. Two idiopathic diseases are also discussed. For each pathogen, a subjective assessment of risk for causing significant disease in wild sockeye salmon in the Fraser River system is provided. This risk is based on 1) the known or suspected virulence of the pathogen to Pacific salmon in general, and specifically to sockeye salmon and 2) the likelihood that the pathogen would be prevalent in the Fraser River or British Columbia. These conclusions were based on review of the peer-reviewed literature, government documents from Fisheries and Oceans Canada (DFO), and interviews with DFO fish health scientists. I designated the following pathogens as potential "High Risk": IHN virus, three bacteria (*Vibrio anguillarum, Aeromonas salmonicida, Renibacterium salmoninarum*), and two parasites (Ich - *Ichthyophtheirus multifillis* and the myxozoan *Parvicapsula minibicornis*).

The IHN virus is well recognized as a lethal pathogen to fry sockeye salmon in freshwater. It also occurs in marine waters in BC, and has caused several outbreaks in pen-reared Atlantic salmon. Post-smolt sockeye salmon are less susceptible, but recent evidence suggests that there is variability in the virulence of this virus between isolates, and thus it is conceivable that some strains may be more pathogenic to sockeye salmon in the ocean. The three bacterial pathogens are included in the High Risk category as they are recognized as virulent pathogens in both hatcheries and netpens. *Vibrio anguillarum* is ubiquitous in the marine environment, the other two bacteria are occasionally reported in wild salmon. However, outbreaks in wild salmon, including sockeye salmon, in British Columbia have not been documented for these pathogens. In contrast, both Ich and *Parvicapsula* have been documented to be associated with pre-spawning mortality in sockeye salmon, and the latter also infects outmigrant smolts.

Pathogens assigned to the Moderate Risk category were *Flavobacterium* spp., fungi belonging to the genus *Saprolegnia*, the fungus-like pathogen *Ichthyophonus hoferi*, the PKX myxozoan, *Eubothrium* spp. tapeworms, and sea lice (*Lepeophtheirus salmonis* and *Caligus clemensi*). *Flavobacterium* and *Saprolegnia* spp. are recognized as significant, but usually opportunistic, pathogens in salmon in freshwater when environmental conditions are suboptimal, and thus could cause severe disease if the Fraser River system or marine environment is compromised. *Icthyophonus hoferi* is of concern as it recently has been increasing in Chinook salmon in the Yukon River. *Eubothrium* is one worm parasite that has been already shown to compromise wild sockeye when infections are heavy. Last, the caligid copepods were included

on the list. Whereas not documented to cause mortalities in wild sockeye salmon, recent claims of sea lice killing wild pink salmon in British Columbia warrants investigations on the impact of these copepods on post-smolt sockeye salmon. One putative disease was place designated as "Unknown". Here Dr. K. Miller-Sauders at DFO, Pacific Biological Station (PBS), Nanaimo, recently discovered an unusual gene signature suggestive of a virus infection in sockeye salmon, and temporal studies showed that these fish had reduced survival. The list agrees for the most part with one independently developed by Dr. Kyle Garver, DFO-PBS, where he concluded that IHN virus, *Parvicapsula*, and Ich are the pathogens of most concern in sockeye from this system.

All of these pathogens are endemic to British Columbia and most likely have been present in this area for centuries. Moreover, there is no evidence of an exotic salmonid pathogen being recently introduced to the Province. If there has been a dramatic increase in mortality caused by one or more of them in recent years, it is likely due to changes in the susceptibility of sockeye salmon to them or a change in the abundance in these pathogens. Environmental changes could be an underlying cause of either. Fish are very closely tied to their environment, and thus water quality and other environmental parameters play a very important role in their susceptibility and severity of diseases. Changes in water temperature, either in freshwater or seawater, are important likely candidates. Fish are cold-blooded (poikilothermic) and thus both their pathogens and the fish themselves are extremely influenced by temperature.

There are certainly many pathogens that occur in wild sockeye salmon, but their precise impacts on survival in these stocks are poorly understood. Hence, there are not firm links for these pathogens with significant demise in these sockeye populations overall, but some of these are clearly associated with prespawning mortality in freshwater. The absence of data on pathogens and diseases in wild salmon in British Columbia is a reflection of the historical research focus on fish diseases, in both the Province and other regions. Most research on salmonid diseases has been directed toward those afflicting captive fish, either in government hatcheries or private fish farms.

As with many scientific issues, more research is needed to elucidate the impacts of pathogens on Fraser River sockeye salmon. Surveys for pathogens and diseases in wild sockeye salmon must be conducted and maintained over several years to provide the needed raw data. Surveys must include proper identification of pathogens, geographic and host distribution, and abundance or severity of infection. With these data in hand, researchers can conduct the appropriate analyses to infer or document the role that these pathogens have with survival in various life stages. After a pathogen is shown to be associated with mortality, modelers, mathematicians, statisticians, and ecologists could then conduct investigations to elucidate which factors (e.g., water temperature, river flow, land use practices, netpen farming) influence the distribution and abundance of these pathogens. Isolation, identification of agents, and controlled

laboratory studies are needed to elucidate the pathogenesis of newly recognized pathogens, such as the putative virus associated with specific gene functions.

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# INTRODUCTION

Salmonid fishes, including sockeye salmon, are host to a wide variety of pathogens, ranging from viruses, bacteria, fungi, protozoans, helminths, and arthopods. The vast majority of research on diseases of salmonids has been directed towards those observed in captivity, in either government hatcheries or private aquaculture facilities, such as netpens. There are surveys of pathogens in wild salmon in the Pacific Northwest (e.g., Margolis and Arthur 1979; McDonald and Margolis 1995; Kent et al. 1998; Arkoosh et al. 2004), but there have been only a few infectious diseases that have been shown or implicated to cause significant mortality in wild salmon in British Columbia (e.g., Traxler et al. 1998; Johnson et al. 1996; Krkosek et al. 2006). Nevertheless, there are several examples of pathogens causing widespread mortality in other fish species. For example, the VHS virus, which causes viral hemorrhagic septicemia, has recently caused very high mortalities in several fish species in the Great Lakes (Bowser 2009), and a herpes virus caused devastating mortalities in Australian pilchards (*Sardinops sagax*) in Australia following its apparent introduction with bait fish (Murray et al. 2003). Numerous more chronic diseases, usually caused by parasites, have been shown to cause significant mortality in large populations of wild commercial fishes, such *Ichthyophonus hoferi* in herring (McVicar 1999). In contrast to acute, rapid diseases, chronic disease often persist for many months or years, and they may not directly kill their host. In this case, afflicted hosts may grow slower, have reduced swimming abilities, and may be more subject to predation.

Sindermann (1987) provides a rather comprehensive review up to that date on documented impacts of infectious diseases in wild fishes. Regarding salmonids, Vincent (1996) document the impacts of *Myxobolus cerebralis* on wild rainbow trout, and high mortality in Atlantic salmon in Norway was caused by *Gyrodactylus salaris* following introduction of this parasite from Sweden (Johnsen and Jensen 1991). The diseases caused by these parasites emphasizes the potential impact introduced pathogens may have on wild salmon stocks. Fisheries and Oceans Canada has maintained a strict import and quarantine program for salmonids (Kent and Kieser 2003), and it should be noted that to date no exotic salmon pathogen of significance has been documented to have been introduced into British Columbia.

The following is a review of pathogens that have been documented to, or potentially could, cause significant disease in sockeye salmon, particularly in the Fraser River system (Table 1). The emphasis of this report is on infectious diseases, but some discussion on potentially important diseases caused by non-infectious agents or unknown causes is included. This report was based on review of the peer-reviewed literature, reports, presentations, and personal interviews provided by the fish health program at the Pacific Biological Station, Fisheries and Oceans Canada, Nanaimo, British Columbia (DFO-PBS). The peer-reviewed literature was

evaluated using standard search programs such as Google Scholar and PubMed. Reports were obtained with assistance of the Commission. Much of the gray literature (reports) obtained from DFO were from case reports involving screening of enhanced populations (SEP hatcheries, etc.), and hence precise correlation with impacts on wild salmon should be done judiciously. Interviews included phone conversations and in-person meetings at the Pacific Biological Station on 6 December 2010, in which the following DFO scientists were interviewed: Drs. Stewart Johnson, Kristi Miller-Saunders, Kyle Garver, Simon Jones, Mark Higgins, and Garth Traxler.

For each pathogen, a subjective assessment of risk for causing disease in wild sockeye salmon in the Fraser River system is provided. This risk is based on 1) the known or suspected virulence of the pathogen to Pacific salmon in general and specifically to sockeye salmon and 2) the likelihood that sockeye salmon would encounter the pathogen in different phases of their development in the Fraser River or in the ocean. Virulence is assigned based on data from controlled infectious and lethal dose studies or based on the reported severity of a disease in a natural setting. Review of some diseases that have been shown to be severe pathogens in captive or wild salmon other than sockeye salmon are included in this report. However, considerable differences in virulence and lethality may occur when a pathogen infects different salmon species or in different environmental conditions, and thus correlating these diseases with potential problems in wild sockeye salmon should be made with some caution. Information on the history of the occurrence in British Columbia is included. It is important to note that recent reports on the first observed occurrence of a pathogen in a particular location does not confirm that it was introduced at that time, but rather it was at this time that it was first recognized as a problem.

## ASSESSMENT OF PATHOGEN-ASSOCIATED MORTALITY IN WILD SALMONIDS

It is often difficult to evaluate the effects of pathogens on wild fish populations because moribund (sick) fish are often not observed in vast bodies of waters and they are often selectively removed by predators (Gordon and Rau 1982; Bakke and Harris 1998). There are a few examples of actual observations of high mortality in wild salmonids in British Columbia, and these were associated with en route or prespawning mortality. These occurrences are discussed under the sections above on specific pathogens. Approaches to assess the impacts of pathogens on wild fish, particularly salmonids, are discussed here by dividing the pathogens into two categories; those that cause acute disease, are highly pathogenic, and rapidly kill their host, and 2) pathogens that cause chronic infections, in which only heavy infections are associated with sickness or death.

Therefore, with chronic infections, many hosts remain infected for long periods and often appear healthy. These types of infections damage the host by sublethal effects, such as reduced growth and swimming ability, and the host may ultimately die from another cause.

Chronic diseases are discussed here first. Methods have been devised for estimating mortality associated with chronic infections (e.g., parasites) in wild fishes (Anderson and Gordon 1982; Lester 1984) and tested both theoretically (Dobson and May 1987; Rousset et al. 1996) and under natural conditions (Sindermann 1987; Bourque et al. 2006). Lester (1984) provides a concise overview and summary of the common methods used for such analysis with wild fishes. These approaches are well established and are very useful for the elucidation of pathogen-associated mortality in wild fishes. However, most of the tests require relatively large sample sizes for accurate predictions, which may be a limitation with certain salmon populations. Lester's Method 1 is "Decline in prevalence with persistent infections" (Fig. 1a). Parasites capable of persistent infections provide an ideal means for studying parasite-associated mortality in wild fishes because large declines in abundance and prevalence would not be due to immune mediated removal of the parasite by their hosts. There are two requirements to conduct these analyses, the same population must be examined throughout the study and the pathogen in question must persist. In other words, fish do not eliminate the parasite. Hence, a reduction in prevalence between time points indicates pathogen associated mortality. Pertinent to the present investigation, Bradford et al. (2010) showed that fish that died before spawning had more severe *Parvicapsula* infections than surviving fish. Observation of a decline in over-dispersion (i.e., $S^2/X$) is also an indication of parasite-associated mortality. This analysis was recently utilized by Jacobson et al (2008) with coho salmon in Oregon. By evaluating temporal changes in parasite dispersion (mean abundance to variance ratio) in coho salmon populations, they showed that early marine resident mortality of this host was associated with infections by metacercariae of *Nanophyetus salmincola.* Both of these methods are powerful and straightforward, but they require confidence that the same population is sampled overtime. Tracking and collecting samples of salmon from the same population is often difficult because in freshwater fish may grow as separate populations from fry to parr, then commingle as smolts during ocean migration. Tracking specific populations in the ocean would be an additional challenge (Jacobson et al. 2008).

Another method is observation of truncation of the normal negative binomial distribution of pathogen abundance, first described by Crofton (1971) and reviewed by Lester (1984). This is particularly useful with salmon, as it requires only one sample time. The normal distribution of parasite abundance in wild animals is a negative binomial, rather than a bell-shaped curve. This results because in a given population of wild animals, most are not or are lightly infected, while only a few have heavy infections. Parasite associated mortality occurs when observed infection severity (abundance or intensity) is less than would be predicted by a curve generated using

lightly infected animals (Fig. 1b). In other words, Crofton and other parasitologists have shown that truncation of the predicted negative binomial curve at the region of high abundance can be used to estimate a threshold for parasite associated mortality. We recently applied this approach to worm and myxozoan parasites in wild coho salmon in Oregon (Ferguson 2010).

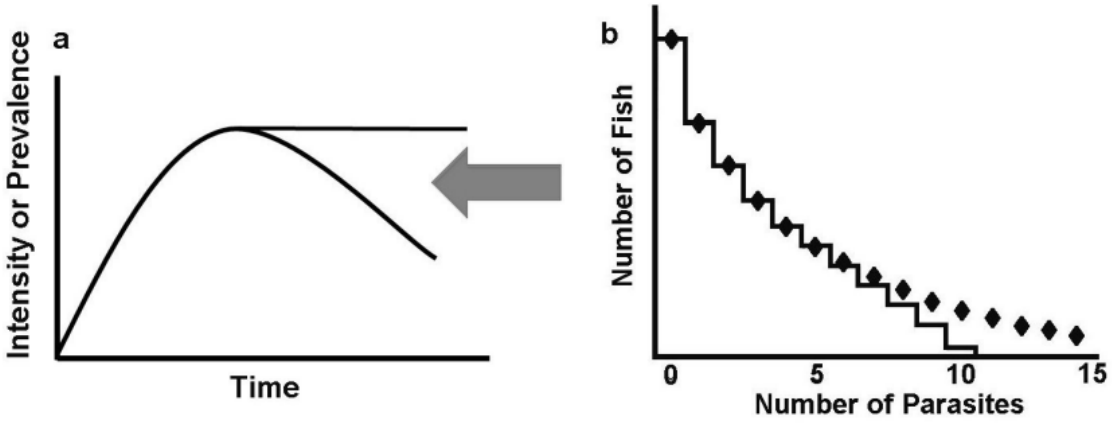


Figure 1. a. Reduction in severity (intensity or prevalence) of persistent pathogen infections over time indicates pathogen-associated mortality (arrow). b. Predicted (diamonds) and actual (lines) binomial distribution of parasites. Note that fewer fish have heavy infections than is predicted, suggesting parasite-associated mortality with heavy infections. Adapted from Lester (1984).

**Highly Lethal Pathogens.** Assessment of pathogen-associated mortality with these infectious agents follows a more straightforward approach. Laboratory transmission studies are used to demonstrate the percent mortality that occurs following infection with different life stages of the host and different doses of the pathogen. Then field observations documenting the prevalence of infections in a given population are used to predict death caused by the pathogen. Laboratory experiments are not direct correlates for the virulence of acute pathogens in the field as the latter is a more complex environment. However, they provide reasonable estimates of mortality that would occur if fish became infected.

Examples of acute pathogens in wild salmon in the Pacific Northwest include the IHN virus, *Ceratomyxa shasta* (Bartholomew et al. 2010), sea lice in returning sockeye salmon in Alberni Inlet (Johnson et al. 1996), and bacterial diseases such as vibrosis in seawater and furnuculosis associated with post-spawning mortality. A third approach that has been used recently is to incorporate field and lab studies together. Here Bartholomew's team exposed salmon at various locations in the Kalamath River in "live cages", transported fish back to the laboratory, and recorded subsequent mortality associated with *C. shasta* (Bartholomew 2010). This group has also developed a model to predict mortalities with *C. shasta* (Ray et al. 2010).

The impact of a specific pathogen may vary dependent on geographical location due to differences in environmental conditions. Therefore, correlating findings based on one geographic area to another area should be done with caution. Sentinel programs involving exposure of Specific Pathogen Free (SPF) salmonids in cages provides a useful method to document the temporal and geographic distribution, but is less useful for assessing the actual pathogen-associated mortality that would occur in wild fish.

## VIRUSES

Several viral diseases have been documented to cause disease and acute mortalities in salmonid fishes. Viruses are obligate, intracellular pathogens that do not replicate outside of their host. Most viruses are fairly host specific, and thus other fishes usually are not important reservoirs for these infections in salmon. Also, many viruses are either mildly pathogenic or not pathogens, and thus the mere presence of a virus does not constitute presence of a disease state.

**Infectious Hematopoietic Necrosis (IHN) virus.** This virus often causes severe, acute systemic disease in fry and juvenile salmonids. In British Columbia, one strain (the U clade) is prevalent in many populations, and has been documented to cause high mortality in sockeye fry in many populations. In the peer-reviewed literature, Traxler and Rankin (1989) documented high mortality in fry in the Weaver Creek spawning channel, with about 50% mortality in a population of about 17 million fish. Adults become infected when they return to freshwater, and serve as a source of the infection to the subsequent generation. Data from DFO collected over the many years has showed a wide variation in prevalence in fry from the Weaver Creek and Nadina River, with levels reaching close to 80% in the 1980s in Weaver Creek and early 2000s in Nadina River (summarized by Traxler 2009; Garver 2010). They also concluded that IHN has not become more prevalent in sockeye salmon in the Fraser system in the last few years. Whereas as the virus isolates in British Columbia all belong to the U clade, there is variability in virulence between isolates (Traxler, G; Garver, K. DFO-PBS, pers. comm. with Kent 6 Dec 2010).

Concerning the marine environment, Traxler et al. (1993) showed that while field observations of clinical disease is confined to fry, experimental exposure of 20 g sockeye salmon in seawater results in low mortality when cohabitated with infected fish. Similar results were found in another study by Traxler and Miller-Saunders (2006), where bath exposure of IHN caused few infections in sockeye salmon smolts. This virus is highly pathogenic to Atlantic salmon in seawater netpens (St-Hilaire et al. 2002; Saksida 2006), and outbreaks occasionally occur in British Columbia. Whereas other fishes, such as Pacific herring, may become infected, salmonids are the apparently the main reservoir for the infections, and Traxler et al. (1997)

detected IHN virus in adult sockeye salmon in seawater. St. Hilaire and colleagues showed that Chinook salmon in seawater may serve as subclinical reservoirs. Indeed, studies by various investigators have all pointed to the conclusion that the source of IHN in seawater netpens is from marine reservoirs (Saksida 2006).

***Risk. High.*** This virus is deadly to fry and juvenile sockeye salmon. Sockeye in seawater are susceptible, but the virus at this stage is less virulent as older and larger fish show fewer mortalities when they become infected. It is conceivable that there are strains within the U clade in British Columbia that would be more pathogenic to sockeye smolts.

**Viral Hemorrhagic Septicemia (VHS) virus.** There are several strains of Viral Hemorrhagic Septicemia virus. The strain found in British Columbia (North American strain) is not highly pathogenic to salmonids, including sockeye salmon (Follett et al. 1997), but can be lethal to Pacific herring, Pacific hake, walleye pollock, and Pacific sardines (Meyers et al. 1990; Hedrick et al. 2004). Another strain of VHS has recently been documented to be lethal to salmonids and other fish species in the Great Lakes (Bowser 2009). The virus was responsible for killing large numbers of freshwater drum (*Aplodinotus grunniens*) and round goby (*Neogobius melanostomus*) in 2005, and was thought to have been disseminated with the movement of culture baitfish. It has not been detected to date in British Columbia. The survey of Kent et al. (1998) revealed no infections by the VHS virus in any ocean-caught salmonids that were examined.

***Risk. Low.*** As VHS (like other RNA viruses) can mutate rapidly it is possible that more lethal strains may be introduced or evolve within years. There is no evidence to date that demonstrates that the lethal Great Lakes strain is in the Province.

**Infectious Pancreatic Necrosis (IPN) virus.** This virus causes a moderate to severe, acute systemic disease in a variety of salmonids. It is common in freshwater hatcheries and marine netpens in Europe in rainbow trout and Atlantic salmon, but has rarely been documented in British Columbia. Most salmonid fishes are susceptible, but no data on sockeye salmon were found. Older literature reports that the sockeye salmon are refractory to experimental exposure (Parisot et al. 1963).

***Risk. Low***. Rare in British Columbia, and not documented in sockeye salmon.

**Viral Erythrocytic Necrosis (VEN) virus.** The VEN virus has been described from various marine fishes, including salmonids in British Columbia (Bell and Traxler 1985). The virus infects red blood cells (erythrocytes), is associated with anemia, and only occurs in seawater.

***Risk. Low***. Severe infections, with numerous infected erythrocytes, would likely cause severe anemia, thus could potentially cause significant disease at the population level if this scenario was prevalent. No data on the occurrence of this virus in wild salmon are available.

**Erythrocytic Inclusion Body Sydrome (EIBS) virus**. Like VEN, EIBS virus infects red blood cells and causes anemia (Piacentini et al 1989). The infection is rather common in coho salmon in freshwater hatcheries, and has been reported in seawater pen-reared fish in other geographic areas. Moreover, it has not been reported in sockeye salmon.

***Risk. Low.*** Not reported in sockeye salmon.

**Putative Novel Virus.** Using microarray technology, Dr. Kristi Miller-Saunders, DFO-PBS and colleagues consistently observed gene expression patterns in the gill tissue of sockeye salmon that was strongly correlated with en route and prespawning mortality (Miller et al. 2011). They concluded that genomic data indicated that a potentially novel disease, possibly caused by a virus, has been affecting a high proportion of juvenile and adult Fraser River sockeye salmon that may weaken fish and directly or indirectly enhance mortality of both smolts and adults. Non-lethal gill samples were collected from sockeye salmon in the ocean, fish were tagged, and then success to spawning was tracked. They found that the unique gene signature was associated with about 13 times greater chance of en route mortality, and almost four times greater chance of dying without spawning once the reach the spawning areas.

In addition to the very recent paper in Science (Miller et al. 2011), Dr. Miller provided a DFO memo that was written in October 2009 (Miller 2009). I also reviewed her Power Point presentation of 07/11/2010 and I met with her on 6 Dec 2010 to discuss this very interesting project. In this PPT presentation, she noted that the gene signatures were seen in smolts affected before leaving natal sites and adults were affected before entering freshwater. Viral screening conducted by Kyle Garver (virologist, DFO-PBS) revealed no viruses, and thus this gene signature does not appear to be the result of a known fish virus that can be cultured. Miller (2009) suggested that the Salmon Leukemia Virus, which causes plasmacytoid leukemia as discussed below, is a possible candidate for this putative virus. This virus has never been cultured.

Similar gene expression patterns were observed in the brains and liver tissues from sockeye salmon collected in 2003, 2005, and 2006 (Miller 2009). Macroscopic changes were observed in the brain of affected fish, including tumor-like growths (Miller 2009), congestion, and possible hemorrhage. However, I reviewed the histological slides from affected fish and found no significant pathological changes. Dr. Gary Marty, BC Ministry of Agriculture, Food and Fisheries, a board-certified veterinary pathologist, also examined slides from affected fish

and found no obvious lesions indicative of a viral infection (K. Miller-Saunders, DFO-PBS, pers. comm.).

This exciting research using functional genomics clearly associates mortality with a unique gene signature. Whereas the pattern of host response suggestive of a virus, this does not prove that a pathogenic virus is present or the cause of high en route or prespawning mortality. Studies are ongoing in Dr. Miller-Saunders' laboratory to resolve the etiology of this interesting phenomenon.

***Risk. Unknown***. An infectious agent associated with this gene signature has yet to be indentified.

**Salmon Leukemia Virus and Plasmacytoid Leukemia (PL).** The histological presentation of this disease is massive infiltration of visceral organs and retrobulbar tissue of the eye by immature lymphocytes or plasmablasts (white blood cells) (Kent et al. 1990). Fish have an enlarged spleen and kidney. The disease causes severe anemia and is usually lethal. It has been most often seen in pen-reared Chinook salmon in British Columbia (Stephen et al. 1996), but has been detected in wild-caught Chinook salmon in the Province and hatchery-reared Chinook salmon in Washington State (Harshbarger 1984; Morrison et al. 1990). Sockeye salmon can be experimentally infected (Newbound and Kent 1991). The cause of PL has been controversial. In the early 1990's various lines of evidence pointed to a retrovirus as the cause (Kent and Dawe 1993; Eaton et al. 1994a,b), which was named the Salmon Leukemia Virus (SLV). The virus was never isolated in culture. In later years, almost all cases that I reviewed from netpen-reared Chinook salmon were associated with infections of the proliferating cells by *Nucleospora salmonis*.

***Risk. Low.*** Not known to naturally occur in sockeye salmon.

## BACTERIA

Bacterial infections of salmon can represent various types of infections. Obligate bacterial pathogens do not proliferate outside of their hosts, and these include *Renibacterium salmoninarum*, *Piscirickettsia salmonis*, and presumably *Aeromonas salmonicida*. These bacteria are usually rather pathogenic and cause disease even in fish that are not compromised by other factors. In contrast, opportunistic bacteria are ubqiutous in the environment and usually cause disease only in hosts that are compromised. This includes surface-infecting *Flavobacterium* spp., but *F. psychrophilum* is capable of causing severe disease in apparently healthy fish.

***Renibacterium salmoninarum*.** This Gram-positive bacterium causes bacterial kidney disease (BKD) in salmonids. It is prevalent in Chinook salmon throughout the Pacific Northwest, including British Columbia. The infection results in acute to chronic, severe systemic disease, and fish die from a few weeks to months following infection. Infections are contracted and spread in both marine and freshwater, apparently by oral – fecal transmission. Also, this bacterium has been shown to be vertically transmitted within eggs (Evelyn et al. 1986). BKD is common in seawater reared Pacific salmon, such as Chinook and coho, but is rare in Atlantic salmon in netpens in British Columbia (Evelyn et al. 1998). Sockeye salmon are highly susceptible. Indeed, review of DFO case reports revealed that this infection was observed at least five times in captive sockeye salmon from a private hatchery from 2006-2008. We found the infection in 25 of 402 sockeye examined from marine waters off British Columbia (Kent et al. 1998).

***Risk. High.*** The bacterium is prevalent in British Columbia, sockeye salmon are particularly susceptible, and the infection progresses after infected smolts migrate to seawater.

***Aeromonas salmonicida.*** All salmonid fishes are susceptible to this bacterial disease, referred to as furunculosis (Hiney and Oliver 1999). The infection can result in an acute, severe disease with high mortality. The infection occurs in both wild and cultured fishes in British Columbia. Review of DFO Case Reports show that it has been detected in a private freshwater hatchery. The bacterium is transmissible in seawater from fish to fish, and is recognized as a potentially serious disease in netpen-reared Atlantic salmon (Evelyn et al. 1998). There are injectable vaccines and antibiotics available to control this bacterial disease. They would be impractical for controlling the infection in wild fish, but might be useful under certain circumstances for larger fish in enhancement observations. The bacterium is transmissible in seawater, both between wild fish and from captive to wild fish.

***Risk. High.*** This bacterium has potential to be lethal to juvenile and adult sockeye salmon in both freshwater and seawater.

***Vibrio (Listonella) anguillarum.*** This is a marine bacterium, and causes vibriosis in unvaccinated smolts shortly after entry to seawater. The bacterium is presumed to be wide spread in marine waters throughout the Pacific Northwest, and was detected in salmon in their first year in seawater (Arkoosh et al. 2004). It has been shown to be lethal to various Pacific salmon species, including sockeye salmon (see review by Actis et al. 1999). The bacterium is a very high risk for unvaccinated smolts shortely after they enter seawater (Actis et al. 1999; Evelyn et al. 1998). It can cause severe disease in seawater pen-reared fish, whereas documentation of mortality in wild salmonids in seawater is less definitive. Infections are exacerbated by chemical pollutants (Arkoosh et al. 2001) or co-infections by parasites (Jacobson et al. 2003).

***Risk. High.*** This bacterium can cause deadly infections in smolts shortly after seawater entry. The bacterium is ubiquitous and thus it is assumed that widespread death associated with this infection requires either debilitated hosts or very high concentrations of the bacterium in seawater.

***Flavobacterium* spp**. Several members of the genus *Flavobacterium* cause disease in fishes in both freshwater and the marine environment (Shotts and Starliper 1995). Most are consider opportunists that only cause significant disease when fish are compromised by suboptimal environmental conditions. However, *Flavobacterium psychrophilum* is considered a primary pathogen. This bacterium causes skin or internal disease, and is referred to as coldwater disease. It is particularly common in coho salmon and rainbow trout in hatcheries (Starliper 2010). To our knowledge, *F. psychrophilium* has not been observed in wild salmonids. The opportunistic bacterium *F. columnarae*, the cause of columnaris, has been associated with mortalities in a variety of wild fish in the Pacific Northwest, and usually associated with high temperatures (Colgrove and Wood 1966; Becker and Fujihara 1978; Wood 1974). Spawning adults are often infected by this opportunist and Colgrove and Wood (1966) reported that the bacterium caused high prespawning mortality in sockeye salmon in the Fraser River some four decades ago. Robins (1964) described the impact of these infections on eggs and fry from pink and sockeye salmon in BC. With the growth of marine aquaculture, diseases caused by marine *Flavobacterium* spp. (now *Tenibacterium* spp.) have been observed. However, they are only recognized as a problem in captive salmonids (Kent and Poppe 1998).

***Risk. Moderate.*** These infections are generally considered to cause disease mostly in hatcheries, but should not be excluded as a cause of disease in wild sockeye if water conditions are poor. There is no evidence that the *Flavobacterium* spp. infections and associated mortality have increased in the Fraser River in recent times.

***Piscirickettsia salmonis.*** This is bacterium is an obligate intracellular, Gram negative pathogen (Fryer and Hedrick 2003; Mauel and Miller 2002). It was first identified about 20 years ago and is recognized as a significant pathogen in fish held in seawater netpens (Kent and Poppe 1998). While it is not found in wild salmon, the pathogen has been detected in open marine waters off Oregon (Mauel and Fryer 2001). It infects macrophages and hepatocytes (liver cells), and causes a chronic disease. To date, it has been documented to cause severe disease in coho, Chinook and Atlantic salmon. As sockeye salmon are not generally reared in netpens, it has not been recognized in this species.

**Risk. Low.** It has not been reported to occur in sockeye salmon and it is probably rare in wild salmon in British Columbia.

## FUNGI AND RELATED ORGANISMS

Several fungal-like organisms are known to cause severe disease in salmonids, including wild salmon. While taxonomists have now removed most of these pathogens from the "true Fungi", fish pathologists and veterinarians still treat them as such, and hence we follow this practice here.

***Saprolegnia* spp.** *Saprolegnia*, referred to as water mold or cotton disease, is a well-recognized pathogen of a wide variety of freshwater fishes. Indeed, the organism occurs in essentially every freshwater body of water. The pathogen infects the skin and gills, and almost always follows damage to these tissues by other causes. Hence, it is considered an opportunist. There are several potential causes of trauma (physical damage) which may cause surface lesions and thus a site for infection in sockeye salmon. For example, to reach spawning grounds sockeye salmon must often negotiate commercial gillnets and hooks from the recreational fishing sector, which may damage skin and result in subsequent *Saprolegnia* infections. The fungus also commonly attacks salmonid eggs.

High mortalities associated with *Saprolegina* spp. have been reported in cultured coho and sockeye salmon in Japan (Hatai and Hoshiai 1993; Hussen and Hatai 1999). With wild salmon, it is one of the most common infections of adult fish once they return to freshwater to spawn, and is associated with skin and gill damage that commonly occurs during the freshwater en route migration to spawning grounds.

***Risk. Moderate.*** This common opportunist is frequently associated with prespawning mortality. Infections are often more common in warmer waters or those with high organic load.

***Ichthyophonus hoferi.*** This internal fungus-like pathogen is a common cause of mortality in herring and some other marine fishes. This is a marine pathogen, but is occasionally found in freshwater fishes. Infections have been reported in freshwater-reared rainbow trout, which were apparently the result of feeding infected marine fish. Salmonids are very susceptible to the infection (Miyazaki and Kubota 1977), and we have observed it in pen-reared Atlantic salmon in British Columbia (Kent and Poppe 1998). We showed that the parasite in Pacific herring was indistinguishable from that found in Pacific salmon (Criscone et al. 2002). Of interest here, Kocan et al. (2004) suggested that *I. hoferi* is an important cause of pre-spawning mortality in Chinook salmon in the Yukon River. Whereas sockeye salmon would certainly be susceptible to the infection, we found only one report of this pathogen in sockeye salmon in the Province, which was characterized by heart lesions in returning sockeye salmon from the Somas River (Tierney and Farrell 2004). Here the infection was associated with impaired swimming ability.

***Risk. Moderate***. Apparently a significant cause of disease in Chinook salmon, but has not been documented to be prevalent in sockeye salmon.

***Sphaerothecum destruens* (rosette agent).** This internal fungus-like parasite is now classified as a choanoflagellate, which are distinct from fungi. It was first observed causing severe disease in pen-reared Chinook in Washington State (Elston et al. 1986). The rosette agent primarily infects macrophages in the spleen and kidney, but it may occur in other organs in heavy infections. Mortality due to the disease is highest in the summer and fall, and losses of over 90% have occurred in some years. We observed a similar parasite in returning sockeye from the Babine system and rarely in pen-reared Chinook salmon.

***Risk. Low.*** Whereas this is a deadly marine pathogen, it appears to be very rare in British Columbia.

***Dermocystidium* spp.** This is another choanoflagellate, but in this case it targets the gills of freshwater fishes. White, macroscopically visible cysts are seen in the gills of returning salmon in freshwater. These infections can cause prespawning mortality when they are severe, and there are a few reports of high mortalities in salmon hatcheries (Olson and Holt 1995). This infection is common throughout the Pacific Northwest, and presumably all salmon are susceptible. There are two "grey literature" reports cited by McDonald and Margolis (1995) documenting the occurrence of this parasite in sockeye salmon in British Columbia.

***Risk. Low.*** The infection is common, but usually only appears to cause mortality in prespawning salmon in suboptimal, warm, small rivers, such as the Elwha River in Washington State.

## PROTOZOA

Numerous protozoa have been reported to infect fishes. Many are merely commensals, and do not cause disease, whereas some are opportunistic and cause disease when fish are compromised by environmental issues. Last, others are recognized as severe pathogens in salmon, particularly those causing internal infections.

***Ichthyophthirius multifiliis*.** This ciliate protozoan (called Ich) is a recognized serious pathogen, infecting a wide variety of freshwater fishes. The parasites cause severe damage to the skin and gills, often killing fish by asphyxiation due to the tissue reaction to the parasite in the gills. Ich induces severe epithelial hyperplasia in the gills and skin, and heavy infections can certainly cause high mortality in salmonids, including wild stocks of sockeye salmon (Traxler et

al. 1998). This paper described the infection in sockeye from the Babine system, but heavy infections in sockeye salmon prespawners from the Nadina River on the Fraser River system have also occurred (Traxler et al.1998). Garver (2010) summarized the findings from the Fraser River. Whereas prespawning mortality as high as 70% has occurred in the Nadina River, it has not increased in severity since 1990.

***Risk. High.*** This ciliate protozoan has already been documented to cause significant prespawning mortality, and severity would certainly increase with increased water temperature and reduced water flows.

***Cryptobia salmositica.*** This blood flagellate is common in salmonids from fresh water throughout the Pacific Northwest where the leech vector (*Piscicola salmositica*) is present. Although the parasite is usually transmitted with leeches, direct fish to fish transmission also occurs when fish are held in crowded culture conditions (Bower and Margolis 1983). In wild fish, the infection is usually seen in sexually-mature salmon that have returned to fresh water to spawn. However, juveniles are also susceptible to the infection, and the parasite can persist in fish after they are transferred to sea water. Infections in wild salmonids, both in adults and juveniles, are often lethal. Deadly infections are occasionally seen in seawater pen-reared Chinook salmon (Kent and Poppe 1998).

Sockeye salmon are susceptible (Bower and Margolis 1984; Bower et al. 1995). There are numerous reports of the infection in sockeye salmon from British Columbia (see summary by McDonald and Margolis 1995), and the infection was detected in 2008 in a private hatchery and was observed associated with prespawning mortality in sockeye salmon at Weaver Creek. (Pacific Biological Station, memo 2009-167). A survey from about 30 years ago showed heavy infections in sockeye from various rivers in the Fraser River system (Bower and Margolis 1984), and there is variability in the susceptibility to infections between strains of sockeye salmon (Bower et al. 1995).

***Risk. Moderate.*** Whereas the pathogen is capable of causing severe disease, we have no reports on the prevalence in Fraser River sockeye salmon. Interestingly, Weaver Creek sockeye are quite resistant to the infection compared to Fulton River stocks (Bower and Margolis 1984).

***Loma salmonae.*** This microsporidium is a well-known pathogen in aquaculture and has caused high mortalities in Japan and North America. The infection targets the gills, and lesions are characterized as lamellar fusion and marked epithelial hyperplasia (Kent and Speare, 2005). Severity of tissue damage is more related to destruction of xenomas (large aggregates of parasites) than merely density of infection (Speare et al., 1998). With infections in freshwater, intact xenomas are associated with little damage. Indeed, Magor (1987) reported minimal gill damage in coho salmon from freshwater, which was consistent with our observations.

Sockeye are susceptible and Shaw et al. (2000) reported the infection in wild sockeye salmon from British Columbia.

***Risk. Low.*** The parasite does not appear to be highly pathogenic in wild salmon

***Nucleospora salmonis.*** This microsporidium infect lymphocytes, and is associated with a disease indistinguishable from plasmacytoid leukemia (see viruses). *Nucleospora salmonis* is an unusual microsporidium in that it infects the nuclei of hemoblasts, particularly lymphoblasts or plasmablasts, in salmonid fishes (Chilmonczyk et al. 1991). This microsporidium was first observed in netpen-reared Chinook salmon in Washington State, where it was associated with anemia (Elston et al. 1987). The parasite has also been reported in freshwater-reared chinook, kokanee, and steelhead trout in Washington (Morrison et al. 1990) and California (Hedrick et al. 1990; 1991). Although we have only seen one case of *N. salmonis* in freshwater-reared salmon in British Columbia, it has been observed in Chinook at several seawater netpen sites in the Province.

***Risk. Low.*** To our knowledge, while sockeye salmon can be experimentally infected with this parasite, no natural infections have been reported in British Columbia.

**Other protozoans.** Numerous other protozoans have been reported from wild salmonids, including sockeye salmon in British Columbia. Several of these parasites have been documented to cause significant disease in the confined environment of netpens (e.g., *Ichthyobodo*, *Spironucelus* spp. (Kent and Poppe 1998) or in hatcheries (e.g., *Spironucleaus salmonis*, and many surface opportunists such as trichodina or *Epistylus* spp.). However, there is no indication to date that these parasites are significant causes of disease in wild salmonids.

## MYXOZOA

The phylum Myxozoa represents well over 1,000 species that are obligate pathogens of fishes. Once included with the Protozoa, they are clearly multicellular, metazoan parasites with complex life cycles that require in invertebrate alternate hosts. Most are only mildly pathogenic, but some, as discussed below, are significant pathogens of salmonids.

***Parvicapsula minibicornis*.** Of all the reported pathogens of sockeye, the most definitive data as it relates to Fraser River sockeye is for *P. minibicornis*. This parasite was originally described from sockeye salmon from Weaver Creek on the Fraser River system by Kent et al. (1997). Recently there have been numerous reports of a high prevalence of the infection in adult sockeye in the Fraser River (Bradford et al. 2010) as well as in outmigrating smolts (St Hilaire et al. 2002). The infection is chronic, and targets the glomeruli of kidneys, which is a very

important structure for filtering blood and maintaining osmoregulation in fish. Wagner et al. (2005) showed that infections reduce swimming ability, and Bradford et al. (2010) showed a correlation of infections with impaired osomoregulation. Bradford et al. (2010) found that the infection was more severe in sockeye adults suffering prespawning mortality compared to successful spawners. As with other myxozoans, the life cycle requires development in an annelid worm. For *P. minibicornis*, this is a freshwater polychaete (*Manayunkia speciosa*). This is the same worm that *Ceratomyxa shasta* uses in its life cycle, and it occurs in the lower reaches of various watersheds throughout the Pacific Northwest.

DFO had an active research program investigating this parasite in sockeye salmon until around 2003/2004. At this time, sea lice became a major concern in the Province, and fish health research efforts were diverted from *P. minibicornis* to study sea lice (Dr. Simon Jones, DFO-PBS, pers. comm. with Kent 14 Oct 2010).

***Risk. High.*** As stated above, this is one of the few pathogens that have been documented to occur in a high prevalence in Fraser River sockeye salmon. However, while it occurs in smolts shortly after seawater entry, it was not detected in older fish in seawater. Therefore, assuming that fish do not spontaneously recover, this myxozoan is linked to parasite-associated mortality in seawater. See discussion above *"Assessing impact of pathogens in wild fish populations"*.

***Ceratomyxa shasta.*** This myxozoan causes an acute and lethal systemtic disease in various salmonids (Stocking et al. 2006; Ray et al. 2010). It has been reported from the Fraser River, but sockeye salmon appear to be one of the most resistant salmonid species. The infection is contracted in freshwater from an infectious stage released by the freshwater polychaete *Manayunkia speciosa*. Margolis and Arthur (1979) and McDonald and Margolis (1995) list no infections by *C. shasta* in sockeye salmon.

***Risk. Low.*** We found no reports of natural infections in sockeye salmon in British Columbia by this myxozoan.

**PKX (*Tetracapusolides bryosalmonae* syn. *T. renicola*)**. This myxozoan causes severe kidney inflammation, the associated disease is called proliferative kidney disease (PKD). The organism is referred to in many older papers as PKX. Infections are directly related to temperature, with clinical disease occurring above 15 $^{o}$C. Most *Oncorhynchus* spp. are susceptible to the infection, and we documented the infection and disease in sockeye (kokanee) exposed at the Puntledge Hatchery on Vancouver Island (Higgins and Kent 1998). Also, I observed severe infections in kokanee from the Puntledge Lake above the hatchery. We showed that the infection persists in Chinook salmon smolts after they migrate to seawater and it was associated with osmoregulatory problems (Kent et al. 1995). ***Risk. Moderate.*** Whereas this

parasite has not been documented in Fraser River sockeye, it is widespread and could cause severe disease if temperatures exceed 15 $^{o}$C. Moreover, the infection persists in smolts after seawater migration. However, fish recover from the infection after a few months, and the parasite usually does not cause direct mortality.

***Myxobolus arcticus.*** Several species of *Myxobolus* infect the brain of salmonids. Whereas *M. cerebralis* is recognized as a severe pathogen of wild salmonids, it has not been reported in British Columbia. Other species, including *M. arcticus*, cause much less tissue damage to the central nervous system, and thus are generally considered not to be pathogenic. However, Moles and Heifetz (1998) demonstrated that sockeye salmon with the infection have reduced swimming ability. *M. arcticus* was detected in some prespawning mortalities from Weaver Creek, along with a variety of other pathogens (see PBS memo 2009-16).

***Risk. Low to Moderate.*** As this parasite is common in sockeye salmon and one report documents impaired swimming, this parasite should be considered as a possible contributing factor to mortality.

**Muscle Myxozoans.** Two myxozoans, *Kudoa thyrsites* and *Henneguya salminicola,* are common in salmonids in British Columbia (Kent et al. 1994). They infect the muscle and reduce the market value of heavily infected fish, but are not associated with significant, if any, mortality.

***Risk. Low.*** Not associated with mortality, but *Henneguya salminicola* is common in the flesh of wild sockeye salmon from certain watersheds.

## HELMINTHS (WORMS)

Wild fishes, including salmonids, are infected with a variety of worms, including monogenes on the skin and gills, and internal nematodes, tapeworms, and trematodes. Indeed, wild salmonids often harbor what appear to be heavy infections, without obvious clinical changes. Synopses of helminth parasites in sockeye salmon (Margolis and Arthur 1979; McDonald and Margolis 1995; Love and Moser 1983) list some 50 helminth parasites infecting sockeye salmon in the Pacific Northwest. However, only a few of them have been implicated to cause significant impacts in wild salmon**.**

***Eubothrium* spp.** The adult stages of these tapeworms are found in the intestine and stomach of juvenile sockeye salmon. Heavy infections may reduce swimming stamina, growth, and survival (Boyce 1979), saltwater adaptation (Boyce and Clarke 1983), and may alter migration orientation (Garnick and Margolis 1990).

***Risk. Moderate.*** These infections would only be of concern with moderate to severe infections.

***Diphyllobothrium dendriticum* and *Proteocephalus* sp. (Cestoda) *Philonema oncorhynchi* (Nematoda).** There is one report (Garnick and Margolis 1990) that documents a potential impact of these helminths on sockeye salmon. These authors showed a statistically significant association with these infections with orientation of smolts, which could impact smolt migration.

***Risk. Low.*** These parasites are probably only a concern if infections are heavy.

## PARASITIC CRUSTACEANS

About five species of marine or freshwater parasitic copepods have been described from salmon in the British Columbia (see monographs by Margolis and Arthur 1979; McDondald and Margolis 1995; Kabata 1988). Here we review the species that would most likely be considered as possible causes of significant mortality in sockeye salmon in BC, particularly Fraser River stocks; caligid copepeods (sea lice) *Lepeophtheirus salmonis* and *Caligus clemensi.* and *Salminicola californiensis*.

**Caligid copepods.** *Lepeophtheirus salmonis* is a marine parasitic copepod, and has been reported from all *Oncorhynchus* spp. from the ocean for many years. Johnson et al. (1996) documented mortality in adult salmon in Alberni Inlet returning to spawn associated with this parasite. Numerous recent articles claim that *L. salmonis* causes significant mortality in pink and chum salmon in British Columbia, particularly associated with fish farms (Krkosek et al. 2006, 2007, 2010; Morton and Williams 2003; Morton et al. 2004). This is a controversial issue, and other research has failed to support the claims of both significant mortality due to this parasite in wild salmonids and fish farms being the major source of such infections (Beamish et al. 2005; 2006; Brooks and Stuchi 2006). A recent paper found an association between sea lice on farms and wild juvenile salmon, but not a negative association with overall survival of the latter (Marty et al. 2010). The role of fish farms as a source of sea lice is also addressed in the report on impacts of fish farming (Project 5. 2011. Salmon farm impacts on Fraser sockeye salmon. *In prep.* Cohen Commission Tech. Rep. 5). Jones et al (2008) concluded based on experimental

studies with pink salmon that elevated risk associated with *L. salmonis* infection occur only in fish less than 0.7 g. In contrast, sockeye salmon smolts are much larger when they enter seawater. Hence, there are reports of *L. salmoni*s infections on sockeye salmon, but there is not direct indication that the parasite causes significant mortality in this species. Ocean surveys conducted in May and June 2010 revealed a prevalence of only about 4% *L. salmonis* with abundance at less than one louse/fish.

*Caligus clemensi* is another caligid copepod that infects both farmed and wild salmon in British Columbia. It is smaller than *L. salmonis*, and on a per parasite basis is considered less pathogenic (Boxshall and Defaye 2006). Unlike *L. salmonis*, this copepod infects many different marine fish species. Ocean surveys conducted in May and June 2010 by Dr. Simon Jones showed that over 70% of the sockeye salmon were infected, with a range of 1-16 lice/fish.

Another concern with parasitic copepods, such as *L. salmonis*, is that they are potentially vectors for other pathogens (Nese and Enger 1993; Barker et al. 2009). *Caligus* spp. are capable of moving from host to host, and recently Connors et al. (2008) showed that *L. salmonis* may move to predator salmon when infect prey are eaten.

***Risk. Moderate.*** There are several claims of significant mortality in pink salmon in British Columbia due to sea lice, but none have been documented in sockeye salmon. As *C. clemensi* is prevalent on sockeye salmon smolts, it should be considered as a candidate for parasite associated mortality in these fish.

***Salminicola californiensis.*** This copepod, referred to as gill maggots, is common on adult salmon. To our knowledge, it has only caused significant disease in wild salmon that are captured and held in captivity as part of captive brood programs.

**Risk. Low**. This parasite appears to cause significant disease only when fish are held in captivity.

Table 1. Summary of pathogens and parasites in sockeye salmon in Pacific Northwest,. Life stage: J = juvenile, A = Adult. Geographic Location: BC = British Columbia, FR = Fraser River. PNW – Pacific Northwest. In culture or wild: NP = Seawater netpens, HA = freshwater hatchery or spawning channel, W = wild.

| Pathogen or Disease | Life Stage | Risk | Location | Fresh (FW) or Marine (M) | Culture/Wild | Hosts |
|---|---|---|---|---|---|---|
| **Viruses** | | | | | | |
| IHN virus, infectious hematopoetic necrosis | Fry | High | PNW, BC, FR | FW, M | HA, NP, W | Salmonids, herring, etc |
| VHS, Viral hemorrhagic septicemia | A | Low | PNW,BC | FW, M, M only in BC | HA, W | Salmonids, other fishes |
| IPN virus, Infectious pancreatic necrosis | Fry, J | Low | PNW, BC, FR? | FW, M | HA, W | Salmonids, No data on sockeye |
| EIBS virus, erthyrocytic inclusion body syndrome | J | Low | BC, FR? | FW, M | HA, | Salmonids, severe in coho, no data on sockeye |
| VEN virus, viral erythrocytic necrosis | J | Low | PNF, BC | M | NP, W | Salmonids, other marine fishes |
| **Unknown Etiology/Putative Viral** | | | | | | |
| SLV virus, plasmacytoid leukemia | J,A | Low | PNW | FW, M | H. NP, W | Chinook, Sockeye experimental |
| Miller Microarray Agent | J, A | Unknown | BC, FR | FW, M | F, M | Sockeye |
| **Bacteria** | | | | | | |
| *Vibrio* (*Listonella*) *Anguillarum, vibriosis* | J, A | High | BC, FR | M | W, NP | Salmonids, other marine |
| *Aeromonas salmonicida,* furunculosis | J, A | High, | BC, FR | FW, M | C, W? | All salmonids and other fishes |
| *Flavobacterium,* coldwater disease, etc. | Fry, J, A | Moderate | BC, FR? | FW | C, W | All salmonids |
| *Flavobacterium* spp. marine | J, A | Moderate | BC | M | NP | Salmonids, others |
| *Piscirickettsia salmonis,* Salmonid rickettisal septicemia | J, A | Low | BC, | M | NP, W? | Salmonids, rarely other fishes |
| *Renibacterium salmoninarium,* bacterial kidney disease | J, A | High | BC, FR | FW, M | H, NP, Wild | All salmonids |

| Pathogen & Disease | Life Stage | Risk | Location | Fresh (FW) or Marine (M) | Culture/Wild | Hosts |
|---|---|---|---|---|---|---|
| **Fungal and Related Organisms** | | | | | | |
| *Saprolegnia* | F, J | Moderate | BC, FR | FW | H, W | Salmonids, other fish |
| *Ichthyophonus hoferi* | A | Moderate | BC | FW, M | W, NP | Salmonids, marine fish |
| *Sphaerothecum destruens*, | J, A | Low | BC (Babine) | F, M | W, NP | Salmonids |
| *Dermocysitidium salmonis,* gill disease | J, A | Low | BC | F | C, W | Salmonids |
| **Protozoa** | | | | | | |
| *Loma salmonae, gill microsporidiosis* | J, A | Low | BC, FR? | F, M | H, NP, W | Salmonids, sockeye |
| *Nucleospora salmonis* Leucocyte prolferiation/plasmacytoid leukeima | J, A | Low | BC, FR? | F, M | C/W/NP | Salmonids Lab infections with sockeye |
| *Ichthyophthirius multifiliis, Ich or White spot* | A | High | BC (Babine) | F, | Spawn Channel | Salmonids, Many others |
| *Cryptobia salmonicida* | J ?, A | Severe | BC | F, M, | H, NP, | Salmonids, sockeye |
| Trichophyra, and other gill protozoa | J, A | Mild | BC | F | H, NP, W | Salmonids |
| **Myxozoans** | | | | | | |
| *Parvicapsula minibicornis,* | J, A | High | BC | F, M | W | Chinook, sockeye |
| *Myxobolus arcticus, brain myxobolosis* | J, A | Low | BC | F, M | W | Salmonids, sockeye |
| *Ceratomyxa shasta* | J, A | Low | BC, FR | F, M | H, W | Salmominds |
| *Tetracapusolides bryosalmonae* Proliferative kidney disease | J | Moderate | BC, FR? | F, M | H, W | Salmonids, sockeye |
| Muscle Myxozoans | A | Low | BC, FR | FW, M | NP, W | Salmonids, sockeye |
| **Worms and Copepods** | | | | | | |
| *Eubothrium spp.* | A | Moderate | BC | FW, M | W | Salmonids, sockeye |
| *Lepeophtheirus salmonis* | J, A | Moderate | BC | M | NP, W | Salmonids, sockeye |
| *Caligus clemensi* | J, A | Low | BC | M | NP, W | Salmonids, sockeye |
| *Salminicola californiensis* | A | Low | BC, FR | FW | H, W | Salmonids, sockeye |

# ENVIRONMENTAL FACTORS AND INFECTIOUS DISEASES IN SALMON

Fish are very closely tied to their environment, and thus water quality and other environmental parameters play a very important role in their susceptibility to disease (Snieszko 1974). First, particularly in a confined environment, fish drink and eat in the same water in which they urinate and defecate. Hence, pathogens can easily be transmitted amongst fish in the water environment, and the degree of transmission is greatly influenced by density of fish in water. This might not be considered an important concern in the ocean or within large rivers, but can come into play in small rivers, and spawning channels, and thus infectious agents are particularly transmitted from fish to fish before and during spawning, or as fry. As fish such as salmon aggregate in schools, direct transmission of pathogens from fish to fish may even occur when fish are in the open ocean (Dobson and May 1987).

**Climate change and temperature**. Fish are cold-blooded (poikilothermic) and thus both pathogens and host are extremely influenced by temperature. There have been numerous models projecting spread and increase of terrestrial infectious disease with climate change, and these extend to the aquatic environment. High water temperature also has been documented to dramatically increase the replication rate of parasites. For example, temperature-mediated changes in cercarial output from snails had a high as 200-fold increase in response to a 10 $^{o}$C rise in temperature (Poulin 2006). Moreover, the seasonality of most salmon pathogens would likely broaden as water temperatures increase (Marcogliese 2001). However, free-living stages of pathogens that are not feeding in the environment may have shorter life spans in the environment as temperatures increase (see review by Lafferty 2009).

Increase in water temperature often causes stress in fish (Marcogliese 2001) and reduces their general immune status (Bowden 2008). This leads to increased susceptibility to disease. This would particularly relate to salmonids in freshwater. Temperatures approaching the upper tolerance limits for salmonids are stressful and can reduce the immunological capability of fish, rendering them more susceptible to pathogens and disease and possibly predation**.** Thus it is logical that most pathogens in salmon are affected by temperature, and this phenomenon has been recognized for many years (see early review by Roberts 1975). However, empirical studies are somewhat limited, but there have been some conducted on the pathogens of concern for this report. These include *Cryptobia* (Bower and Margolis 1985), the PKX myxozoan (Clifton-Hadley et al. 1986), and the IHN virus (Hetrick et al. 1979). *Parvicapsula minibicornis*, a high risk pathogen, uses the same annelid host (*Manayunkia speciosa*) as *Ceratomyxa shasta*. Increases of this worm and *C. shasta* are associated with anthropogenic changes – e.g., dams,

diversion of water for irrigation, and ultimately warm summer water temperatures (Bartholomew 2009).

**Pollution.** Lafferty (1997) and others have reviewed, documented, and hypothesized on effects of pollution on infectious diseases in the aquatic environment. Like temperature changes, pollution can cause reduction in the immune competence of the fish host. Also, increases in eutrophication will cause increase in invertebrate vectors, such as snails and oligochaete worms. Eutrophication is the addition of substances, such as nitrates and phosphates, through fertilizers or sewage, to a fresh water system. Organic load in water is well recognized to influence fish pathogens, particularly opportunists like gill ciliates and *Saprolegnia* fungus. However, extreme contamination may actually reduce infections by parasites requiring intermediate hosts because certain chemicals are extremely toxic to invertebrates. Indeed, some highly polluted areas actually show reduced parasite fauna (Lafferty 1997).

**Land use practices.** Several studies have shown the effects of land use practices on aquatic habitat (Allan 2004; Brown et al. 2005; Johnson et al. 2007), and environmental change has often paralleled the decline in salmon populations. Progressively more land is being altered, and several land use practices (i.e. deforestation, urbanization, and farming) have been linked to direct loss of habitat for salmonid species (Lackey et al. 2006). Anthropogenic changes to landscape and climate may alter complex assemblages of organisms and favor the proliferation of pathogens (Paul and Meyer 2001). For example, removal of riparian vegetation and increased nutrient loads to stream systems promote higher water temperatures and eutrophication, respectively. Changes resulting in eutrophication, increased sediments, increased temperatures, and reduced flows have been associated with dramatic increases in both snails and oligochaetes (Marcogliese 2001; Cairns et al 2005). Several of the digenean or myxozoan parasites of sockeye salmon use either aquatic snails or oligochaete worms as intermediate hosts. Changes in land use and water flow may therefore indirectly increase the level of these parasites. Moreover, one of the most dramatic effects of land use activity is on water temperature, which can profoundly affect infection dynamics as discussed above. Land use changes may also increase the density of fish eating birds that frequent salmonid streams (Bryce et al. 2002). These birds are definitive hosts for trematodes, and thus increased bird predation could be linked to increased parasites in snails and fish.

## STATE OF THE SCIENCE

Numerous pathogens have been reported in sockeye salmon, but only a few of them have been documented to be real (or likely) causes of significant mortality in this fish in the Fraser River system. The state of the science for understanding the impacts of pathogens on wild salmon in British Columbia is minimal, particularly compared to our understanding of diseases in salmon and trout in public or private aquaculture. This is because, as with most other government supported fish health programs, the majority of research efforts in British Columbia have been directed towards the study of diseases afflicting fish reared in hatcheries or in netpens. Declines in Fraser River sockeye salmon after seawater entry are of most concern (Peterman et al. 2010). It is also difficult to study the impacts of diseases on wild fishes, particularly in the marine environment. It should be noted that Peterman et al. (2010) also concluded that en route and pre-spawn mortality in adult sockeye are significant factors that reduce the number of effective female spawners, and thus may pose a threat to the long-term viability of the populations that are particularly affected.

The effects of pathogens on wild fish populations are challenging to elucidate because moribund fish are usually undetected in vast bodies of waters or are selectively removed by predators. However, there are various well-accepted approaches that have been used to evaluate impacts of diseases in wild animal populations, including fishes. These approaches require evaluation of both prevalence and severity of infection in large numbers of samples. In recent years, this type of research has not been well supported as it is considered by some funding agencies to be merely survey work and not hypothesis driven. These types of investigations have not been applied to Fraser River sockeye salmon, but there are a few scientific reports that have documented outbreaks of infectious disease in sockeye salmon in British Columbia, such as those caused by sea lice and the Ich protozoan. In addition, several researchers in recent years have shown that the myxozoan *Parvicapsula minibicornis* is prevalent in sockeye salmon in the Fraser River system, and indirect evidence suggests that it may impact survival.

Review of data provided by Fisheries Oceans Canada (DFO) showed that, regarding sockeye from the Fraser system, the vast majority of investigations were conducted on fish in freshwater and we found very few investigations on sockeye salmon after they enter the ocean. Of the some 100 case investigations conducted by DFO on sockeye salmon from the Fraser River system that were reviewed, only five were designated “wild loses”. As there is particular concern about marine survival, we specifically recommend that efforts be made to investigate pathogens and their role in survival in this life cycle phase of sockeye salmon from the Fraser system and other regions of British Columbia.

For the present review, a subjective assessment of risks of causing significant disease in wild sockeye salmon in the Fraser River system was provided for each pathogen. The following pathogens were considered to be potential "High Risk" to Fraser River sockeye salmon: the IHN virus, three bacteria (*Vibrio anguillarum*, *Aeromonas salmonicida*, *Renibacterium salmoninarum*), and two parasites (Ich - *Ichthyophtheirus multifillis* and the myxozoan *Parvicapsula minibicornis*). All of these pathogens are endemic to British Columbia and most likely have been present in this area for centuries. Hence, if there has been a dramatic increase in mortality caused by one or more of them in recent years, it is likely due to changes in the susceptibility of sockeye salmon to them or to a change in the abundance of these pathogens. Environmental changes could be an underlying cause of either, as fish are so closely tied to their environment. In agreement with the Peterman et al.(2010), we cannot conclude that a specific pathogen is the major cause of demise to the Fraser River sockeye salmon. However, pathogens cannot be excluded at this time as adequate research on the impacts of disease on this population has not been conducted.

## RECOMMENDATIONS

There are certainly many pathogens that could cause significant mortality in wild sockeye. As stated above, documenting the role of these pathogens in wild fish, particularly with salmonids, requires a significant amount of research effort.

**1) Multiple Year Surveys.** Surveys must be conducted and maintained over several years to provide the needed raw data. Surveys must include proper identification of pathogens, geographic and host distribution, and data on abundance or severity of infection.

**2) Data analyses.** With data from surveys, researchers could conduct the appropriate analyses to infer or document the role that these pathogens have with survival in various life stages. Suggestions for appropriate methods are outline above, including retrospective evaluations to determine thresholds for pathogen associated mortality, as first described by Crofton (1971).

**3) Environmental Factors.** After a pathogen is shown to be associated with mortality, modelers, mathematicians, statisticians, and ecologists could then conduct investigations to elucidate which factors (e.g., water temperature, river flow, land use practices, netpen farming) influence the distribution and abundance of these pathogens.

**4) Diagnostic Methods.** Appropriate diagnostic methods for specific pathogens should be used. Inclusion of histological analysis is recommended as it is the most appropriate diagnostic method for screening fishes for underlying pathological changes and unknown diseases

(including those caused by non-infectious agents). For example, Dr. Simon Jones (DFO-PBS) has recently detected liver lesions consistent with netpen liver disease in sockeye salmon in marine waters off British Columbia (pers. comm. with Kent 6 Dec 2010). This disease is apparently caused by microcystin toxin, probably from cyanobacteria naturally occurring in marine waters (Kent et al. 1990; Williams et al. 1998) and has been observed in wild Chinook salmon in BC (Stephen et al. 1993). Liver lesions associated with this condition are often severe and certainly compromise the health of salmon. Last, frozen tissues should be archived for further evaluation by PCR and other methods as warranted.

Along the lines of this recommendation, Dr. Stewart Johnson, Head, Fish Health Section at DFO at the Pacific Biological Station has recently initiated a health survey of marine sockeye salmon in British Columbia, which includes both sea lice counts and general histopathology (S. Johnson, DFO-PBS pers. comm., with Kent, 6 Dec 2010).

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## APPENDIX 1 – Statement of Work

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

### "Effects of Diseases and Parasites on Fraser River Sockeye Salmon"

**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.

**SW2 Objective**

2.1 A Veterinary scientist to prepare a technical report evaluating the documented and potential effects of parasites and diseases on Fraser River sockeye salmon and their role in the 2009 run failure.

**SW3 Scope of Work**

3.1 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.

3.2 The role and impact of parasites and diseases caused by other agents on the overall mortality schedule of Fraser sockeye salmon will be evaluated both qualitatively and quantitatively by review and analysis of fish disease data, the peer-reviewed literature and available government documents. Where data are unavailable, an assessment of potential pathogen impacts will be based on the available literature.

**SW4 Deliverables**

4.1 The Contractor will organize a Project Review meeting to be held within 6 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 2 reports evaluating the effects of diseases and parasites on Fraser river sockeye: 1) a progress report, and 2) a final report. The style for the Reports will be a hybrid between a scientific style and a policy document. An example of a document which follows this format is the BC Pacific Salmon Forum Final Report (www.pacificsalmonforum.ca).

4.3 A Progress Report (maximum 20 pages) will be provided to the Cohen Commission in pdf and Word formats by Nov. 1, 2010. Comments on the Progress Report will be returned to the contractor by Nov. 15, 2010.

4.4 A draft Final Report will be provided to the Cohen Commission in pdf and Word formats by Dec. 15, 2010. 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 Jan. 15, 2011 with revisions due by Jan. 31, 2011.

4.5 The Contractor will make themself available to Commission Counsel during hearing preparation and may be called as a witness.

4.6 The Contractor will participate in a 2-day scientific workshop on November 30 - December, 2010 with the Scientific Advisory Panel and other Contractors preparing Cohen Commission Technical Reports to address cumulative effects and to initiate discussions about the possible causes of the decline and of the 2009 run failure.

4.7 The Contractor will participate in a 2-day meeting presenting to and engaging with the Participants and the public on the results of the diseases and parasite investigations on February 23-24, 2010.

Report Title: Infectious Diseases & Potential Impacts on survival of Fraser River Sockeye salmon

**Reviewer 1: Sonja Saksida**

Date: Jan 5, 2011

**1. Identify the strengths and weaknesses of this report.**

**General Response to Reviewer - This was a thorough review, including statements on strengths and very helpful suggestions regarding weaknesses. I have complied with most of the suggestions unless otherwise noted. Responses are in Times Roman and Bold.**

**Strengths**

- The author is very familiar with the common infections/diseases that occur in Pacific salmon and addressed the significant gaps in peer reviewed data by interviewing the key DFO personnel who conduct fish health research.
- The report was concise. The rating that Dr Kent provided for the various infectious disease agents appears objective and accurate.
- The environmental factors and infectious diseases discussion is good as is the discussion of the assessment of pathogen-associated mortality in wild salmonids.
- Recommendations provided to the commission are appropriate; however, it would have been good to set a period of time (e.g. minimum of 10 -15 years) for data to be collected and the program to be re-evaluated. Open ended recommendations tend not to be followed up on.

**Weaknesses**

- The document's organization did not flow well and made reading difficult. For example, the Executive summary should be in the front of the document
- 1a. and needs to include recommendations
- 1a, I added the "Recommendations" to the Executive Summary, but in a shorter form without citations as found in the TOC
- 
- 1b. and 'State of the Science' information. 1b. **State of the Science was included in the Executive Summary in the first draft, but was expanded in the revised version.**
- 
- 1c. The document needs to provide more background information in order to inform an audience that is not familiar with either fish health or the sockeye salmon issue being considered.

- **1c Following discussion from Dr. Levy, background was not expanded as this is covered in other sections in the overall report.**

**Specific recommendations to improve the document**

A. More comprehensive introduction which includes:

1. Issue Background – that is, decline of sockeye salmon (in general over the last 20 years), the unexpected low returns in 2009 and equally important the extremely high returns in 2010. Perhaps provide a figure illustrating the decline. The author needs to discuss why infectious disease is being considered as a possible cause. N**ot included as this is part of other reports**
2. "It may be helpful to provide examples of where infectious disease have or have not been attributed to large scale mortality in other animal species (i.e. VHS in Pilchard in BC or perhaps VHS in the Great Lakes- there are probably better terrestrial examples)". **Done**
3. Scope of this document - examine affects of disease on the sockeye salmon The author should provide a statement that emphasis will be on infectious agents with a limited discussion on non-infectious diseases (Idiopathic diseases). **Done**
4. Methodology/ activities - included peer-reviewed and gray (not reviewed, unpublished) literature. Names of individuals who were interviewed need to be provided.
5. The document examines diseases affecting Pacific Salmon –author needs to discuss the shortfalls of this method since not all salmon react the same way to infectious agents.
   **A statement regarding variation in host susceptibility was added.**
6. Author needs to discuss limitations faced - lack of data for sockeye specifically and particularly when at sea. It would be appropriate to include the example described on page 23 (lns 10-15). Also may want to discuss that much of the available gray data from DFO is from screening of enhanced populations (SEP hatchery data or enhanced systems) – if this is the case, what is the consequence/limitations?
7. Add a section introducing disease which could include
   a. What is disease (introduce and define acute and chronic)
      I included a statement defining acute versus chronic.
   **b.** Define and discuss morbidity, mortality – **I have addressed this – changed to sickness or death in Executive Summary**
   c. With mortality discuss how it can be directly related to the disease or indirectly (i.e. decrease thriftiness of the animal makes it more conducive to succumbing to secondary infections or predation)
   d. What is an infectious agent? - discuss how the presence of an

infectious agent does not necessarily equate to disease. Include the discussion of binomial distribution of pathogens (include a figure illustrating this).

e. MOVE the section entitled ENVIRONMENTAL FACTORS SECTION INTO THE INTRODUCTION. The author already provides a good discussion of the interplay be between disease and the environment. I think this section should be before the discussion of the specific agents.
f. Next section can be the one in your original document

B. The comprehensive introduction described above can be followed by information on specific infectious agents. For each, there should be a small introduction as to what the agent class is - i.e. what is a virus. This doesn't have to be long, but would be useful.

C. There should be more discussion of diseases where there is data available - i.e. IHN. IHNv has been monitored in several runs of sockeye salmon it would be useful to provide a figure illustrating the variation that has been reported in the last decade - (Garth Traxler's work). Also, it may be appropriate to include Meyer et al. 2003, J Aquat Animal Health (15:21-30).

For other agents include signs, effects mortality (acute vs chronic) versus morbidity – if known. If not, that needs to be stated too.

It would be useful to provide a summary tables and figures (for example, showing the relative ranking of the agents).

D. Summary Section needs to be added
This is where the author discusses his opinion on the likelihood that infectious disease resulted in the poor sockeye salmon returns in 2009 or if disease played a similar role in the Fraser sockeye declines seen over the last 20 years. The author can state the limitation in making an unequivocal conclusion one way or another and why.
As well, this section could include the section entitled "An assessment of pathogen-associated mortality in wild salmonids - the author provided a good section describing different methodologies. This would be followed by the author's recommendations.

E. The author should review the reference section closely as there is inconsistency in formatting as well as several errors. I listed some of the issues below in the last section of the review but not all.

Overall comments – I would suggest that the author looks at the structure and layout used in the Predation Report and set up their document in a similar way.

**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?**

Available data on disease in sockeye salmon, particularly during the marine phase is scant and this was well demonstrated in this document. This report does represent the best scientific interpretation of the available data.

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

There has been so little done on the area of health monitoring on wild fishes. This report demonstrates the total lack of coordinated fish health surveillance programs. Even so, it may have been useful to provide examples where data has been reported on - i.e. in Alaska (Meyer et al. 2003, J Aquatic Animal Health 15:21-30) where IHNv in Sockeye was reported on for a number of years.

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

I agree with the author about the need for coordinated fish surveillance programs with dedicated funding that ensures that money is not diverted from these programs. I also agree that such a program needs to assess health (which should include histopathological examination) and not just be an exercise in 'bug hunting'. As the sockeye lifecycle is four years, a minimum time of 10-15 years is suggested. This would place the onus on DFO to follow through on this program and at the end of the period evaluate the value of it and determine whether to continue with it.

Also, better screening, monitoring programs for enhanced populations (SEP hatchery reared) need to be considered as they may be a good proxy for the wild runs. Finally, it may be useful to improve fish health at the SEP facilities by incorporating standard health tools such as vaccination prior to release (where applicable) to reduce the chance that these populations will become a source of infection to the wild populations.

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

Long term monitoring programs with dedicated funding for selected populations.

**6. Please provide any specific comments for the authors.**

See response to question 1 for suggestions on reorganizing the document. General comment - scientific names should be provided when a fish species is first discussed.

Virus section - provide an introduction of what is a virus. Are there any viruses that infect fish but are not considered disease causing – i.e. commensal or mutualistic versus parasitic or pathogenic?

Pg 3 Ln 27 - become infected? Should this be diseased not infected?

6 a, Pg 3 Ln 29-35 - could a figure be provided to show changes in prevalence in these populations?
**6a.This would require obtaining a copy right or permission from a journal or author and time restraints do not permit this.**

Pg 4 Ln 4 - add reference - Saksida S. (2006) The 2001-2003 infectious hematopoietic necrosis (IHN) epidemic in farmed Atlantic salmon (Salmo salar L.) in British Columbia, Canada. Diseases of Aquatic Organisms 72:213-223.

Pg 4 Ln 9 - replace this reference with the one above.

6B. Pg 4 Ln 18 - herring (provide scientific name). Add Pilchard (Pacific Sardine)
**6b 1 also added additional references.**

Pg5 Ln 22 - add Dr (Kyle Garver)

Pg5 Ln 13-29 - would it be appropriate here or elsewhere to discuss an example where an infection may not actually be a disease? Also the risk of interpretation of results from novel techniques (i.e. micro-arrays)?
**6c. I included a statement in the introduction on the virus section that mere presence of a virus does not indicate disease.**

6d. Pg 5-6 Ln 34-6 - Plasmacytoid leukemia (marine anemia) this could be moved to parasite section.

**6d. This was maintained with the virus section as the first pathogen associated with this condition was the SLV virus.**

Bacteria - intro similar to virus section

Pg 6 Ln 18-19 - expand on this point. Provide summary of the data on BKD in sockeye salmon

Pg6 Ln 32 - would it be appropriate for enhanced populations?

6e Pg6 Ln 38 - genus name changed to *Listinella*

**6e I believe it has been changed back to *Vibrio*. I have included genus names "Listonella anguillarum should revert to their previous placement in Vibrio" Dikow Cladistics 27 (2011) 9–28.**

Pg 7 Ln 37-38 - add space

Pg 9 Ln 4-5. Dr Gary Marty of BC MAL diagnosed Ichthyophonus hoferi in 1 sockeye salmon sampled Feb 05 (in heart and kidney) in the Broughton Archipelago.

**I do not have this reference and hence I had to omit this.**

Protozoa - intro similar to bacteria

Pg 15 Ln 2-7. Should add reference: Marty, G.D., Saksida, S.M., and Quinn II, T.J. 2010. Relationship of farm salmon, sea lice, and wild salmon populations. Proc. Natl. Acad. Sci. U. S. A. **107**(52): 22599–22604. doi:10.1073/pnas.1009573108. This paper finds an association between lice on farms and wild juvenile but no negative association with overall survival of the population. This adds to the complexness of presents of a parasite does not necessarily result in disease.

6g. Pg 16 Ln 15 - should read 2010 not 20101. I think you also mean 4% L.salmonis with an intensity of 1 louse/fish. Cannot have an intensity of less than 1.

**6g. I clarified this, adding "abundance". Intensity could not be less than 1, but abundance can as abundance includes uninfected animals.**

Pg 16 Lln 18-22. This louse species has a broad host range.

Pg16 Ln 9 - add 1) those that cause . . .

Pg 16 Ln 36 - should this read second not third? It would also be appropriate to provide a figure showing what a negative binomial looks like.

Pg 17 Ln 13 - add (prevalence) and intensity

Pg 18 Ln 3 - would transmission as a concern be greater where salmon

congregate to feed (i.e. Pacific Gyre) in the marine environment? What if animals travel in large schools?

Pg 20 Ln 2 - *Listenella* not *Vibrio*

*6h.* Pg 20 Ln 30-33 - why is this included in the executive summary if there is no indication of disease associated with the signature? Maybe there is a need to get a better understanding of what novel techniques are indicating.
**6h. I included this in the summary as there is great interest in this phenomenon, but I agree that at this point an infectious agent has not been verified. Moreover, I was specifically directed to include a review of this topic in my report**

Pg 22 Ln 20 - delete 'a' before agreement

Pg 22 Ln 22 - delete 'this' before pathogens

References in general need to be made consistent in formatting - journals abbreviated or not. Periods after initials. Please note not all the formatting errors are outlined in the following comments.

Pg 24 Ln 15 - incorrect authorship order - should be Mary R. Arkoosh, Ethan Clemons, Paul Huffman, Anna N. Kagley, Edmundo Casillas, Nick Adams, Herb R. Sanborn, Tracy K. Collier, John E. Stein

Pg 24 Ln 21 - period missing after E and between LL and TK

Pg 24 Ln 25 - Harris, P.D. ?

Pg 24 Ln 32 - Bartholomew, J. 2009. Long-Term fish disease monitoring program in the Lower Klamath River Annual Report. Funding: Bureau of Reclamation Investigator: Jerri Bartholomew, Department of Microbiology, Oregon State University. ?

Pg 25 Ln 10 - correct page numbers 25:373-383.

Pg 25 Ln 36 - correct journal - Can. J. Fish. Aquat. Sci. **40**(6): 821–824 (1983)

Pg 26 Ln13 - authorship format needs to be the same as the others.

Pg 26 Ln 17 - journal abbreviation J. Environ. Manage.

Pg 26 Ln 28 omit Vol.

Pg 27 Ln 2-3 - add space between citations.

Pg 28 Ln 31 - volume 354 not 435

Pg 29 Ln 4-5 - add space between citations

Pg 29 Ln 12 - Ketcheson not Ketchenson

Pg 30 Ln 9 - vol 30 not 20

Pg 30 Ln 12 - omit 2004

Pg30 Ln 34 - missing date. Remove date from the next line

Pg 31 Ln 7 - remove k from end of aquat

Pg 31 Ln 19 - complete title A microsporidium-induced lymphoblastosis in chinook salmon *Oncorhynchus tshawytscha* in freshwater

Pg 32 Ln 21 - replace with this citation Saksida S. (2006) The 2001-2003 infectious hematopoietic necrosis (IHN) epidemic in farmed Atlantic salmon (*Salmo salar L.)* in British Columbia, Canada. Diseases of Aquatic Organisms 72:213-223.

Pg 21 Ln 34 - correct spelling Snieszko

Pg33 Ln 6 **-** Chartrand, S. Not Chartr

Pg 33 Ln 6-9 - title of reference- Infectious hematopoietic necrosis virus antibody profiles in naturally and experimentally infected Atlantic salmon (*Salmo salar*)

Pg 33 Ln 10 - 15 - move references to before first St Hilaire.

Pg 33 Ln 19 - should be Aquaculture 212:49-67

Pg 33 Ln 25 - Hilaire not Hiliare

Pg 33 Ln 26 - remove &

Pg 33 Ln 31 - remove 2006

Pg 33 Ln 37 - Traxler not Traxker

Report Title: Infectious Diseases and Potential Impacts on Survival of Fraser Sockeye

**Reviewer 2**: Dr. Steven J. Cooke

Date: January 6, 2011

**1. Identify the strengths and weaknesses of this report.**

**General Response to Reviewer 2. The charge of item 1 was to identify both strengths and weakness. This reviewer provides extensive criticisms, but no comments on strengths. Nevertheless, the review provided some very useful suggestions, which were incorporated in the revised report. I did not comply with requests to greatly expand the recommendations as this goes beyond the scope of work and allocated funds for generation of this report.**

**If no response is provided then I complied with the particular recommendation.**

The contractor was tasked with preparing a technical report to evaluate the documented and potential effects of parasites and diseases in Fraser River sockeye salmon and their role in the 2009 run failure. Admittedly, this report would be difficult to generate given the many unknowns associated with disease in wild sockeye salmon. Nonetheless, the report suffers from a number of weaknesses that could be addressed to improve the value and credibility of the document. The document is also in need of a copy edit to address numerous typos.

Although it is certainly not possible to do a meta-analysis, a review or synthesis such as this should be repeatable at least in terms of the ability to locate the materials. Indeed, an evidence-based approach to science (or medicine) requires that one is exceedingly transparent and detailed with the methods used to locate materials for inclusion (and equally important – exclusion) in a review. The “methods” for the paper constitutes a single sentence.

1aThere is no information on the data-bases searched, the search terms used, etc. The author needs to both convince the reader that their search was exhaustive (I am not convinced) and provide adequate detail such that it could be repeated.

One of my general issues with the report is that it is subjective. This is not the fault of the author given that the literature is not sufficiently dense with information on sockeye disease to enable anything less subjective. It is also worth noting that the terms of reference also indicate that the Cohen Commission is looking for a subjective assessment. However, there is need for additional information on what criteria were used to base the assessment. At present, the risk basis assessment is described in a single sentence.

1b. There is need for more information on how the author weighed evidence and how decisions were made sometimes in the absence of reasonable data. The word subjective is in and of itself a broad term and the author can provide more information on the specific factors used to determine risk.

**Pertaining to 1 b. This is expanded some, but the data available now clearly demonstrate that we can not go beyond a subjective assessment at this time.**

One of the considerations in the risk assessment is "the likelihood that the pathogen would be prevalent in the Fraser River or British Columbia". I think that a more reasonable assessment would be to include all of the waters that are "used" by sockeye throughout their life which includes waters outside of British Columbia and Canada.

In its current form, each "disease" is tackled in sequence where the author provides a brief overview of what is known about the disease followed by the risk assessment. The document assumes that the reader has extensive knowledge of a given disease which makes the review less accessible and useful for those without training in aquatic animal health. **Ic** To remedy that problem I would suggest that each disease section be structured with a series of subheadings. Additional text (with references) should be added to provide more background on the diseases. For example, for each disease it would first be useful to know something about it – perhaps a subtitle called "Description of Disease". Next it would be useful to then comment on "Distribution and Prevalence". Another section could be "Transmission/Mode of Infection", "Impacts on Pacific salmon", etc. Not being an aquatic animal health specialist I am sure that these are by no means the correct subtitles. However, these titles do cover the type of information that I think is needed. In some cases there will be little information – but I feel that some of this information is known but is assumed to be common knowledge of the reader.

**Ic. The revised report includes a table. This provides a summary with the categories (more or less) as requested. Therefore, I have chosen not to include the same subtitles in the body of the text.**

1d. One of the areas in need of improvement is better support for statements made within the report. In some passages, there are sentences that require references to provide credibility. Every sentence needs to be read with a critical eye – does the sentence stand on its own or is a reference needed?

**1d. Citing every statement with a reference would make the report too long and cumbersome to read. However, I have judiciously added some addition references to address this concern.**

1e.In general, I am surprised that more scientists/aquatic health professionals were not consulted in preparing the document. I am most familiar with the work on Parvicapsula. There are a number of folks with additional data that could certainly be included here… Contact Simon Jones, David Patterson and Scott Hinch. There is evidence of degree day thresholds – see climate change paper for more thorough description.

**1e. I relied on peer-reviewed literature and supplemented this with grey literature reports and conversations with many scientists. Time restraints under the contract do not provide for interviewing a vast number of researchers, and the peer-reviewed literature is the most reliable source. For example, regarding *Parvicapsula,* there are several recent peer-reviewed papers that cover the subject adequately.**

1f. An important mediator of opportunistic disease is injury. The report failed to discuss the potential sources of injury that would be experienced by salmon. For example, to reach spawning grounds sockeye salmon must often negotiate FN and commercial gillnets and hooks from the recreational fishing sector. Fish that drop out of gill nets experience a range of injury (e.g., scale loss, slime loss) which can promote development of infections such as Saprolegnia.

**1f. Role of trauma relating to Saprolegnia was added to the revised report.**

Similarly, fish that are captured, handled and released (or breakoff/escape) can also experience localized injuries and slime/scale loss. These sources of injury must also be recognized in the section on “Environmental Factors”.

**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?**

Because the risk assessment is subjective (as stated in the terms of reference and the report) and given the many “unknowns”, one could certainly argue over the relative risks of the different diseases. However, I think that one of the take home messages is that there are many different diseases that exist in the wild and that sockeye salmon have the potential to be exposed to many of them. The author also concludes that “there are no firm links for these pathogens with significant demise in these sockeye populations overall, but some of these (diseases) are clearly associated with prespawning mortality in freshwater”. I would concur that the author really can’t say much more and that this is a reasonable conclusion. 2a. I think that one of the things that must be noted is that the risk assessments tended to be not based on mediators. For example, although Parvicapsula seems to be problematic at higher temperatures (due to rapid accumulation of degree days), at cool temperatures it may not be as problematic.

**2a. Revised text expanded some to emphasize importance of temperature.**

Similarly, Saprolegnian infections are probably not meaningful for uninjured fish but if there were interactions with lots of predators or fishing gear (e.g., drop-outs or releases), then one might consider Saprolegnia to be problematic. Adding "if then" or other caveats to all of the various risk assessments is not really logical, but it is worth noting that the risk assessments tend to be based (from what I can discern) on "average" situations. As such, a missing link is specific reference to the environmental conditions faced by fish in 2009 as well as in earlier phases of their life-history (i.e., 3 or 4 yrs prior to 2009). I am not sure where that integration should occur – perhaps in this report – or perhaps in a summary synthesis that I presume will be generated.

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

Some ideas are presented above.

3a. Please pay particular attention to the suggestion for use of more subheadings for each disease, the need to provide more background information, and more liberal referencing.

**3a. The new table addresses this concern.**

3b. It is critical that the "methods" be expanded such that the reader can evaluate thoroughness of the review and the criteria used to assign risk.

3c. I would suggest that the author construct a summary table with each disease that summarizes risk as well as other information. For example, I could envision columns on prevalence, mode of transmission, etc, and relative assessment of the amount of information on a given topic. The text for each disease would serve as the detailed information and the table would be a quick reference and synthesis.

**3c. The table added to the revised report addresses this request.**

3d. The section on "state of the science" could include a quantitative analysis. For example, of the X number of diseases examined in the report, what percent have adequate information to enable a reasonable risk assessment. There are all sorts of simple summary stats that could be generated to emphasize what is and is not known.

**3d. I have added the total number of diseases that are reviewed in the Executive Summary.**

**4. Are the recommendations provided in this report supportable? Do you have any**

**further recommendations to add?**

4a. The recommendations could benefit from being presented as a series of bulleted points with supporting material. Currently one has to work hard to pick the specific recommendations from within the text.

The research plan provided by the author would benefit from much more detail. For example, the authors suggest that surveys should be conducted over several years. I would argue that an entire brood cycle may be an appropriate time period. 4b.I would like to see a list of specific research needs for each disease.

**4b. The amount of detail on recommendations is appropriate in its present form. Please refer to the Scope of Work and Objectives (Appendix 1). Here there is clearly no directive to provide <u>any</u> recommendations. DFO and other scientists can take the general recommendation guidelines provided in the present report and design appropriate research projects. In other words, I provide a recommendation on the general approach to assess the impacts of a particular pathogen. Once the pathogen is identified and the impact is determined, then specific management methods might be considered to ameliorate the severity of the particular infections. Each case would be different, and thus it is not appropriate to provide details on management recommendations for every possible scenario.**

4c I am surprised that the author does not comment on the ramifications of the various risks. For example, does this knowledge inform how non-selective fisheries should be executed to minimize potential for disease spread? What are the biosecurity implications for stock enhancement, stock assessment and research?

**4c. Again, this is beyond the Scope of Work. Regardless, all of these recommendations would follow based on conclusions generated from data collection and appropriate analysis.**

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

Clearly any information on disease must be collected on a range of sockeye populations. The recommendations indicate that surveillance should be conducted in different geographical areas but does not explicitly recognize the potential for different responses among populations.

**5a. A statement regarding the need to conduct surveys in various locations was added to the section ASSESSMENT OF PATHOGEN-ASSOCIATED MORTALITITY IN WILD SALMONIDS.**

Several sentinel populations (representing different life-histories and environmental conditions) should be selected (on spawning grounds and rearing areas) that are monitored annually as part of routine monitoring programs (just like stock assessment).

**5b. Sentinel programs could be initiated on AFTER it is determined which pathogens are of concern. Sentinel fish are very useful for determine the temporal and geographic distribution of pathogens, but provide little information on the impacts in the adjacent wild populations.**

Some of these activities occur through the Fraser River Environmental Watch Program but there is room for more sampling and over longer temporal periods. It is worth noting that biotelemetry studies can be used as part of a suite of tools to study disease consequences on wild fish, particularly when combined with non-lethal biopsy to collect tissues for analysis (including gene array data).

5c. The research agenda is weak. I suggest that the authors provide a list of disease-specific knowledge gaps. Please be very explicit. I would encourage using bulleted points.

**5c. I disagree that the research agenda is weak based on the Scope of Work (Appendix 1). Determination of the impacts of various pathogens of sockeye salmon in BC should be done first. The point is that we do not have strong empirical data that document, with any certainty, the impacts of the pathogens discussed. After impacts are determined using the approach provided, scientists would then proceed with the appropriate control measures.**

**Please re-visit Scope of Work in Appendix 1. The directive was to provide a broad view [review] of the diseases and parasites in salmon, and the role and impact they have on Fraser sockeye. I was not directed to provide an extensive research agenda.**

Report Title: **Infectious Diseases & Potential Impacts on Survival of Fraser River Sockeye Salmon**

**Reviewer 3:** M. Rosenau
Date: December 22, 2010

Note: extensive and detailed comments by reviewer (Rosenau) are embedded into the body of the Kent report, in an electronic form. This is attached as an appendix to this review. **I went through these editorial comments and incorporated appropriate changes.**

**1. Identify the strengths and weaknesses of this report.**

**General Comment. This reviewer requested significant increase on attention to the sea lice issue. As per reviewer 2, citations regarding sea lice were increased but this section was not greatly expanded as this is covered in another report.**

**Regarding introduction of pathogens – there have been no documented introduction of salmonid pathogens in British Columbia. I included a short reference to this and a few citations regarding the potential impacts introduced, including a paper that I wrote with D. Keiser.**

Strengths

1. This report appears to review, except for sea lice, the major diseases that might have impact to sockeye in BC.

2. It also ranks, what likely might have had an impact on sockeye over the last 20 years, from high to low.

Weaknesses

1. I believe that this report needs a better structure, including an Introduction, Objectives, maps of disease locations, etc.

   **Maps – this was not included as there are no significant documented geographic patterns of the pathogens of concern.**

2. I think that an Executive Summary should be included at the front of the report.

3. Any Recommendations need to be clear, precise and in a bullet form.

4. I think a table outlining the diseases and their risk, and other pertinent characteristics would be very useful.

5. There are lots of editorial errors within the body of the report that need to be cleaned up, and I have dealt with this in my attached copy.

6. The References section has lots of editorial errors, which I didn't provide comments on in detail, that need to be addressed.

7. To me, the report does not tie in the actual or potential historical issue of novel strain/disease transfer into the province through fish culture, either via agency facilities (FFSBC and its pre-cursor, or DFO hatcheries) or the fish farming industry which, I think, is really the question on everyone's mind.

**Historical issues – I added a section regarding the fact that there is no evidence of introduction of novel diseases, and I also bring up this point as appropriate when discussing apparent "new" diseases.**

General

For me, the report needs some sort of greater synthesis in respect to the likelihood of diseases being the causal agent of the collapse of Fraser River stocks of sockeye. To me, right now, the report simply stands as a list of diseases, but doesn't provide any geographic or historical basis for their implication of any large outbreaks that might of happened in southwest BC salmonids, in general, and Fraser River sockeye stocks, in particular. So the question that I, and a lot of other people have is, was there a novel disease, or strain of an existing disease, that was transferred into the province via a fish-culture facility, federal or provincial, or private.

**As stated above, there is no indication that an exotic salmon pathogen has been introduced into BC.**

In that context, the report fails to provide any clear thoughts as to whether disease, or any particular disease, was likely to have been the cause of the declines. In other words, to me this is simply a list of the possible diseases and the report superficially categorizes the risk at high, medium or low without providing any hypotheses or review of actual incidents in BC. While this might be all that the author might be able to do given the constraints of time, budget and information, I think that, if this is the case, it really has to be emphasized that there is no available information on disease that can be drawn from the available data (if this is true).

**The fact that there are no available data that clearly link the demise of sockeye salmon to a specific disease is emphasized even more in the revised report.**

A really looming question that hasn't been covered in the report surround the questions relating to fish farms and the potential of this component of their disease history, and they being a vector, was largely glossed over in the report. As an example, the

opponents of fish farming have come up with a number of disease scenarios (sea lice, IHN) and I think these questions need to be assessed more thoroughly and the report state, “yes”, “no” or “this is how we would find out”. If the questions surrounding fish farms are not more unequivocally addressed, then the public will be unhappy with this analysis. To be fair, however, perhaps that is a much bigger study, or it is an impossible study given the resources that have been made available, but in any event the public should be told as such.

**Fish farms and sea lice are dealt with in more depth in another report (Report 5).**

A big issue is sea lice; questions surrounding sea lice were only lightly touched on. This is a large issue in the minds of many. The coverage of this issue in this report was highly superficial and left the reader wondering what the problem might be between sea lice, fish farms and sockeye, when it is clear that there is a lot more material within the greater body of scientific literature that has been published. This report suggests that this issue is being dealt with by another, or other, investigator(s). If this is the case, then the analysis on sea lice in this report should state that it is being covered elsewhere and the subject of sea lice be completely dropped, *holus bolus,* for this report.

**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?**

To be honest, I really didn’t see a strong presentation of the historical presence of the various diseases in British Columbia, where they were found, when outbreaks occurred, and did Fraser sockeye see the brunt of infections. The history of the introduction of diseases via fish culture in BC, with respect to any diseases that would affect sockeye, would really help this report, I think.

**I have added an indication about newly discovered diseases. However, even for these, there is no evidence that they have not been in BC for a long time. This is added to the revised text.**

I think, also, the description of the wild-infection/mortality analysis could really use some figures to pictorially describe what the author is talking about. I think such figures would clarify in a lot of peoples minds exactly what the author is talking about.

**Done.**

**3. Are there additional quantitative or qualitative ways to evaluate the subject**

**area not considered in this report? How could the analysis be improved?**

The author has outlined some of these in his recommendations. I think, however, the author needs to be more descriptive in this regards. I would point out that Vic Palermo did an analysis about 6 years ago in respect to infection rates in the Broughton Arch as fish moved through the channels from their natal streams out to the open ocean. I am not sure that this stuff has ever seen the light of day. In particular, he was using the kinds of analyses that medical scientists use when they are looking at, say, cancers in human populations as you fan out from a nuclear waste site. I think that Vic's work was predominantly sea lice, but may have included other pathogens. The author of this report needs to incorporate the Palermo analysis in his study.

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

The recommendations are too fuzzy, in my opinion. These need to be laid out in a clear bullet form, and need to be immediately below the EXSUM so that the politicians/senior managers know what the author is thinking.

**Recommendations were not a specific directive part of the Scope of Work. Thus this section is not as extensive as others. I provide recommendations on the general research approaches that should be used, but details would be adjusted depending on the life stage and pathogen species of most concern.**

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

Issues surrounding the linkages between fish culture (and, specifically, fish farms), disease and the potential/likely-unlikely cause of the collapse of Fraser River sockeye. This is the big question that is on the tips of everyone's mind and is a key issue that needs to be addressed. I am not sure that the author tried to go into any of the recent historical outbreaks of diseases in fish farms in British Columbia, and reporting on this issue. Alexandra Morton et al. keep talking about these outbreaks—are they real, or not? And if so, they should be addressed in this report, in my opinion.

**Again, this is for the fish farm report.**

**6. Please provide any specific comments for the authors.**

All of the specific comments are embedded in an electronic copy with I have supplied. This report needs considerable upgrade in terms of the editing, of which I have supplied

considerable comments. The References also need to be gone through with a sharpened pencil and edited, which I have not done for the author.