



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

June 2011

**TECHNICAL REPORT 5D**

# Impacts of salmon farms on Fraser River sockeye salmon: results of the Dill investigation

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Recommended citation for this report:

Dill, L.M. 2011. Impacts of salmon farms on Fraser River sockeye salmon: results of the Dill investigation. Cohen Commission Tech. Rept. 5D. 81p. Vancouver, B.C. [www.cohencommission.ca](http://www.cohencommission.ca)

## 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**

Open net pen aquaculture, as currently practiced in British Columbia, has the potential to create problems for wild salmon populations because the pens are open to the environment, allowing wastes, chemicals and pathogens to move freely back and forth. Indeed, wild salmon populations have tended to decline wherever this form of aquaculture is practiced, although the reason for this is not always apparent. In one of the best studied cases, wild Pacific salmon in the Broughton Archipelago, BC appear to have been negatively impacted by sea lice from fish farms.

Declines in Fraser River sockeye salmon returns, and in particular the spectacular crash of 2009, have led many to wonder whether fish farms could be implicated, given that most of the migrating sockeye have to pass through the narrow channels among the Discovery Islands, dotted with numerous Atlantic salmon and Chinook salmon farms, on their way north out of the Strait of Georgia.

The hypothesis that there is an effect of farms on sockeye survival was tested by examining the support for its predictions that there would be negative relationships between fish farm production levels - and such farm metrics as lice levels, disease levels and farm mortality rates - and Fraser sockeye survival. These various relationships were statistically analyzed and reported separately to the Commission by Dr. Brendan Connors (Connors B. 2011. Examination of relationships between salmon aquaculture and sockeye salmon population dynamics. Cohen Commission Tech. Rept. 5B).

Unfortunately, it turned out that the data provided by Provincial government (BCMAL) and the BC Salmon Farmers Association (BCSFA) were insufficient in both quantity and quality to allow a rigorous analyses capable of answering these questions with certainty. The biggest problem was the very short length of the time series available for analysis, basically only 4-5 year classes.

However a longer-term analysis, using production data since 1982, did reveal a relationship between farm production and salmon survival, i.e., the greater the farm production the lower the survival of the sockeye. This analysis also revealed a very interesting interaction with pink salmon abundance in the North Pacific Ocean: the negative effect of the farms appeared stronger when pink salmon were more abundant, suggesting that any farm effect may be mediated through changes in the growth and/or competitive ability of the sockeye.

Despite the *a priori* predictions, these results cannot be considered conclusive, as they are only correlations in the data. However, the fact that the 2006 brood year interacted with half as many pink salmon as the 2005 brood year, and that the corresponding 2010 returns were much greater than those in 2009, suggests that the Connors statistical model may be capturing some underlying causal relationships, and thus motivates the search for what these might be.

Several potential drivers of any farm effect were considered. If such an effect exists, it is most likely to be due to either disease or sea lice, or both. Impacts on sockeye from other factors, such as escapes or waste and chemical inputs and their effects on the benthic and pelagic zooplankton communities, are likely to be quite local and unlikely to be sufficient, alone or in concert, to cause either the long-term population declines or the especially low returns in 2009. However, the cumulative impacts of several farms in close proximity have not been adequately addressed.

The viral and/or bacterial pathogens considered the most risky to wild sockeye are *Renibacterium salmoninarum* (causing bacterial kidney disease, BKD), the IHN virus (causing infectious hematopoietic necrosis, IHN) and *Aeromonas salmonicida* (causing furunculosis). There are a variety of ways these may be transferred from farmed fish to wild sockeye, including horizontal transfer of shed pathogens, via farmed salmon escapees, via movement of infected sea lice (vectoring), and through discharge of untreated "blood water" from processing facilities. Horizontal transfer and vectoring by

sea lice are likely to be the most important routes of transmission, but the role of processing facilities needs to be examined further.

ISA (infectious salmon anemia) has not been confirmed on BC fish farms, but several of the veterinary records refer to symptoms that are highly suggestive. A close watch should be kept for indications of this disease, and biosecurity rigidly enforced, since ISA could be devastating to BC wild salmon populations. Recently there have been reports of a possible retrovirus (the so-called "Miller virus"); its role in Fraser sockeye declines is currently uncertain. It is suspected to be a contributory factor to the recently elevated levels of pre-spawning mortality (PSM) in adult Fraser sockeye, but PSM is not the cause of reduced survival as examined in this report, since the definition of "recruits" includes any mortalities due to PSM. Thus we are looking for the cause of declining survival over and above whatever effects this virus has on returning adults. Of course this does not exonerate the involvement of this presumed virus in mortality of sockeye at earlier life stages.

It is naïve to believe that the present report, and the Cohen Commission in general, will identify *the* cause of the sockeye salmon decline, and in particular the return failure of 2009. Nature is complex and factors do not act in isolation on the population dynamics of any species. Pathogens from fish farms are just one factor among many that may influence the mortality rate of sockeye. There are several ways in which these various factors may interact, and a number of these are discussed. Although some are hypothetical at this stage of our knowledge, they highlight the complexities in the real world system in which farms and wild sockeye are embedded, and caution against any simplistic single-factor explanation.

There are a number of knowledge gaps surrounding the farm-wild fish interaction, in particular those related to the dynamics of disease transfer. These are listed in a separate section of the report. Several management options are also briefly considered, with closed containment being the preferred option if it can be shown to be economically feasible, a

hypothesis currently under test by several such facilities in BC, both land-based and in the ocean.

It must be understood that the short time series of data available for this investigation precluded identifying salmon farms as an important driver of the decline of Fraser sockeye. But it must be equally understood that at this stage of our knowledge is it not possible to say they are not implicated. It is recommended that a well-organized farm database be maintained in an ongoing fashion by Fisheries and Oceans Canada, and that annual analyses of the sort performed by Dr. Connors be conducted to firm up conclusions as more data become available.

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## INTRODUCTION

Every fall, millions of adult sockeye salmon (*Oncorhynchus nerka*) ascend the Fraser River to spawn in its numerous tributary streams and then die. And two years later, after spending a year in a rearing lake, their offspring descend the Fraser to the ocean, where they will spend the next two to three years of their lives before returning to spawn in the same stream in which they were born.

After entering the Strait of Georgia via the Fraser's estuary, the young sockeye, measuring only about 75-90 mm in length, spend only a short time there, feeding on zooplankton. Most of the fish then move northwards and exit the Strait through the complex of passages through the Discovery Islands, north of Campbell River. They move through this area relatively quickly (Groot & Cooke 1987; Groot et al. 1989, Welch et al. 2011), exiting through Johnstone Strait and into Queen Charlotte Strait and then Queen Charlotte Sound, on their way to the open ocean. By early July the vast majority of Fraser sockeye have headed out to sea by this route, although a few, primarily from the Harrison River, are believed to enter the open Pacific through Juan de Fuca Strait. The abundant food in the North Pacific supports the growth of the sockeye to adult size, and powers their eventual return migration to their natal streams, normally after two years in the ocean, at the age of four.

This cycle has gone on for at least 10,000 years and with a few exceptions, like the Hell's Gate blockage of 1913, there have always been enough fish returning to replace their parents. Fisheries biologists refer to the returning fish as "recruits", and measure survival as the ratio of recruits to the number of spawners that produced them. If this ratio exceeds 1.0, then the population is replacing itself, and again (with those few exceptions) this has always been the case for Fraser sockeye, even in the face of a large commercial fishery.

But beginning in the mid-1990's, things began to change, and not for the better. The survival of the Fraser sockeye stocks, in aggregate, began to decline (Figure 1). This reached a nadir in 2009, when 10.5 million fish were predicted to return and only 1.5

million did so. As a consequence the Government of Canada established the Cohen Commission of Enquiry into the Decline of Sockeye Salmon in the Fraser River, to look into possible reasons for this economic and biological disaster. More precisely, the Commission’s mandate was “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.” Among the possible issues that needed to be assessed in relationship to the decline, and the subject of the present technical report, is the role of open net salmon farms, i.e., salmon aquaculture as practiced in British Columbia. Indeed, this issue appears foremost in the public’s perception of culprits (61% of all public submissions to the Commission through September, 2010 dealt with aquaculture; Cohen 2010).



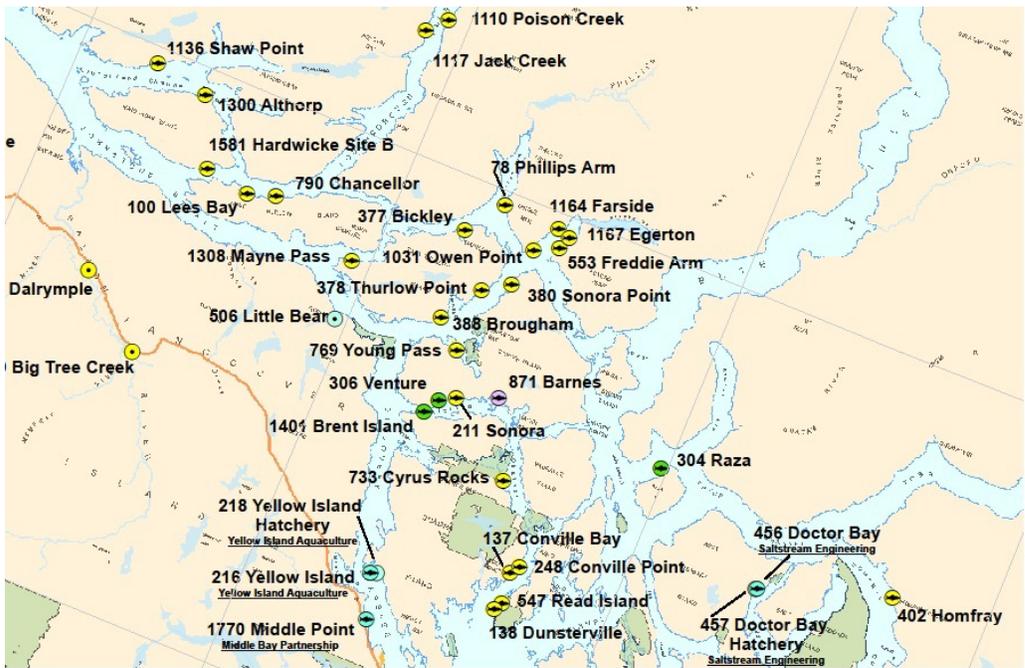
**Figure 1. Time series of Fraser sockeye adult returns per spawner, plotted as a 4-year running average (from Cohen 2010).**

### **Discovery Islands Farms and Fraser Sockeye Juvenile Migration Routes**

Fish farming officially began in BC in 1972, and expanded into the Discovery Islands area in the late-1980s (Keller & Leslie 2004). Originally the most common species raised in the net pens were coho (*Oncorhynchus kisutch*) and chinook (*O. tshawytscha*) salmon, but industry production is now dominated by Atlantic salmon (*Salmo salar*). The proportion that chinook salmon made up of the production on the east coast of Vancouver Island dropped continuously from 2003-2007, averaging about 24% during that period.

The Discovery Islands salmon farms are shown in Figure 2. Conservation organizations have been especially concerned with farms in Okisollo and Hoskyn Channels, the area they have called “The Wild Salmon Narrows”, namely Cyrus Rocks, Barnes Bay, Venture Point, Sonora, Brent Island and Conville Bay (see Fig 2). These channels are certainly used by seaward migrating juvenile sockeye and, based on recent fieldwork (M Price UVic, pers comm), perhaps more so in recent years than in the past (Groot et al. 1989).

Once the Fraser sockeye exit the Discovery Islands and enter Johnstone Strait they pass by the several entrances to the Broughton Archipelago, another major fish farm area on the mainland side. There is no evidence to suggest that the sockeye enter the Broughton archipelago and pass by the many farms there, but in Johnstone Strait they would meet potentially infected pink (*O. gorbuscha*) and chum (*O. keta*) salmon who have passed the farms on their seaward migration from Knight Inlet and other streams in the region, providing a route for pathogens from those farms to secondarily infect the Fraser sockeye.



**Figure 2. Salmon farms in the Discovery Islands (section of BCMAL map).**

## Negative Impacts of Salmon Farms on Wild Salmonids

There have been numerous reviews of the potential impacts of salmon aquaculture on wild fish stocks (some recent ones include Noakes et al. 2000, EVS 2000, Nash 2003, Waknitz et al. 2003). Ford & Myers (2008) conducted a meta-analysis to show that wild salmonid stocks have declined, often as much as 50%, wherever aquaculture production has increased. Despite this there are surprisingly few demonstrations of the mechanisms underlying negative impacts. Escaped fish are believed to be the source of furunculosis in a number of Norwegian rivers in the late-1980s and early 1990s (Hastein and Linstad 1991, Heggberget et al. 1993) and an increased prevalence of infectious pancreatic necrosis has been found in wild fish near fish farms in Scotland (Munro et al. 1976, Wallace et al. 2008).

Sea lice (*Lepeoptheirus salmonis*) are a conspicuous exception to this general lack of empirical information on impacts of farms on wild fish (Costello 2009). In Ireland, for example, there is good evidence that some stocks of sea trout (*Salmo trutta*) have been negatively affected by lice originating in farms (Tully et al. 1999, Tully & Nolan 2002, Gargan et al. 2003). According to Gargan et al. (2006) "the data ...strongly indicate that infestations by sea lice...made an important contribution to the sea trout stock collapse on Ireland's west coast," and "exceptions of good survival were associated with whole-bay fallowing by adjacent marine salmon farms." In Norway both sea trout and Arctic charr (*Salvelinus alpinus*) have been shown to have higher lice loads near farms than further away (Bjørn et al. 2001, Bjørn & Finstad 2002), and this has been implicated as a cause of population declines of these species. In a very interesting experiment, hatchery Atlantic salmon protected with an anti-lice treatment prior to release survived at a slightly greater rate than untreated ones (Hvidsten et al. 2007), suggesting lice as the cause of mortality. However, the sample size for the experiment was small and the result was not consistent across years. A similar experiment in Ireland (Jackson et al. 2011) also found that pre-release anti-lice treatment (in this case with SLICE®) reduced mortality significantly, but only to a very small extent.

Here in British Columbia, there is considerable evidence for impacts of sea lice on wild pink and chum salmon juveniles in the Broughton Archipelago. It is now widely accepted that the source of the high levels of infestation observed on these wild fish, at least until recently, has been the farms (e.g., Marty et al. 2010). Atlantic salmon in the farms pick up sea lice from adult wild salmon moving past them towards their spawning streams in the fall. The net pens, with their high densities of susceptible hosts, essentially act as incubators for the lice over the winter, and their numerous progeny (Orr 2007) then infect the juvenile pink and chum moving seaward in the spring. These fish are quite small, and lack protective scales, and have not been prepared by their evolutionary past to deal with an infection at this early stage of their life (normally any infection would not occur until the fish meet the returning adults later in the summer, when the juveniles are less vulnerable; Krkošek et al. 2007b). If the intensity of infection is high enough the young wild salmon can die, either from the direct effect of the parasite, or from indirect effects such as secondary infections or predators (Krkošek et al. 2011). If enough individuals die, and these would not have died from some other cause in the absence of lice, then the population will decline, and there is evidence that this has happened to pink salmon in the Broughton (Krkošek et al. 2005, 2006, 2007a). There is even evidence to suggest that Broughton area coho salmon, who can pick up lice when feeding on infected pinks (Connors et al. 2008, 2010a), have also shown population declines attributable to lice (Connors et al. 2010b).

Not everyone agrees with the picture I have just painted. The Krkošek papers have engendered a lively interchange of criticism (Brooks & Jones 2008, Riddell et al. 2008) and rebuttal (Krkošek et al. 2008a, b). Most recently, Marty et al. (2010) failed to find a relationship between lice levels on farmed fish in the Broughton and pink salmon survival; however, their analysis had a very small probability of being able to detect such an effect (it had what statisticians call low power), and was flawed in other ways as well (Krkošek et al. in review). Jones et al. (2008) conclude from laboratory studies that pink salmon are only susceptible to *L. salmonis* when weighing less than 0.7 g. and so quickly outgrow the period of vulnerability. However, these sorts of studies do not place the fish in challenging natural environments where they must find food and avoid predators, both

of which abilities may be compromised by lice. In addition they do not replicate a critical feature of the field situation – the possibility of multiple infection as a result of sequentially passing several farms in close proximity (Wagner et al. 2008). On balance, I believe the science strongly supports the conclusion that pink salmon in the Broughton Archipelago, and perhaps other salmon species there as well, have been negatively impacted by lice from fish farms.

Some have pointed out that sockeye smolts are large relative to pink and chum salmon and well covered with silvery scales that act as a physical barrier, so the likelihood of being affected by lice should be negligible. However, this ignores the fact that even adult sockeye can be killed by lice in sufficient numbers and under adverse environmental conditions (Johnson et al. 1996). It also ignores the fact that fish as large or larger are affected by lice in Europe.

In summary, the available evidence suggests that salmon farms can be deleterious to sympatric wild salmon, at least under some circumstances and in some places. The question that will be addressed in this report, to the extent possible with the data available, is whether this is likely to be true for Fraser River sockeye salmon.

### **The Cohen Commission Contract**

As stated in the contract (see Appendix A for the Statement of Work) this project, i.e., Scientific Project #5, was intended to provide “An assessment of the impacts of salmon farms on Fraser sockeye .... to evaluate their importance to the ecology and survival of Fraser sockeye and to determine their role, if any, in the reductions in Fraser sockeye abundance.” The objective of the contract was stated as: “To prepare a technical report containing a review and evaluation of the effects of salmon farms on Fraser River sockeye salmon.” This report is one outcome.

However, the role of other scientists in Project 5 should also be noted.

- Dr. Josh Korman, of Ecometric Research, collated and summarized the data provided to the Commission by the BC Ministry of Agriculture and Lands (BCMAL) and the BC Salmon Farmers Association (BCSFA) and prepared a summary report; and
- Dr. Brendan Connors, Simon Fraser University, conducted statistical analyses of these data in relation to sockeye production data made available by the Commission. The purpose of this analysis was to test specific hypotheses about impacts. Connors provided a report to the current author (LMD) and to Dr. Donald Noakes, Thompson Rivers University, who was tasked to produce his own report in a parallel process.

The results of their subprojects were published as stand-alone documents (Korman 2011, Connors 2011). The Noakes version of the present analysis has also been published as a Cohen Commission technical report (Noakes 2011).

## SUMMARY OF THE CONNORS ANALYSES

Connors (2011) carried out two separate sorts of analyses – a long-term analysis of the relationship between farm production and sockeye survival, and several short-term analyses of the relationships between salmon farm metrics and sockeye survival. The latter analyses used the data provided by BCMAL and the BCSFA to the Cohen Commission.

The long-term analysis relates sockeye survival to farm production since 1982 but has less spatial resolution than the short-term analyses, i.e., it does not have data for individual farms. Other factors, identified independently as potential drivers of sockeye declines, were also included in the analysis. Connors found support for main effects of farm production, sea surface temperature (SST), and North Pacific pink salmon abundance: increases in any of these were associated with lower sockeye survival. There was a positive interaction between farm production and SST, i.e., the effect of farms was less when water temperature was higher, perhaps suggesting compensatory mortality (in lay terms, only one thing can kill you). He also found support for an interaction between exposure to salmon farms during early marine life and pink salmon abundance on sockeye survival: the negative effect of farm production was strongest in years when pink salmon were most abundant in the north Pacific. Pink and sockeye salmon have diets that overlap extensively (Landingham et al. 1997) and pink salmon are known to be competitively dominant over sockeye and thus reduce the amount of food available to them, leading to reduced growth and survival (Ruggerone & Nielsen 2004, Ruggerone et al. 2003, 2005). This synergistic interaction between salmon farm production and pink salmon abundance is estimated to have increased sockeye mortality by a considerable amount in some years, including 2009 (Connors 2011, Fig. 6). Importantly, this finding also suggests that an attempt to find a single cause for the decline in survival of Fraser sockeye stocks is unlikely to be successful (see **The Futility of Looking for The Cause of Fraser Sockeye Declines**).

The analysis was run without data for the 2006 brood year (passing the farms in 2008).

However, it is important to note that the 2006 brood year had to interact with half as many North Pacific pink salmon as the 2005 brood year (2005: 628 million; 2006: 296 million; see Fig. 5 in Connors 2011). Given that the effect of farms was strongly mediated by pink salmon abundance this qualitatively fits with the improved survival of the 2006 brood year over the 2005 brood year, as predicted by the model (Fig. 6 in Connors 2011) and observed in the fishery itself, likely because of the much smaller number of pink salmon these sockeye had to compete with. This provides a level of confidence that the statistical model is capturing some real underlying processes. Nevertheless, the results are correlative, and as such provide no proof of causation. Also, as Connors (2011) himself points out, there is considerable uncertainty around these estimated effects, which precludes drawing strong inference from the results.

The short-term analyses failed to find any significant negative relationships between a number of salmon farm metrics (production, lice levels, fish health events, disease audit findings, mortalities) and salmon survival. Many of these relationships had negative slopes (see Connors 2011, Table 4), but none of them approached significance.

Thus, while the findings of the long-term analysis suggest that (1) high standing stocks of fish in floating net pens in the Discovery Islands can negatively impact Fraser sockeye survival, and (2) the strength of that relationship depends on pink salmon abundance in the north Pacific, the short-term analyses cannot identify a causal mechanism underlying the relationship.

It is important to bear in mind, however, that the short-term analyses were seriously constrained by the number of years for which data on various farm parameters were available. Data for most metrics (mortality, fish health, production) were available only from 2003 - only from 2004 for sea lice - and the last brood year for which survival data were available was 2005 (and even then the return of 5-yr olds from this brood had to be estimated). This means only the five sockeye brood years (2001 to 2005) that passed the farms from 2003 to 2007 could be included in the analysis (4 brood years for lice variables, 2002-2007). Korman (2011) showed that given these short time series there is

low statistical power to detect relationships should they truly exist. In addition the dataset did not allow for a closer look at the effect of individual farms (data were aggregated across fish health zones), or for a breakdown according to proximity of the farms to the presumed migration route of the majority of juvenile Fraser sockeye. As a consequence the findings of this report are not nearly as conclusive as many surely had hoped.

## POSSIBLE DRIVERS OF FARM EFFECTS

Given the results of Connors' long-term analysis, and the failure to identify any underlying driver(s) for the observed relationship, it is important to look at some of the possible casual factors. Open net salmon farms have a number of potential impacts on the marine ecosystem in which they are placed and become a part. One or more of these might be responsible for the observed effects on Fraser sockeye populations; each will be examined in turn.

### **Benthic impacts**

There can be considerable accumulation of uneaten food and faeces (and sometimes dead fish and fouling organisms removed from the nets) beneath farms, causing changes in species composition in the bottom community and, in some cases, anoxic conditions. These impacts have been reviewed several times in recent years (Brooks & Mahnken 2003, Hargrave 2003, Wildish et al. 2004, Black et al. 2008). There have also been at least two detailed studies of the deposition of wastes around farms in BC (Sutherland et al. 2001, Brooks 2001) as well as one in Puget Sound, WA (Weston 1990). Reviews of this literature and personal communication with experts (S Cross, UVic) suggest that any impacts will be very local, perhaps 200 m from the farms at most, even in strong currents such as found in the Discovery Islands. There will certainly be impacts on bacterial communities (Black et al. 2008) and benthic invertebrate species, favouring groups like nematodes and polychaetes that are tolerant of low oxygen conditions (Hargrave 2003). However, it seems highly unlikely that such local effects could impact Fraser sockeye survival to any great extent.

The only possible exception to this might be if invertebrate species that are intermediate hosts of parasites flourish below the farms (see **Routes of transmission**, below).

## **Pelagic impacts (plankton)**

The inputs mentioned above can contribute nitrogen to the water column, which could conceivably affect the phytoplankton and zooplankton communities there. The most important form of nitrogen with respect to phytoplankton growth is nitrate – if natural amounts are limiting, increased anthropogenic inputs can cause phytoplankton blooms (i.e., eutrophication). However, there is strong mixing in these waters down to 90 m (M Foreman IOS, pers comm) and any form of nitrogen would dissipate quickly, certainly within 100 m (S Cross, UVic, pers comm). In any event, nitrate is not considered to be limiting to phytoplankton growth in these waters (S Allen UBC, pers comm; D Mackas IOS, pers comm), and therefore no significant effects on phytoplankton communities are likely (Brooks & Mahnken 2003). Only sites with little to no water movement (certainly not the norm in the Discovery Islands) would show locally increased phytoplankton biomass (Buschmann et al. 2007). This conclusion applies as well to harmful algae such as *Heterosigma akshiwo* (Rensel 2007, Cross 2007). Thus, although it has been suggested that intense and prolonged *Heterosigma* blooms in Georgia Strait may be associated with low survival of some Fraser River sockeye stocks (Rensel et al. 2010), fish farms are very unlikely to be the cause of the blooms.

Another possible impact on the plankton community could occur if decomposing material in the benthos below the farms caused a reduction in oxygen levels in the water column. But studies near the Barnes Bay salmon farm (Okisollo Channel) reveal that dissolved oxygen (DO) values in top 15 m of water show no sign of depletion (M Foreman IOS, pers comm). It is therefore quite unlikely that there would be any but the most minor effects on plankton communities, whether phytoplankton or zooplankton. Brooks & Mahnken (2003) also concluded that there “appears to be little risk associated with reduced concentrations of DO associated with salmon culture”.

In the early part of their marine stage, juvenile sockeye consume insects and a variety of species of zooplankton, especially copepods but also amphipods, euphausiids and fish larvae (Healey 1978, 1980). The small shrimp-like euphausiids seem to be the major diet

item in other areas where juvenile feeding has been observed (Tanasichuk & Routledge, in press). Zooplankton feed on phytoplankton, so any change in phytoplankton production, however unlikely, could affect the populations of the zooplankton prey that make up the bulk of the diet of the juvenile sockeye. But with no change expected in the primary producers there is not likely to be changes in the zooplankton consumer populations either, and thus no change in food available to juvenile sockeye. If there were any effect on phytoplankton a knock-on effect on zooplankton populations would likely be lagged by about one month, occurring only after most of the sockeye had left the area for waters further north.

SLICE® is an in-feed chemotherapeutant that can be found in excrement and excess food under the farms, largely bound to particulates (S Cross UVic, pers comm). It has the potential to affect zooplankton populations (see next section) but given the low initial concentration and subsequent dilution, this is considered quite unlikely.

## **Chemical inputs**

A number of chemicals are used on and around salmon farms, including antibiotics, chemotherapeutants (notably SLICE®), antifoulants (especially copper), antibiotics and disinfectants. In addition zinc, a common component of fish feeds, is released into the environment inadvertently in excess food and fish waste products. There have been several recent reviews of the pathways and possible environmental effects of these chemicals (Buschmann et al. 2007, BurrIDGE et al. 2010). The general conclusion from these reviews is that owing to the high currents characteristic of farm sites in the Discovery Islands, and the consequent likelihood of only very local impacts (as for farm wastes; see above), impacts on salmon populations are unlikely. This is true of both direct impacts and indirect impacts via effects on food availability (i.e., zooplankton populations).

However, Buschmann et al. (2007) provide an important caveat, also raised by BurrIDGE et al. (2010), namely that while effects from a single farm may be negligible, this may not

be the case where there are cumulative impacts from multiple farms in close proximity, a situation that will be experienced by juvenile sockeye migrating through the Discovery Islands. They recommend “three dimensional hydrodynamic modeling to scientifically determine site selection” in an attempt to predict whether essential pelagic ecosystem functions will be compromised.

Given concerns that have been expressed about its use, a more detailed consideration of SLICE® is warranted. Currently this is the only in-feed therapeutant used in British Columbia; its active ingredient is emamectin benzoate (EB). Some laboratory studies (Willis & Ling 2003, Mayor et al. 2008, Kuo et al. 2010) have found that this chemical can have deleterious effects on marine invertebrates, including zooplankton. However, the amounts of EB used in these experiments is much higher than amounts found in the field near salmon farms, in part because it binds to particles and precipitates out. Indeed, field studies have failed to detect any toxic effects on invertebrates near farms using EB (Willis et al. 2005, Tefler et al 2006). Like other chemicals it is therefore unlikely to be responsible for killing Fraser sockeye, directly or indirectly.

### **Structural and operational impacts**

Sockeye juveniles move through the Strait of Georgia at a rate of 6-14 km/day (Groot & Cooke 1987, Welch et al. 2011), traveling mainly in the top 10-15 m (Groot & Cooke 1987, R Beamish DFO, pers. comm.), at least during the day and during favourable current conditions. When the currents are against them they are reported to rest in backeddies (M Price pers. comm.) and move downward to await the more energetically favorable outward flow (C. Groot pers. comm.). Farm structures themselves can create backeddies in fast flowing channels, perhaps encouraging juvenile sockeye to rest there during migration. In addition, in order to encourage growth of the farm fish, lights are kept on at night, even during the sockeye migration period. These factors (along with excess food falling through the pens) could attract sockeye, as well as other species, including predators and competitors. Fish farm lights are known to attract zooplankton and fish (e.g., McConnell 2010) and several species of wild fish have been reported to be

concentrated near fish farms elsewhere (e.g., Carss 1990, Dempster et al. 2002, 2004, 2009). For sockeye, this could mean an increased risk of infection by sea lice, bacteria and viruses, and perhaps increased mortality due to predation. Only the transfer of pathogens is likely to be important but there is no direct evidence of this. There is also no evidence of Atlantic salmon eating smaller Pacific salmon swimming into their pens (Hay et al. 2004).

Lights on the farms could also cause changes in diel migration pattern of zooplankton and fish in the immediate vicinity, altering the nighttime depth distribution of both. Without the normal crepuscular periods when most feeding probably takes place, there may be an effect on food intake by sockeye. It is difficult to predict the extent of this, but it is likely to be quite a local effect.

Sea lice copepodids, the stage that infects fish, have a reverse vertical migration pattern to most zooplankton, being near the surface during the day and moving to deep water at night, just as the fish are coming to the surface (Heuch et al. 1995). It has been suggested that louse-fish encounters occur while the two species are passing in their reverse migrations. This dynamic could be affected by night lighting, but whether this would increase or decrease encounter probability is impossible to say.

## **Escapes**

The escape data provided to the Commission (in some cases too late to be included in the Korman data compilation) were not of sufficient detail to be included in the Connors' statistical analysis, primarily because they were not disaggregated (i.e., provided for individual farms) and the format was inconsistent for the data that did exist. Also, for a variety of reasons it is probably not very accurate (Sumaila et al. 2005, A Thomson DFO, pers comm). Nevertheless, I consider it quite unlikely that escapes are implicated in the sockeye decline, because:

- very few Atlantic salmon have been found in the Fraser River (Burt et al. 1992, Korman 2011) so there is no prospect of competition for breeding space or mates with sockeye adults; and
- those re-captured in the ocean show very little sign of feeding (McKinnell and Thomson 1997; Morton and Volpe 2002). A few have been found with unidentified fish pieces in their guts, but none have been confirmed as sockeye. Thus predation on juveniles or competition with sockeye of any size is unlikely.

There is, however, a slight potential for disease to transfer to wild sockeye via escaped Atlantics (see **Disease** section below).

## **Lice**

Sea lice appear to be the major concern of the public, based on submissions to the Commission. And there is evidence that farms in the Discovery Islands are a source of sea lice infesting passing Fraser sockeye. Morton et al. (2008) were the first to report heavy sea lice infestations on juvenile sockeye near farms in the area. Price et al. (2011) found lice levels on juvenile sockeye in this area to be an order of magnitude higher than in areas of the North Coast without farms. They also found lice levels on the fish to be higher downstream of the farms (i.e., after passage) than upstream. The lice were mainly *Caligus clemensi* rather than *Lepeophtheirus salmonis* and the changes in their proportions across the two years of the study (2007-2008) matched changes in lice species proportions on local farms (2009 fit the pattern as well; M Price pers. comm.). In 2010 M Price (pers comm) followed sockeye northward from the Fraser estuary; they were exceptionally clean in mid-Georgia Strait but began to pick up juvenile *Caligus* just off Cortes Island, near the start of the Discovery Islands area. The hypothesis that farms are the source of lice is also supported by results of a companion study (Price et al. 2010) on lice levels on pink and chum salmon exposed to farms in this same area.

Despite this, there have been no experimental studies done on the impact of lice of either species on individual sockeye survival or on population dynamics. As noted above it has

been argued that the sockeye are too large (roughly 80-100 mm in length) and well scaled to be affected by the relatively small number of lice they host: the average incidence in the Price et al. (2011) study, i.e., the number on those fish who were infected, was 2.3-5.7 *Caligus* and 1-1.5 *Lepeophtheirus*. However small numbers of lice can have detrimental effects on large and well-scaled sea trout in Europe (Tully et al. 1993a,b, Bakke & Harris 1998). Some have argued that this latter observation is irrelevant because there are genetic differences between Atlantic and Pacific lice (Yazawa et al. 2008). But it has not been established that these genetic differences are related in any way to overcoming host immune responses, i.e., to pathogenicity, and there has been no common garden experiment conducted, testing both forms of lice against various salmon species in a common environment. Furthermore it is known that salmonid species vary in their susceptibility to infection, even by the same form of louse (reviewed in Wagner et al. 2008), so it is important that host species be part of the design of any such experiment.

A factor that could mitigate against lice having a significant impact is the speed at which the juvenile sockeye move through the area (Welch et al. 2011). Despite passing several farms in succession their total lice load might be accumulated over only a few days. This would be more analogous to a pulse infection rather than a multiple-infection scenario and the lice might be shed before they had a chance to do serious damage (Krkošek et al. 2009).

The Connors analysis failed to find a significant relationship between the number of lice in the farms during the spring migration period and survival of Fraser sockeye. This was true for both the larger *Lepeophtheirus* (including gravid females) and the smaller *Caligus* that, on a per parasite basis, are considered less pathogenic (Boxshall & Defaye 2006). So although there is evidence from field sampling that lice produced on the farms in the Discovery Islands are infecting wild sockeye juveniles, and lice are known to have deleterious effects on salmonid hosts elsewhere (Europe, the Broughton Archipelago), there is no evidence of a direct harmful effect in this system. However, lice may still be playing a role as pathogen vectors (see **Routes of transmission**, below), even if only attached for a short time before being shed.

## **Disease**

Open net fish farms can provide an abnormally high focus of infection due to the large numbers of susceptible hosts, a process sometimes called biomagnification. Furthermore, the high density of hosts and the treatment of infections on fish farms create conditions for parasite growth and transmission that are very different from those found in the wild. These conditions are likely to select for fast-growing, early-transmitted and more virulent pathogens, including lice (Mennerat et al. 2010, Poulin 2010, Rimstas 2011). Murray & Peeler (2005) provide an excellent discussion of this evolutionary process and suggest it might have occurred with ISA virus, which appears to have evolved independently from a wild avirulent ancestor on at least two occasions.

Kent (2011) in his report to the Cohen Commission reviews in considerable detail the pathogens to which wild sockeye are susceptible. He considers the following as potentially “high risk”: IHN virus (IHNV), three bacteria (*Vibrio anguillarum*, *Aeromonas salmonicida*, *Renibacterium salmoninarum*), and two parasites (Ich -*Ichthyophtheirus multifillis* and the myxozoan *Parvicapsula minibicornis*). Three of these are not infrequently diagnosed on Discovery Islands farms and make up the “high risk” category in the Connors analysis: *Aeromonas* causes furunculosis, *Renibacterium* causes bacterial kidney disease (BKD), and IHNV causes infectious haematopoietic necrosis. However, reports of their occurrence, either in the BCSFA Fish Health Events or the BCMAL Audits, are not associated with sockeye survival.

Two other salmon diseases of note, and which have figured prominently in the news of late, also deserve some consideration.

### ***Infectious salmon anemia (ISA)***

This is an important viral disease of farmed Atlantic salmon in some parts of the world (Europe and Chile in particular). No records of it can be found in the BCMAL or BCSFA

records, and according to M. Sheppard (pers comm) there have been “no suspect cases of ISA in BC since sampling began in 2003”. However, in his diagnostic reports on dead fish collected from salmon farms Dr. Gary Marty (fish pathologist with BCMAL) reports “classic symptoms of ISA” (see BCP002864), which according to the World Organization of Animal Health (OIE) should make any one of these what they call a “suspect case”. These “classic symptoms”, according to the BCMAL document, are sinusoidal congestion of the liver and interstitial hemorrhage/congestion of the kidney.

It is certainly worth being watchful for this disease in BC aquaculture facilities since it:

- can be carried avirulently (subclinical infections) in some salmonids (Anon 2010);
- can be transmitted indirectly in water (Anon 2010);
- may remain infective for a long period outside the host (see Rimstad 2011);
- could be vectored by sea lice (see **Routes of Transmission: Lice**, below); and
- could have been transferred to BC in eggs, despite claims to the contrary, as there is no other way for the disease to have gotten to Chile (Robertsen 2011).

### ***The so-called “Miller virus”***

A recent paper by Miller et al. (2011) provided evidence for a virus-like particle associated with early freshwater entry (by returning adults) and high pre-spawning mortality (PSM) in several Fraser sockeye stocks. Subsequent work by Dr. Miller’s group has provided increased support for the viral etiology hypothesis, perhaps involving a novel retrovirus (K Miller DFO, pers. comm.). Some have expressed the opinion that this virus is the cause of reduced Fraser sockeye survival with which the Cohen Commission is concerned. However, it is important to realize that PSM is not the cause of reduced survival as examined in this report, since Connors’ definition of “recruits” includes any mortalities due to PSM. Thus we are looking for the cause of declining survival over and above whatever effects this virus has on returning adults.

Of course this does not exonerate the involvement of this presumed virus in mortality of sockeye at earlier life stages. It is known that a high percentage of the juveniles leaving

the Fraser River show the genomic signature associated with the virus. If they then were to pick up other pathogens while passing through the Discovery Islands this could prove fatal, particularly since it is known that such co-infection can select for increased virulence of both pathogen species (e.g., Keusch & Migasena 1982, May & Nowak 1995) and retroviruses are well known to cause immunosuppression. It is also possible that the virus may be found on farmed fish (chinook are the most likely, as the genomic signature is also found in wild chinook; K Miller DFO, pers comm) further increasing viral load on any passing infected sockeye. However, the genomic signature and the retrovirus with which it appears to be associated, have not been looked for on farmed fish.

In the early 1990's a retrovirus was reportedly found in chinook salmon, associated with a disease state known variously as marine anemia and salmon or plasmacytoid leukemia (Eaton & Kent 1992, Kent & Dawe 1993, Stephen et al. 1996) causing high mortality rates in farmed chinook. Dr. Miller (pers comm) cannot rule out the possibility that it is the same virus. The disease was shown to be transferrable to sockeye salmon by injection of a tissue homogenate from infected fish (Kent & Dawe 1990, Newbound & Kent 1991). However, a viral etiology of marine anemia has not been confirmed and the hypothesis has not been universally accepted. According to Kent (2011) the microsporidean *Nucleospora salmonis* "is associated with a disease indistinguishable from plasmacytoid leukemia", and may well be the actual causative agent. In any case, as the putative virus is difficult to detect with histology alone (K Miller pers comm) it may be worth screening fish farm "fresh silvers" (recently dead fish without apparent cause) for retroviruses, something which apparently is not currently done (M Sheppard pers comm).

### ***Routes of transmission***

If pathogens are involved in the decline of Fraser sockeye stocks, this then begs the question of how viruses or bacteria move from farmed Atlantic salmon to wild sockeye. There are several possibilities, some of which have been noted in passing in earlier sections, and some of which are more likely than others:

#### a. direct horizontal transfer

All three of the high risk diseases found in the BCFSA and BCMAL records (i.e., IHN, furunculosis and BKD) are known from both wild and farmed fish in BC and can be transmitted to new hosts in water (Hammell et al. 2009). As these authors make clear, “simply because a pathogen can be waterborne, does not mean it is capable of moving from fish inside a net pen to fish outside.” But if Atlantic salmon get infected by passing wild fish, as fish farmers are quick to claim, then it is extremely unlikely that disease will not transfer in the opposite direction as well.

To take one example, evidence suggests that IHNV may spread from farm-to-farm in the water (Saksida 2006). If so, there is no reason it could not be spread to passing sockeye. Sockeye are known to be highly susceptible (Yasutake & Amend 1972, Amend & Nelson 1977) and post-smolts in seawater can pick up the virus from cohabiting Atlantic salmon (Traxler et al. 1993).

The large number of farmed salmon in the Discovery Islands region can be expected to discharge millions of virus particles and bacteria into the water column, where they could infect passing juvenile sockeye. The percentage of farmed fish that die from a disease (and thus show up in BCFSA and /or BCMAL records) may represent only a small proportion of those infected with the pathogen and swimming around in their pen, apparently well but shedding infective particles. If sockeye are attracted to the farms by lights, waste food, etc., this could increase their rate of contact with these pathogens.

#### b. benthos

Faeces from farmed fish could contain pathogens that could potentially be picked up by wild fish outside the cage. For example, *Renibacterium* (the causative agent of BKD) can survive in faecal matter for up to 21 days (Hammel et al. 2009). Also, as noted above, there will be impacts on benthic species beneath the farms, perhaps favouring some that are intermediate hosts of parasites that infect fish later in the parasite’s life cycle. An

example is the myxosporean parasite *Kudoa*, whose intermediate hosts are worms found in the benthos (Tlustý et al. 2001, p 177). *Capitella capitata* (a polychaete worm) is known to flourish in the low oxygen and organically enriched conditions created by wastes below farms (Brooks 2001), and may be the intermediate host of *Kudoa thyrsites*, which causes muscle liquefaction after death (Kent et al. 1994) but apparently no mortality (Kent 2011).

### c. escapees

Escaped farm salmon may be a significant source of furunculosis infections in wild Atlantic salmon (Hastein and Linstad 1991, Johnsen & Jensen 1994). However, there is no reason to believe that the disease status of escaped fish is any different from that of the fish in the pen, which would be a greater source of infection to wild sockeye. But it may be that a higher percentage of the fish in the pen (and thus of those who escape) are infected than health records would indicate. As Hammel et al. (2009) say: “A variety of salmon diseases can result in a carrier state in asymptomatic fish (such as furunculosis, bacterial kidney disease, infectious hematopoietic necrosis, infectious salmon anemia, infectious pancreatic necrosis). We found no studies that examined what proportion of escaped salmon includes asymptomatic disease carriers and how many survive long enough to transmit their pathogen to another fish. Asymptomatic, persistently infected fish typically do not shed as much pathogen as a sick fish, but their shedding happens over an extended time period”

However, the number of escaped farm salmon (even if grossly under-reported) is a small percentage of those in the farm pens, and they will be spread throughout a far greater water volume. Thus it is very unlikely that they would be a substantial source of disease for wild sockeye, relative to other routes of transfer.

#### d. lice

Both species of lice are blood-sucking ectoparasites whose feeding activities leave holes in the integument of their hosts, and sometimes cause considerable surface tissue damage. The lice are also known to carry pathogens on their surface (or in their gut), and be capable of transferring these to their host, perhaps through these feeding wounds. They thus may be vectoring disease from farmed to wild fish (see Table 1). This is most likely to occur when the motile stages of the lice switch hosts, something that is most common for *Caligus* but also occurs not infrequently in *Lepeophtheirus* (Connors et al. 2011). Even in the absence of a louse, the epidermal damage caused by a past infestation may make the fish more invadable by bacteria or viruses.

It has also been reported that infection with sea lice and lice treatment may lead to suppression of the immune system (Mustafa et al. 2000), suggesting another possible way in which sea louse infections could increase the likelihood of disease in otherwise healthy wild fish.

#### e. processing plants

Facilities where farmed fish are processed can be point sources for pathogen infection of passing wild sockeye, particularly if the wastewater (called “blood water”) is not filtered and/or disinfected. There are at least two such facilities in the Discovery Islands area (Walcan, just north of Cape Mudge on Quadra Island, and Brown’s Bay) and one just to the north, in Port Hardy (Alpha). The Walcan facility is known to be discharging large volumes of untreated effluent into the water (Morton 2010 p38, Price et al. in review) and could be a source of pathogens and lice (Price et al. 2011). This concern has been raised in the past (NovaTec Consultants Inc. 2004), and a number of recommendations made to address the problem, but whether these were ever acted upon is unknown. Since these plants apparently process fish from non-local farms (e.g., Walcan processes Grieg fish from Nootka Sound on the west coast of Vancouver Island) they would confound the spatial analysis by transferring pathogens from one fish health zone to another.

**Table 1. Examples of vectoring of pathogens ("transmit"), or the potential for vectoring them ("carry") by sea lice**

Pathogen or disease	Evidence supports ability to	Source
Infectious pancreatic necrosis virus (IPNV)	carry	Johnson et al. 2004
Salmonid alpha virus (SAV)	carry	Karlsen et al. 2005
ISA virus (ISAV)	transmit transmit* transmit	Nylund et al. 1991,1993, 1994 Hammell & Doho 2005 Rolland & Nylund 1998
IHN virus (IHNV)	carry	Stull et al. 2010
Furunculosis ( <i>Aeromonas salmonicida</i> )	carry transmit carry transmit	Nese & Enger 1993 Nylund et al. 1993 Lewis et al. 2010 D Barker pers comm
Bacteria ( <i>Tenacibaculum maritimum</i> , <i>Pseudomonas fluorescens</i> , and <i>Vibrio</i> spp.)	carry	Barker et al. 2009
Microsporidian <i>Paranucleospora theridion</i>	carry	Nylund et al. 2011

\* Lack of sea lice control has been linked to increased risk of ISA transmission (Hammell and Dohoo, 2005). As an indirect measure of reduced level of sea lice infestations, the number of delousing bath treatments was used as the variable in which greater bath frequency was considered an indication of a more aggressive sea lice control policy. Greater than two delousing bath treatments was protective against ISA outbreaks compared with one or no treatment (see also McClure et al. 2005)

## THE FUTILITY OF LOOKING FOR THE CAUSE OF FRASER SOCKEYE DECLINES

Much of the public, and the press, seem to think that the present report, and the Cohen Commission in general, will identify the cause of the sockeye salmon decline, and in particular the return failure of 2009. This is a naïve expectation. Nature is complex and factors do not act in isolation on the population dynamics of any species. Pathogens from fish farms are just one factor among many that may influence the mortality rate of juvenile sockeye. There are several ways in which these various factors may interact, some of which have been noted in passing in earlier sections of this report.

Small and/or diseased sockeye from the Fraser River may be more susceptible to anything they might pick up while passing the farms. This was mentioned above with respect to the putative “Miller retrovirus”. Beamish et al. (2010) reported that sockeye experienced poor growth in the Strait of Georgia in 2007 which could exacerbate this effect, as these fish would have been smaller than normal when reaching the Discovery Islands. This could be a factor in the especially low 2009 returns.

Should the young sockeye be infected at sub-lethal levels while passing the farms they may be more susceptible to starvation in years when food is less available (e.g., when sea surface temperature is above average) and/or competitors more abundant. These effects could occur hundreds or even thousands of kilometers from the Discovery Islands and could explain the interaction with pink salmon abundance detected in the Connors analysis. It could also explain the findings of the tracking study reported by Welch (2010).

Welch summarized data from his POST (Pacific Ocean Shelf Tracking) experiments (Welch et al. 2009, 2011) which suggest that the region of high mortality of sockeye smolts in 2007 was shortly after they passed through the Discovery Islands and Queen Charlotte Strait, and likely occurred in Queen Charlotte Sound. This is not inconsistent with the Connors finding, and may simply suggest that passage by fish farms reduces the sockeyes’ ability to compete for food in both the short term and in the open ocean in later

life. Indeed, conditions were apparently quite poor for sockeye feeding in Queen Charlotte Sound in 2007 (McKinnell et al. 2011, R Tanasichuk DFO, pers comm). Interestingly, Welch (2010) considers it a possibility that the die-off could have been a delayed effect of disease transfer from farms as the sockeye passed them on their outbound migration.

Fish stressed by other factors could be more vulnerable to any pathogens that may be originating on the farms. Snieszko (1974) reviews several examples of the interactive effects of pathogens and environmental stressors (temperature, eutrophication, sewage, metabolic products of fishes, industrial pollution and pesticides) on the development of disease. More recently, it's been shown that low pH (i.e., acidified) water may affect the ability of Atlantic salmon smolts to resist sea lice infestation (Finstad et al. 2007). So water quality in the Fraser River or in the Strait of Georgia could influence the impact of any pathogen picked up while passing the farms.

Just as sockeye weakened by pathogens or lice (and/or compromised by resulting smaller body size) will be more vulnerable to starvation (and vice versa), so too may they be more vulnerable to predators. These include spiny dogfish (Beamish et al. 1992, Beamish and Neville 2001) and several other species discussed in the Christensen and Trites (2011) Cohen Commission report, including sea birds, salmon sharks, daggertooth, sablefish, arrowtooth flounder and Humboldt squid. And although no single species seems likely to be the culprit in the "Murder on the Orient Express", as Christensen and Trites (2011) put it, their cumulative impact could be considerable. One way this increased predation effect of farms might operate – at least in juvenile sockeye - is through reduced swimming ability, which presumably affects their ability to escape a predatory attack. Both sea lice in sufficient numbers (Mages & Dill 2010) and BKD (Jones & Moffitt 2004) are known to have this effect on their salmonid hosts.

In some species of Pacific salmon, reduced early growth is associated with lower marine survival (e.g., coho salmon; Beamish et al. 2004). It is also well established that larger sockeye smolts survive better than smaller ones, based on comparisons at both the

population (Koenings et al. 1993) and individual levels (Henderson & Cass 1991). So perhaps a slight reduction in growth/ size resulting from a farm effect in early marine life may compromise future survival, through any of the mechanisms just discussed.

Parasites and diseases can also influence predation mortality in a more indirect fashion, by causing infected individuals to take greater risks to obtain the extra food they need to offset the energetic demand of dealing with infection (Krkosek et al. 2011), and this behaviourally-mediated effect will be more apparent when competition is important and the fish are more energetically stressed as a result (see Dill et al. 2003 for a discussion of these sorts of behaviourally-mediated indirect effects in marine ecosystems).

Other sorts of indirect ecosystem effects can also be operating in the farm-wild salmon system. For example, herring can buffer sockeye from predation by seabirds (Scheel & Hough 1997), so a decline in herring stocks caused by *Caligus clemensi* (a host generalist) could increase sockeye mortality rates. Similarly, reduced herring populations (or populations of any alternative prey species) could mean that predators would switch their attention to sockeye smolts, again reducing their survival. Conversely, an increase in alternate prey availability, perhaps through local energy and materials inputs near farms, could lead to larger predator populations and more sockeye consumption. These hypothetical examples are intended to illustrate the complexities in the real world system in which farms and wild sockeye are embedded, and to caution against any simplistic single-factor explanation.

## SUMMARY

The relationship between farm production and Fraser sockeye survival in the long-term data set suggests that the farms are having some sort of negative impact on wild salmon productivity, most likely in concert with other factors in the marine environment.

However the quantity and quality of the individual farm data available for detailed analysis makes it impossible to zero in on the mechanism(s) responsible, although the most likely candidate is disease transfer. None of the other possibilities considered (lice, benthic and pelagic impacts, escapes, etc.) are likely to be sufficient, alone or in concert, to cause either the long-term population declines or the especially low returns in 2009.

The biggest problem facing this analysis is the fact that the impact of farms could only be examined for a few year classes (brood years) of wild sockeye, because:

- good fish health records were only available from 2003 (2004 for lice), and
- complete sockeye escapement data were only available up to 2009 (the 2004 brood year).

Such a short time series barely even captures the 4-yr cycle of such sockeye stocks as the Adams River population. The variability in survival from one year to the next (viz. 2009 to 2010) illustrates the difficulty of establishing or predicting relationships from a short time series. Korman (2011) has discussed this problem in some depth in his report to the Commission.

Although the Commission thought that they were going back far enough (10 years) to capture enough history of farm-wild salmon interactions, this unfortunately was not the case, and an adequate analysis was therefore impossible. It is recommended that the sort of analysis conducted by Dr. Connors be repeated annually, perhaps by Fisheries and Oceans Canada scientists, to see if a pattern begins to emerge when more wild sockeye year classes can be included. This will require that a single consolidated database be maintained of farm production, lice, disease and mortality on a farm-by-farm basis. I would also recommend that this be accessible to *bona fide* researchers, if not to the public in general.

It is very important to realize that just as we cannot claim that some specific aspect of salmon farming is part of the cause of Fraser sockeye population declines, we cannot (for the same reasons) say that it is not. In fact, the evidence from the long-term data set is compelling that the farms are implicated in some way. Certainly it is the case that there are negative environmental impacts from wastes and chemicals, and the aquaculture industry can and should continue in its efforts to become “greener”. Some management options to achieve this goal, and to reduce the risks to wild salmon, are briefly considered in the next section, followed by gaps identified in our knowledge that need to be filled in by further research.

## MANAGEMENT OPTIONS

There are a number of salmon farm management methods that could mitigate risk to Fraser sockeye salmon.

1. More frequent fish health audits and better diagnostic procedures could reduce the prevalence of disease on farms, and its transmission to wild sockeye.
2. Lower densities of fish in the farms might reduce the likelihood of disease epidemics, and limit the supply of pathogens being exported from the net pens. As McVicar (1998) says: “the higher densities of fish within a fish farm tend to be conducive both to the spread of infection and to alteration of the susceptibility of the fish through stress effects on the fish.”
3. Scheduling of harvesting could be planned and coordinated regionally so that at least adult Atlantic salmon (who likely present the greatest risk to wild fish) are not present in the farms at the time most juvenile Fraser sockeye pass them (May and June).
4. Farms could be relocated to other areas of the coast, although this might simply shift the problem onto other stocks of wild fish. If the farms were moved out into the open ocean, where only larger wild fish would be exposed to them, this would reduce risk to Fraser sockeye. Not being an engineer, I do not know whether this is feasible.
5. As was done in the Broughton Archipelago, coordinated and timely application of chemotherapeutics such as SLICE® would reduce sea lice populations during the critical May-June wild sockeye migration period. If lice are important as disease vectors, or facilitate pathogen infection in other ways, this could reduce disease transfer. However, this may only be a short-term solution if sea lice become resistant to emamectin benzoate (or other chemotherapeutants), as they have done elsewhere (e.g., Lees et al. 2008). Genetic analyses suggest this may not be a major concern in BC, at least in immediate future, due to extensive panmixis creating homogeneous population structure throughout

the northern Pacific (Messmer et al. 2010). Indeed, based on a relatively short-term analysis, a decline in efficacy of SLICE® is not evident on BC salmon farms (Saksida et al. 2010).

6. The most obvious solution to the risk of pathogen infection of wild sockeye (and to several other environmental issues as well) is closed containment - either on land or in the water. With proper effluent disinfection and water treatment, this could prevent disease transfer to wild sockeye. Closed-containment technology has advanced considerably in recent years and appears to be both technologically and economically feasible as an alternative to open net pens. This conclusion was reached both by Wright and Arianpoo (2010) in their review for the Save Our Salmon Society SOS (see [http://www.saveoursalmon.ca/files/May\\_draft\\_05-04-10.pdf](http://www.saveoursalmon.ca/files/May_draft_05-04-10.pdf)), and in a report from DFO's Science Advisory Secretariat (Boulet et al. 2010), although the latter report was considerably less optimistic about the economic feasibility. In our region several such farms are already in operation or in the late stages of development:

1. Sweet Spring, WA – coho
2. Larry Albright, Langley – sockeye
3. Swift Aquaculture, Agazzi – coho
4. Namgis First Nation, pilot plant near Nimpkish hatchery – Atlantics
5. Agrimarine, Middle Bay, Vancouver Island – chinook (floating solid wall tank)

It is also my understanding that Marine Harvest, one of the major farm stakeholders in BC (and globally), is planning a pilot project of their own.

In considering these options it would be wise for managers to keep in mind the conclusions of Frazer (2008), based on an analysis of his mathematical model of farm pathogen – wild fish interactions: “Declines of wild fish can be reduced by short growing cycles for farm fish, medicating farm fish, and keeping farm stocking levels low. Declines can be avoided only by ensuring that wild fish do not share water with farmed fish, either by locating sea cages very far from wild fish or through the use of closed-containment aquaculture systems.”

## STATE OF THE SCIENCE: KNOWLEDGE GAPS

1. Detailed information on migration behaviour and pathways of sockeye smolts through the Discovery Islands area;
2. The attraction of sockeye juveniles (and other species) to the net pens;
3. The cumulative impact of repeated exposure to poor water quality and pathogens (including lice) when passing multiple farms in succession;

The following set of gaps relate to a conclusion in the Hammell et al. (2009) report for the World Wildlife Fund, namely: "In general, information on the movements of fish pathogens in the marine environment, the transmission ecology affecting wild and farmed fish, and the viability of pathogens shed in the marine environment is usually lacking."

4. The possible presence of a retrovirus on farmed Atlantic and chinook salmon, and the relationship (if any) of this to the causative agent of salmon leukemia (aka marine anemia) found in chinook;
5. The infective state of apparently healthy salmon in net pens (i.e., their potential to be sources of shed viruses and bacteria);
6. The potential for lice to act as vectors of high risk pathogens causing such diseases as BKD, IHN and furunculosis;
7. The impact of both species of lice (*Lepeopthierus salmonis* and *Caligus clemensi*), and of other pathogens, on feeding and anti-predator abilities and survival of sockeye smolts;
8. The potential for bloodwater from processing plants to be a source of infection;
9. The evolution of resistance and/or increased virulence in sea lice treated with SLICE®;

10. Interactions of lice and other pathogens with other stressors in the marine environment, such as low food availability and pollutants;

11. Disease incidence and levels in wild sockeye;

12. The potential for biological control of pathogens on farms (perhaps using mussels, who have been shown to effectively remove *Renibacterium salmoninarum* from seawater; Paclibare et al. 1994).

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## GLOSSARY

**Abundance:** mean number of individuals of a particular parasite species per host examined); it equals mean intensity times prevalence.

**Amphipods:** a group of small, mostly planktonic crustaceans belonging to the Order Amphipoda.

**Anoxic:** lacking in oxygen.

**Antifoulant:** a substance applied to structures and nets to prevent fouling by barnacles, mussels, etc.

**Avirulent:** not virulent, not extremely infectious.

**Benthic:** of or relating to the seafloor.

**Biomagnification:** the capacity of aquaculture populations of host fish to locally amplify or magnify the potential of a parasite or pathogen to infect adjacent wild populations of host fish.

**Chemotherapeutant:** chemical such as emamectin benzoate (SLICE®) that's used to treat sea lice infestations on farmed salmon.

**Compensatory mortality:** animals dying from the compensatory factor would have died anyway of some other cause.

**Copepods:** small marine and freshwater crustaceans of the subclass Copepoda. Sea lice are parasitic members of this group.

**Copepodid:** the free-living, non-feeding, planktonic larval stage of the sea louse; this is the stage that infects salmonid hosts.

**Diel migration:** a pattern of migration, normally vertical, that aquatic organisms perform on a daily cycle

**Disease:** a host fish is diseased if it is behaviourally or physiologically compromised (e.g., by sea louse infestation)

**Ectoparasite:** a parasite, like a sea louse, that lives on the exterior of its host.

**Etiology:** the cause of a disease.

**Euphausiids:** small pelagic shrimp-like crustaceans of the Order Euphausiacea, also known as krill.

**Eutrophication:** the addition of artificial or natural substances, such as nitrates and phosphates to an aquatic system, often leading to a great increase of the phytoplankton.

**Fallow:** the period of a few weeks between harvesting cycles, when fish are absent from a site after harvesting and before the next restocking; also, the practice of site rotation where a site may be left empty for one or more years to allow the sediments to recover.

**Genomic signature:** characteristic pattern of gene expression, revealed on a microarray - an arrayed series of thousands of microscopic spots each containing tiny amounts of a specific DNA sequence used as a probe to screen large numbers of samples.

**Horizontal transmission:** the direct transfer of an infection from fish to fish

**Immunosuppression:** a reduction in the ability of the immune system to deal with infection, increasing the susceptibility of the host to other pathogens

**Intensity:** number of individuals of a particular parasite species in/on each infected host.

**Meta-analysis:** a statistical procedure for combining the results of several studies testing the same hypothesis.

**Microsporidia:** a phylum of spore-forming unicellular parasites now classified as fungi. Microsporidia are restricted to animal hosts, and all major groups of animals host them; they are responsible for common diseases of fish.

**Myxosporidian:** any parasite of the phylum Myxosporidia, also called Myxospora; primarily parasites of fish, they also attack amphibians and reptiles.

**Nitrate:** an ion consisting of one atom of nitrogen and three atoms of oxygen (NO<sub>3</sub>).

**Panmixis:** random mating within a breeding population, resulting in a high degree of genetic uniformity.

**Pathogen:** an agent (like a virus, a bacteria or a sea louse) that causes disease.

**Pathogenicity:** the ability to cause disease.

**Pelagic:** of or relating to the open ocean, as opposed to the bottom.

**Phytoplankton:** small planktonic organisms, mostly plants, that manufacture their own food by turning sunlight into chemical energy; this is called autotrophy.

**Plasmacytoid:** innate immune cells that circulate in the blood, ready to respond to pathogens, but not specific to any particular type.

**Polychaetes:** a class of segmented worms, generally marine; each body segment has a pair of fleshy protrusions called parapodia that bear many chitinous bristles, called chaetae.

**Power (statistical):** the probability that a statistical test will reject the null hypothesis of no effect when the null hypothesis is in fact false, in other words reach the correct decision.

**Prevalence:** percentage of individuals of a host species infected with a particular parasite species.

**Producer:** an organism (such as a plant) that manufactures its own food by turning sunlight into chemical energy.

**Retrovirus:** any of a family of single-stranded RNA viruses containing an enzyme that allows for a reversal of genetic transcription, from RNA to DNA (rather than the usual DNA to RNA); the newly transcribed viral DNA is then incorporated into the host cell's DNA strand for the production of new RNA retroviruses; the family includes the AIDS virus (HIV).

**Salmonid:** members of the family Salmonidae, including salmon, trout, charr, grayling and whitefish.

**Smolt:** a young salmon at the stage when it becomes covered with silvery scales and migrates from freshwater to the sea.

**Sympatric:** living in the same place.

**Vectors:** organisms that carry disease-causing microorganisms from one host to another

**Virulence:** a measure of the severity of a disease or parasite's impact on its host's fitness.

**Zooplankton:** weakly swimming and drifting planktonic organisms, mostly protozoa and small animals like crustaceans, that must consume phytoplankton (or detritus) to survive, a process called heterotrophy.

## **APPENDIX 1 – STATEMENT OF WORK**

### **Cohen Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River (the “Commission”)**

#### **“Impacts of Salmon Farms on Fraser River Sockeye Salmon: Assessment by Lawrence Dill, Ph.D. (the “Contractor”)”**

##### **SW1 Background**

- 1.1 The Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River ([www.cohencommission.ca](http://www.cohencommission.ca)) was established to investigate and report on the reasons for the decline and the long term prospects for Fraser River sockeye salmon stocks and to determine whether changes need to be made to fisheries management policies, practices and procedures.
- 1.2 An assessment of the impacts of salmon farms on Fraser sockeye is required to evaluate their importance to the ecology and survival of Fraser sockeye and to determine their role, if any, in the reductions in Fraser sockeye abundance.

##### **SW2 Objective**

- 2.1 To prepare a technical report containing a review and evaluation of the effects of salmon farms on Fraser River sockeye salmon.

##### **SW3 Scope of Work**

- 3.1 The Contractor will evaluate the linkage between salmon farm operations and Fraser sockeye spawning returns, if any. This research will consider any impacts on Fraser sockeye from sea lice exposure, farm wastes that affect benthic and pelagic habitat quality, Atlantic salmon escapees and disease.
- 3.2 Although the focus will be on Fraser sockeye, the Contractor may consider research related to other salmon species insofar as it informs the analysis with respect to Fraser sockeye.
- 3.3 The Contractor will consider the practicality and outcome of salmon farm management methods for mitigating risks to Fraser sockeye, if any. This should include consideration of using closed containment systems, scheduling of net pen harvesting to reduce contact with sea lice and disease, re-locating farms, compressing maturation schedules, optimizing densities and using SLICE and other chemotherapeutics to control sea lice.

- 3.4 The Contractor will evaluate whether the current state of scientific research on Fraser sockeye is sufficient to estimate the extent to which reductions in Fraser sockeye abundance are associated with salmon farms.
- 3.5 The Contractor will review and analyze data that will be organized and provided by Dr. Josh Korman of Ecometrics Ltd, as described in the Statement of Work “Impacts of Salmon Farms on Fraser River Sockeye Salmon: Assessment by Josh Korman, Ph.D.” attached here as Annex A.

#### **SW4 Research methods and sources of information**

- 4.1 While the primary source of information will be peer-reviewed journal articles and technical data, the Contractor may also draw on non-peer reviewed reports and articles (“grey” literature), as well as interviews with individual scientists, representatives of the salmon farming industry, commercial, sport and First Nations fishers and NGOs. All sources of information must be cited in the report. The Contractor will consider all available sources of information, including international sources where relevant.

#### **SW5 Deliverables**

- 5.1 The Contractor will participate in a Project Inception Meeting to be held within 2 weeks of the contract date in the Commission office. The meeting will involve Commission scientific staff and 2 researchers, Dr. Don Noakes and Dr. Josh Korman, who are also being engaged by the Commission to evaluate and report on salmon farm impacts.
- 5.2 The Contractor will participate in a second Project Development Meeting to be held on, or around March 15, 2011 involving Commission scientific staff and Dr. Don Noakes and Dr. Josh Korman. The objective of this meeting is to ensure the integration of the statistical analysis into the Contractor’s work product.
- 5.3 The main deliverables of the contract are two reports evaluating the effects of salmon farms on Fraser River sockeye: 1) a progress report, and 2) a final report.
- 5.4 The Contractor will provide a Progress Report (maximum 20 pages) to the Commission in pdf and Word formats by May 1, 2011.
- 5.5 The Contractor will provide a draft Final Report to the Commission in pdf and Word formats by June 1, 2011. The draft Final Report should contain an expanded Executive Summary of 1-2 pages in length as well as a 1-page summary of the “State of the Science”. The Commission may obtain and forward comments on the draft Final Report to the Contractor by June 15, 2011. The Contractor will provide any revisions to the Commission by June 30, 2011.

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

## APPENDIX 2 - REVIEWS AND RESPONSES

Report Title: Impacts of Salmon Farms on Fraser River Sockeye Salmon

Reviewer Name: Rick Routledge

Date: June 14, 2011

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

This report provides a balanced overview of the potential for aquaculture impacts on Fraser River sockeye salmon. I found no serious weaknesses in the report other than those stemming from the short time series available for relating anything but overall farm production to Fraser sockeye productivity.

### **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?**

I found the conclusions to be reasonable and scientifically defensible.

### **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 are other analyses that could have been conducted, but I do not believe that they are worth pursuing at this stage. In particular, time series data can often be analyzed in several markedly different ways. In this instance, I anticipate that one could custom-design a more sophisticated way of analyzing the time-series data on the relationship between fish farm production and Fraser sockeye productivity that would combine the advantages of the two competing analyses conducted for this and the Noakes report. Nonetheless, it seems unlikely to me that much would be gained from doing this beyond confirming the complementary conclusions to the existing analyses. (I have provided a summary of what I believe to be the appropriate conclusions to be drawn in my response to Item 6 below.) I do not anticipate that any sweeping new insight would be generated from such an effort, and therefore do not recommend any further refinement to these analyses.

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

I found the recommendations to be supportable and reasonably comprehensive in terms of both management changes and information needs. In addition though, I would encourage the Commission to consider carefully a more overarching concern – namely the administrative, scientific, and regulatory framework under which the reforms should be implemented. From my perspective as a research scientist, I would particularly encourage the Commission to explore changes to the current structure that would firmly and clearly channel government support for research on conservation issues through a framework that keeps the researchers at arm's length from vested interest groups. There has been much commentary on perceived weaknesses in the current structure within Fisheries and Oceans

Canada, especially since the decline in northwest Atlantic cod<sup>1</sup>. Indeed, this concern extends well beyond the confines of Fisheries and Oceans Canada. The general issue of insulating scientific inquiry from vested interests has a long history, and currently is of much concern in the context of climate change and the evolving nature of funding for university-based research, to name two topical examples. Such structural reforms could well have a far more lasting impact than specific recommendations for research initiatives, and I would encourage the Commission to give them serious consideration.

***LMD: I would support such a recommendation.***

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

In addition to the collection of information, I would recommend that whatever information is collected be made as generally available as possible.

***LMD: Agreed, as stated in the report (pg 34).***

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

In addition to providing more detailed commentary on this report, I would like to put forward some thoughts on how some of the conclusions of the analyses summarized in this report might be reconciled with divergent conclusions in the Noakes report.

These two reports differ substantially on the statistical analysis of a potential association between (i) aquaculture production along what is thought to be the major juvenile migration route for Fraser sockeye salmon between Vancouver Island and the mainland and (ii) the so-called productivity of Fraser sockeye salmon. Although the competing analyses are relatively complex and are based on markedly different approaches, some general conclusions seem to emerge. First, there were not enough years of data available to the analysts on potentially important risk factors on the farms to be able to detect a statistically significant influence. This is not surprising. Many influential and uncontrollable factors can impact Fraser River sockeye from when the eggs are laid until the survivors return to the approaches to the Fraser River. The notorious inaccuracy of forecasts of returning abundance provides plenty of evidence of the unpredictable year-to-year variation in adult returns. With all that uncertainty, it is not reasonable to expect definitive evidence of aquaculture impacts from so few years of observations.

Second, I believe that the somewhat divergent conclusions in the two reports

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<sup>1</sup> **Hutchings, J.A.,** Walters, C., and Haedrich, R.L. 1997. Is scientific inquiry incompatible with government information control? *Canadian Journal of Fisheries and Aquatic Sciences* 54: 1198-1210.

regarding potential correlation between the longer-term time series on farm fish production and Fraser sockeye productivity can likely be reconciled in the following way:

One analysis found a correlation. The other, which pre-screened the data to remove trends (and other potential complicating factors) did not. The divergent results of the two analyses suggest to me that the reason for the correlation is likely mostly associated with the similar longer-term trends in the two series, and not with short-term, year-to-year fluctuations. In my assessment, this dilutes the strength of the case for aquaculture impacts, but does not eliminate it. There could well be long-term aquaculture impacts that are building up over time that could be substantial but that would not produce short-term, transient fluctuations in Fraser sockeye productivity in direct response to year-to-year fluctuations in farmed salmon production.

In light of this, I am inclined to conclude the following: (i) There is some correlation between the time series most likely attributable to the similar overall trends, (ii) this evidence warrants attention, but (iii) it does not constitute definitive proof that the low Fraser sockeye productivity in many recent years is attributable to aquaculture impacts.

***LMD: As I have not seen the Noakes report, it is difficult to comment. However, it is correct that the failure to find an effect in an analysis in which the data are first de-trended may simply mean that it is the long-term impacts of aquaculture production that are important, rather than the year-to-year fluctuations.***

I agree with the conclusion in the Dill report: that, of the potential mechanisms for fish farming impacts on Fraser sockeye productivity, “the most likely candidate is disease transfer.” The potential for disease evolution, magnification and transfer is, in my assessment, serious and worthy of considerable further attention. In addition, although as suggested in the Noakes report, such a transfer may well take place without the need for a transmission vector, the role of sea lice, especially *Caligus clemensi*, with its more diverse base of host species, may well be important. (In that context, I also note that Morton *et al.* (2008) [cited in the Dill report] found this species on larval Pacific herring as well as sockeye salmon. The potential for fish farm impacts on Pacific herring warrants consideration as well.)

***LMD: Morton et al. (2008) report Caligus clemensi on larval herring. One possible way that this could impact sockeye is mentioned in the report (pg. 33). It is also possible that Caligus adults (which are highly motile) could later jump from herring to sockeye.***

Though others with more expertise in fish pathogens could provide a more knowledgeable assessment, I found the discussion of the potential impacts of

pathogens on Fraser River sockeye salmon to be balanced and reasonable. In my assessment, the potential impact of a virulent pathogen on a population which has not been previously exposed to it cannot be lightly dismissed.

The reports also differ markedly in their presentation of the evidence surrounding the infectious salmon anemia (ISA) virus. The Noakes report states, "To date, these [BC Ministry of Agriculture and Lands] audits have not found ISA in any farmed salmon." By contrast, the Dill report, after reporting the same result, adds, "However, in his diagnostic reports on dead fish collected from salmon farms Gary Marty (fish pathologist with BCMAL) reports 'classic symptoms of ISA' (see BCP002975, BCP002976, BCP002977), which according to the World Organization of Animal Health (OIE) should make any one of these what they call a 'suspect case'". Again, given the impact that ISA has had on wild (and farmed) salmon in other parts of the world, this evidence deserves considerable attention.

***LMD: The document cited in the report is now BCP002864.***

I would also like to comment on the cautions raised in the Dill report regarding the importance of watching for complex interactions between such factors as viruses, other diseases, food availability, and predator avoidance. These are indeed important complexities that must be kept in mind. Furthermore, the issue of inclement weather in the Queen Charlotte Sound area in 2007 was specifically mentioned in that report and others as a potential contributing factor, especially for fish that might also have been stressed by exposure to a pathogen. I have been contributing to a multi-year study in nearby Rivers Inlet on the juvenile sockeye salmon migration down that fjord. The early spring weather was indeed unusually stormy that year, and the first spring phytoplankton bloom in the inlet was delayed until late April. Fish migrating down the inlet later that spring were not significantly larger at the mouth of the inlet than at the head whereas in other years, the fish caught at the inlet mouth have often been double the weight of those caught at the head. Hence, coastal food chain development could well be an important contributing factor to the marine survival of Fraser sockeye and other salmon species. The combined effect of such a food shortage on fish already challenged by exposure to a pathogen needs to be considered.

***LMD: Agreed. This has been added to the list of knowledge gaps.***

In addition, Rivers Inlet sockeye, along with many other Central Coast sockeye salmon populations, have shown similar (and sometimes far more substantial) declines in abundance in recent years to those in the Fraser. And a recent paper<sup>2</sup> provides preliminary evidence that Central Coast sockeye salmon may linger in the area while other populations pass through. Hence, pathogens carried by Fraser sockeye salmon or other salmon populations originating on the South Coast may well be transmitted to Central Coast sockeye salmon and other populations there and further north. Once again, I would encourage the Commission to keep in mind that Fraser River sockeye salmon cannot be viewed

in isolation from other components of the complex ecosystems with which they interact, including other commercially and culturally important fish populations and species.

***LMD: Agreed.***

- 2 Tucker, S. Trudel, M., Welch, D.W., Candy, J.R., Morris, J.F.T., Thiess, M.E., Wallace, C., Teel, D.J., Crawford, W., Farley, E.V., Jr., and Beacham, T.D. 2009. Seasonal Stock-Specific Migrations of Juvenile Sockeye Salmon along the West Coast of North America: Implications for Growth. Transactions of the American Fisheries Society, 138: 1458-1480.

Report Title: Technical report 5D: Impacts of Salmon Farms on Fraser River  
Sockeye Salmon: Results of the Dill Investigation  
Reviewer Name: Professor A.P. Farrell, Canadian Research Chair (Tier I) in Fish  
Physiology, Culture and Conservation

Date: 20<sup>th</sup> June 2011

Abbreviations used:

DR= Dill Technical Report; 5D KTR = Korman Technical Report 5A; CTR =  
Connors Technical Report 5B; SS = sockeye salmon; FR = Fraser River

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

A strength of the DR is a writing style that is very accessible to the general reader who is a non-scientist. Lines of reasoning are presented.

The DR does not identify a smoking gun left by salmon farmers that can explain the catastrophic collapse of FR SS in 2009. In fact, despite an accessible writing style, the conclusion in the executive summary is too cryptic: “It must be understood that the short time series of data available for this investigation precluded identifying salmon farms as an important driver of the decline of Fraser sockeye.” It does not reflect clear concluding statements in the main text. Also, the somewhat more definitive statement in the closing summary then appears to be contradictory: “None of the other possibilities considered (lice, benthic and pelagic impacts, escapes, etc.) is likely to be sufficient, alone or in concert, to cause either the long-term population declines or the especially low returns in 2009.”. Greater clarity is needed for the executive summary.

***LMD: The Executive Summary has been revised to state more clearly the conclusion concerning those factors unlikely to be important. I see no contradiction regarding the short time series of data being insufficient to allow identifying any particular environmental impact of salmon farms as the driver of the long-term decline of Fraser River sockeye.***

A strength of the DR is its precautionary stance. For example: “But it must be equally understood that at this stage of our knowledge is it not possible to say they are not implicated.” The DR report identifies two main issues in this regard: a) a paucity of high-quality, long-term data for statistical analysis, and b) the danger of looking for a single factor to explain the cause of the general decline and the 2009 collapse of FR SS productivity. I support the need for continued caution and for data collection, even if our current knowledge does not point to a major negative impact of salmon farming on FR SS productivity.

For an evidence-based report, the DR contains far too much speculation centered on theoretical predictions for cause-and-effect relationships. These hypothetical ideas then lead into warnings.

***LMD: I'm not really sure what the reviewer is getting at here. In the absence of good data, I felt it imperative to use to use my judgment, based on over 40 yrs. of experience, to assess potential risks of open-pen aquaculture to wild fish. Based on his own experience, the reviewer need not agree with my conclusions.***

The scientific rigour of the DR was greatly weakened by the absence of a comprehensive and an objective consideration all available literature and information. Some of the relevant literature was omitted completely and some was rather shallowly dismissed. Some complex issues were paraphrased too simply and opposing details were left out. Lacking proper scientific coverage, the DR could easily be viewed as a highly selective and polarized opinion. This would be an unfortunate situation because it reduces the validity of some credible concerns.

***LMD: General negative comments like this are not helpful. Where specific examples are given below, I have addressed them.***

## **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?**

**I agree with the following conclusions, which need to be better summarized/reflected in the executive summary.**

benthic impacts: "... it seems highly unlikely that such local effects could impact Fraser sockeye survival to any great extent."

SLICE impacts: "... given the low initial concentration and subsequent dilution, this is considered quite unlikely."

pelagic impacts: "... fish farms are very unlikely to be the cause of the blooms.", "... appears to be little risk associated with reduced concentrations of DO associated with salmon culture.", and "... there is not likely to be changes in the zooplankton consumer populations either, and thus no change in food available to juvenile sockeye."

impacts from chemical inputs: "... unlikely to be responsible for killing Fraser sockeye, directly or indirectly."

impacts from sea lice: "The Connors analysis failed to find a significant relationship between the number of lice in the farms during the spring migration period and survival of Fraser sockeye. ... there is no evidence of a direct harmful effect in this system."

impacts from escapes: "... quite unlikely that escapes are implicated in the sockeye decline."

structural and operational impacts: "Only the transfer of pathogens is likely to be important but there is no direct evidence of this."

high-risk disease transmission impacts (*Aeromonas* causes furunculosis, *Renibacterium* causes bacterial kidney disease (BKD), and IHNV causes infectious haematopoietic necrosis): "... reports of their occurrence, either in the BCSFA Fish Health Events or the BCMAL Audits, are not associated with sockeye survival."

***LMD: The Executive Summary needs to be fairly short. I feel the (revised) statement there that summarizes these conclusions is sufficient.***

**The focus is too narrow**

The DR leans heavily on the results of the CTR, but appears to be largely dismissive of the KTR, doing little in the way of critically examining its conclusions such as: "Negative effects of salmon farms on returns of Fraser River sockeye between 2002 and 2010 were not apparent based on a qualitative comparison with salmon farming data provided in this report." Likewise, the Executive Summary of the CTR concludes: "My analyses found no statistical support for a relationship between these aquaculture variables and sockeye survival anomalies." Clearly the two technical reports agree in some ways.

***LMD: The Connors report is more rigorous and uses a more appropriate statistical methodology, so its conclusions are the ones addressed in the my report.***

Furthermore, the KTR contains some important general trends and quantitative information on fish farming that run counter to a negative impact on FR SS productivity (eg the average number of sea lice on farmed fish farms has halved while FRSS returns have continued to decline between 2004 and 2009). Ignoring these quantitative data may have resulted in the loose language that sometimes appears in the DR. For example: "Three of these are not infrequently diagnosed on Discovery Islands farms and make up the "high risk" category in the Connors analysis...". The use of "infrequently" is far too vague in this instance. The DR should not resort to qualitative statements when quantitative information is available. It does not adequately recognize a decrease of ~6 high risk-disease events per year over the period of analysis running counter to an overall contribution to the decrease in FR SS productivity during the same period.

***LMD: The quantitative data are available in the Korman report, and the analyses of them are presented in the Connors report. The purpose of my report was to summarize these and place them in the larger context, not to repeat them. The conclusion of the KTR regarding sea lice, and the observation of a decline of disease incidence through time, are not inconsistent with the findings of my report.***

**The discussion of the so-called "Miller virus" does not acknowledge that this research is in the early stages of scientific discovery.**

There is no concrete evidence that a) there is a virus causing the published genomic signature in SS (rather the genomic signature has virus-like qualities), and b) exactly where this genomic signature is being triggered. The research does not support the speculative media claim that a virus is being released from farmed salmon and killing FR

SS, which appears to be the starting point for the DR. A more accurate reporting of results and their limitations is needed.

*LMD: I thought I had made it clear that the “so-called” virus was “putative” and only “virus-like”, although recent work seems to support its existence (K Miller, pers comm). I did not start with the “speculative media claim”; in fact I begin by discounting its association with pre-spawning mortality as being the cause of the decline in Fraser R sockeye productivity the Commission seeks to understand. However, I suggest that it should be looked for in farm fish to see if it is involved in some fashion earlier in the sockeye life history.*

**Interpretation of available data was inadequate regarding sea lice impacts.**

The DR does not comprehensively examine the available literature and, in the absence of studies of lethality and sublethal effects of sea lice on SS, it draws heavily on past works performed on pink salmon in the Broughton Archipelago.

The following excerpt from the DR is an example of a rather limited, polarized and unbalanced treatment of the available literature. “These fish are quite small, and lack protective scales, and have not been prepared by their evolutionary past to deal with an infection at this early stage of their life (normally any infection would not occur until the fish meet the returning adults later in the summer, when the juveniles are less vulnerable; Krkošek et al. 2007b). If the intensity of infection is high enough the young wild salmon can die, either from the direct effect of the parasite, or from indirect effects such as secondary infections or predators (Krkošek et al. 2011). If enough individuals die, and these would not have died from some other cause in the absence of lice, then the population will decline, and there is evidence that this has happened to pink salmon in the Broughton (Krkošek et al. 2005, 2006, 2007a).”

Omitted from this simple explanation of a complex issue is the fact that Krkosek et al. (2007) were criticized for serious errors and omissions.

*LMD: These critical papers were referenced in the text (pg. 11). But it was not my intent to exhaustively review impacts of lice on salmon elsewhere, only to point out that such problems have been identified elsewhere, in order to set the stage for a consideration of impacts on Fraser sockeye.*

Even Krkosek et al. (2009) have subsequently tempered their earlier prediction of a total collapse of pink salmon populations (Krkosek et al. 2007) because they had failed to consider the ability of juvenile pink salmon to readily shed sea lice. Yet, Krkosek et al. (2009) is not cited in the DR. This deliberate omission of relevant literature creates the illusion of selective pruning.

Therefore, it is important to more fully explore the results of Krkosek et al. (2009), who exposed juvenile chum and pink salmon to *Lepeophtheirus salmonis* for at least 30 days. The result was that: “In all cases, the louse populations showed a decline to almost zero abundance after 30 days.” Inspection of the actual data shows a near zero louse

abundance could occur even earlier. This result led to the conclusion that “louse populations rapidly decline following brief exposure of juvenile salmon, similar to laboratory study designs and data.” Therefore, I am astounded that information on shedding of sea lice by juvenile salmon was available in the literature (Morton and Routledge 2005; Jones et al. 2006) but ignored for the mathematical predictions contained in Krkosek et al. (2007), despite some of the information being generated one of the coauthors. The DR perpetuates this omission of important factual information.

The results of Krkosek et al. (2009) also clearly state that “there were few fish mortality events”, and that “the mortality observed later in the trials - particularly after 25 days was associated with emaciated fish... a near zero lice presence ... and few louse-associated scars indicating that some fish had starved”. Clearly, an 80% sea-lice induced mortality, as proposed by Krkosek et al. (2007), was not supported.

Krkosek et al. (2009) correctly suggest that timing of exposure to sea lice is critical factor, which in the case of Broughton pink salmon is potentially a “two to three month migration of juvenile salmon past multiple salmon farms”, and leads to the likelihood of reinfection that offsets the ability of pink salmon to shed sea lice. Juvenile sockeye salmon out-migrating past salmon farms on the east of Vancouver Island do so much faster (“They move through this area relatively quickly” as stated in the DR). Thus, the likelihood of reinfection is reduced proportionately for FR SS.

***LMD: This is an interesting point and I have added it (pg. 23). But note that a main conclusion of the modeling in Krkošek et al. (2009) was to show that when multiple re-infection occurs (such as when fish sequentially pass several farms), the lice can overwhelm the salmon even if each louse has a limited residence time before being shed. And this does not even consider sub-lethal effects, such as increased vulnerability to predators. As a general comment, this reviewer seems to spend an inordinate amount of time on the sea lice issue, even though I clearly say it is probably not the driver of Fraser sockeye declines.***

In view of this, I do not see how the following conclusion can be reliably made by the DR: “Here in British Columbia, there is considerable evidence for impacts of sea lice on wild pink and chum salmon juveniles in the Broughton Archipelago.”

***LMD: This refers in the first instance to negative impacts on individual fish, for which there can be little doubt. Whether populations are impacted is another question, for which there is considerable evidence, including a paper in review co-authored by myself.***

Similarly, I do not agree with the broader statement: “... lice are known to have deleterious effects on salmonid hosts elsewhere (Europe, the Broughton Archipelago)...” With regard to European studies, the DR notes: “In a very interesting experiment, hatchery Atlantic salmon protected with an anti-louse treatment prior to release survived at a slightly greater rate than untreated ones (Hvidsten et al. 2007), suggesting lice as the cause of mortality.” However, this précis does not fully disclose the results.

Hvidsten et al. (2007) considered the impact of sea lice on out-migrating, ranched Atlantic salmon at a much higher abundance and intensity (>10 lice per fish), and in a far more intensive Atlantic salmon farming area (Norway). To summarize this work, a 3-year (1996-1998) tagging and recapture technique was used to study the effect prophylactic feeding of approximated 3,000 Atlantic salmon smolts each year with an undisclosed substance “EX”, which decreases sea lice for up to 16 weeks. Comparisons were made with 3000 control fish similarly tagged and released year. Linear regression analysis on the results capture of 1 sea winter post-smolt capture revealed a no significant effect for all 3 years combined. When each year was analyzed individually, statistical significance was reached ( $P=0.05$ ) only for the 1998 data. Possibly because of low % recapture (0.51 to 2.2%) and the lack of a treatment effect in 2 out of the 3 years, the authors wisely suggest to “... interpret the data with caution”.

A similar, but more comprehensive experiment was presented by Jackson et al. at the 2010 Sea Lice Conference held in Victoria, BC and is now in press after being peer-reviewed for the journal *Aquaculture*. This study, based on the west coast of Ireland where there is intensive Atlantic salmon farming, also examined the consequence of prophylactic feeding SLICE to ranched Atlantic salmon smolts for 7 days, which protected them from sea lice for 9 weeks while they swam well beyond the salmon farms. The study lasted 9 years (2001-2008), using 10 releases of over 100,000 tagged salmon: 3,000-10,000 control salmon per year (total >58,000) and 3,000-6,000 treated salmon per year (total >54,000). An alarming result was the dramatic collapse in the number of returning salmon - a progressive collapse from a ~10% adult returns to a ~1% adult returns between 2001 and 2008. However, the temporal trends were parallel for the treated and control salmon and there was a small effect of the prophylactic SLICE treatment on the return. One type of statistical test (Chi-squared) showed SLICE improved returns significantly in 4/10 experiments, while another statistical test (sign test) showed SLICE improved returns significantly in 9/10 experiments. Overall, there was a small (0.8%) numeric difference in the intercepts of the linear regressions of the control and treated fish for returning adults over time. These results clearly show that Atlantic salmon returns collapsed ~10-fold despite the potential for direct and indirect sea lice effects.

In as much as, an argument still can be made for a weak negative impact of sea lice (a small improvement to the number of returning adult Atlantic salmon as a result of a prophylactic SLICE treatment), here in BC we are faced with an almost 12-fold reduction in FR SS over 20-y period (from 6 million per annum to a near collapse of 0.5 million in 2009) with at best a weak to no association between sea lice and FR SS productivity in the KTR and the CTR.

***LMD: I am grateful for the Jackson et al. reference and have added it (pg. 10); I have also noted there the small sample size and inconsistent results of the Hvidsten study.***

**Sometimes data quality are not challenged when a paper is cited.**

For example, the Abstract of a paper describing sea lice on sockeye salmon (Morton, Routledge & Krkosek (2008) *Am. J. Fisheries Management*) states: “Sea lice abundance

on sockeye salmon and Pacific herring followed the same trends, but samples sizes were too low to support formal statistical analysis.” However, the Results of the same paper contain an apparently contradictory statement: “On Pacific herring ... *L. salmonis* was essentially absent.”

***LMD: There is no contradiction. The lice on the herring were Caligus, not Leps.***

**The DR report leans far too heavily on the CTR without providing a critique of the analyses contained therein.**

The heavy discussion of the long-term analysis in the CTR seems to run counter to the warning contained therein: “However, there was large uncertainty around these estimated effects, which precludes drawing strong inference from these results.” The DR then talks of the “futility” of finding single causes but still focuses on the CRT, which only considers single factors (short-term analysis) or three interacting factors (long-term analysis).

I am not an expert in statistical analysis, but even I can recognize deficiencies in the CTR, especially the sensitivity analysis that was performed, which resulted in a curious and non-intuitive result. The CTR long-term analysis used 18 FR SS populations along with out-group populations from Alaska (5) and Washington State and BC (8), which when removed (and by limiting the analysis to just 17 FR SS populations) increased the predicted direct effect of farmed salmon production on mortality and decreased the uncertainty around the effect. Why the interaction of the pink salmon abundance on FR SS abundance should reverse depending on the inclusion or exclusion of out-groups is non-intuitive and unexplained.

***LMD: There was no “interaction of pink salmon abundance on FR SS abundance” in the Connors analysis; the analysis related sockeye productivity (not abundance) to pink salmon abundance and other explanatory variables. Presumably the reviewer is referring to the statement in the Connors report that the influence of pink salmon abundance on sockeye productivity was reversed when only FR SS (excluding the Harrison) were included in the analysis. Examination of Fig A4.1 reveals a very weak and uncertain main effect of pink salmon abundance (that is reversed). Importantly the strong effect of the interaction between farms and pinks on sockeye mortality remains.***

More worrisome, is why the Harrison River SS population was considered an out-group for the sensitivity analysis - this was the only example where a FR SS population was singled out for special treatment.

***LMD: The reason the Harrison was “singled out for special treatment” was because Connors was requested to repeat the analysis without the Harrison stock by one reviewer (M. McCallister, page 75 of Connors Report).***

There is no explanation why, or any analysis of what might occur had the Harrison River population remained.

***LMD: The Harrison River population was included in all other analyses.***

*A priori* a multitude of factors, not just the migration past a salmon farm, differ for the out-group populations. Therefore, the type of sensitivity analysis performed in the CTR did little to convince me that it was isolating a salmon farming effect. A different sensitivity analysis is suggested below.

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

In general, I agree that relevant data should be made available on an on-going basis to perform analyses on an “as needed” basis. However, I have doubts that such analyses will resolve matters in the near future, at least not beyond the present state of uncertainty claimed in the DR. Indeed, given the level of uncertainty and complexity of correlative analyses suggested in the DR, I would argue that the greatest need is for targeted interventional experiments that *a priori* can identify mechanisms. The DR reported noted the “very interesting” experiments by Hvidsten et al. (2007), but fails to suggest a similar experiment be performed on FR SS. Wild out-migrating SS could be captured prior to reaching fish farms and fed prophylactically with SLICE for a couple of days to protect them from sea lice as they swam past the farms. Then it is a 2-year wait to count surviving adults relative to control fish. This experiment would directly test the impact of sea lice. Even so, the experiment in Ireland took 9 years to collect sufficient data to show a weak effect of sea lice.

***LMD: I agree that this would be a worthwhile and interesting experiment, though logistically rather difficult as it would need to be replicated for a number of different Fraser River stocks, which (as the reviewer notes below) each face a different set of environmental challenges.***

I also agree that additional analysis could be more regional and directed, rather than global as performed for the CTR. Global data treatment is problematic in two regards. Touched on by the CTR, but not fully explored, is the fact that all FR SS populations are not created as equals. Weaver Creek sockeye are more susceptible to high temperature river mortality than interior populations (Farrell et al. 2008) and the Chilko sockeye presently stand out a ‘superfish’ in this regard (Eliason et al. 2011). Therefore, there is good reason (well beyond the concerns of negative impacts of salmon farming) that the multitude of factors affecting FR SS production is population-specific. For example, the Harrison population has continued to do well despite an overall decline. Hence, the selective omission of this population in the CTR long-term analysis is rather peculiar.

***LMD: This stock specificity is an important point to keep in mind. Repeating the approach taken by Connors on a stock-by-stock basis could be informative, however, multi-stock analyses like the analysis performed by Connors enables responses to be more easily isolated from random demographic noise and sampling. And, as noted above, the Harrison River population was not selectively omitted in the Connors***

*analysis.*

I was disappointed by the inability to step outside of the current analytic “box” to provide new fresh insight to the problem of salmon farming impacts. Instead we are left with rather disappointing statements. For example: “This report is not intended to be an exhaustive examination of all possible relationships between salmon aquaculture and sockeye salmon dynamics. Instead I chose to focus on a few plausible relationships that could be examined within the scope of this report. It is important to note that the relationships that are described in this report are correlative and do not on their own establish causation. Nonetheless, these findings should be considered a first step towards understanding the role open net pen salmon aquaculture has played in influencing Fraser River sockeye salmon population dynamics.”

***LMD: This statement comes from the Connors report, not the one under review here.***

I believe that other approaches are possible both with the present data and with different forms of experimentation.

For example, while the type of sensitivity analysis performed in the CTR did little to convince me that it was isolating a salmon farming effect, perhaps the DR should recommend a different precautionary analytical approach.

***LMD: The sensitivity analysis was not meant to isolate a farm effect it was meant to illustrate how robust the estimated influence of salmon farming on sockeye mortality was to the reference populations included and to the underling form of the stock recruit-relationship (i.e., Ricker vs. Larkin).***

Why not input hypothetical farm data to try to trigger highly reliable and predictable negative impacts on FR SS. If weak aquaculture impacts are being missed because of a very noisy background, why not artificially increase the signal to determine the level at which a major impact is triggered for the FR SS. At least this way we would know at what extreme the sky would fall in on wild salmon returns.

***LMD: I would be possible to do simulations with the model (not really a sensitivity analysis) to see the effect of increasing farm production, but I cannot see what would be gained by doing so. Some would say that the sky has already fallen (see Fig. 1).***

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

While the DR finds no major negative impact of salmon farming on FR SS productivity, it warns of the futility of looking for a single cause FR SS declines. “It is naïve to believe that the present report, and the Cohen Commission in general, will identify the cause of the sockeye salmon decline, and in particular the return failure of 2009. Nature is complex and factors do not act in isolation on the population dynamics of any species. Pathogens from fish farms are just one factor among many that may influence the

mortality rate of sockeye. ....Although some are hypothetical at this stage of our knowledge, they highlight the complexities in the real world system in which farms and wild sockeye are embedded, and caution against any simplistic single-factor explanation.”

This statement goes well beyond the objective of a report on the potential impacts of aquaculture. I think this commentary is primarily intended as a justification for the recommendations: “a well-organized farm database be maintained in an ongoing fashion by Fisheries and Oceans Canada’ and “that the sort of analysis conducted by Dr. Connors be repeated annually, perhaps by Fisheries and Oceans Canada scientists, to see if a pattern begins to emerge when more wild sockeye year classes can be included.” I am pleased to see that the DR recommends such complex analyses are left to experts.

*LMD: I disagree that this “goes well beyond the objective of a report on the potential impacts of aquaculture”. I am trying to point out that the impacts of aquaculture cannot be considered except in relation to a host of other factors affecting survival, and that to do so is folly. It was not intended to justify the recommendation to maintain on-going data base (something the reviewer apparently supports) and I am at a loss to see how he connects the two.*

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

If there is a desire to prove cause-and-effect negative impacts by fish farms on FRSS productivity, intervention experiments must be performed locally. As it stands, we are still very much in the observational phase of the scientific method, with few attempts and apparently little willingness (= funding) to test hypotheses with respect to impacts of salmon farming on FRSS.

*LMD: Agreed.*

Lacking are comprehensive, controlled studies of lethal and sublethal effects of sea lice on juvenile sockeye salmon, similar to those performed with juvenile pink salmon. This makes any attempt to assign a mechanism for a cause-and-effect of sea lice on FR SS productivity entirely premature and speculative. The experimental knowledge base recently generated for juvenile pink salmon in the Broughton Archipelago has greatly shifted our thinking relative to earlier speculative and theoretical writings.

*LMD: Exactly such studies are identified under the heading State of the Science: Knowledge Gaps.*

A key interventional experiment that should be performed in BC is one already used in Norway and Ireland. Wild out-migrating SS could be captured prior to reaching fish farms and fed prophylactically with SLICE for a couple of days to protect them from sea lice as they swam past the farms. Then it is a 2-year wait to count surviving adults relative to control fish. This experiment would directly test the impact of sea lice.

Given the perceived importance by the public as well as the highly polarized positions that have been adopted on the issue of the potential aquaculture impacts on wild salmon, I recommend that a working group – not a single individual – assemble existing information in a manner useful for ongoing analyses. The greatest challenge will be the selection of the members of the working group, which must include individuals with a good working knowledge of multivariate statistical analysis, fish stocks, an ability to faithfully represent all existing data, support conclusions with data, have few preconceived ideas other those supported by data, and bring with them a good measure of common sense and willingness to have an open mind to new data and its analysis.

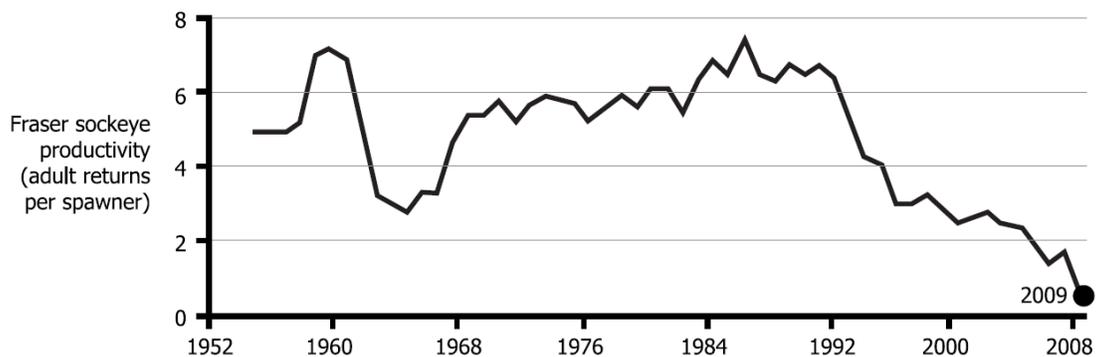
Rates of louse shedding by juvenile SS represent a major knowledge gap in our efforts to assess interactions between farmed and wild salmon populations. For example, if the chances for reinfection are reduced because FR SS pass the farm sites rapidly and if FR SS can shed sea lice as effectively as pink salmon, then juvenile FR SS potentially could have fewer attached sea lice when sampled in the ocean. This possibility could be tested.

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

The author is strongly encouraged to present a more balanced and critical analysis of the available literature to avoid the DR being viewed as providing just a highly selective and polarized opinion, which reduces the validity of some credible concerns.

*LMD: A general statement like this is hard to respond to. I have tried to address it in response to specific points made (some of which were incorrect in any case).*

The DR uses lines of logic to present hypothetical scenarios for impacts. Had this type of logical thinking been applied to the trends in FR SS productivity data presented as Fig. 1 of the DR (see below) and related commercial salmon farming events, then I believe a better framework for exploring impacts might emerge.



As I see the data, 20 years of almost steady FR SS productivity between 1970 and 1990 followed a precipitous decline in SS productivity in the early 1960s. These events preceded commercial salmon aquaculture. FR SS productivity was then punctuated by another precipitous decline, one that was coincident with the onset of commercial salmon farming in BC in the 1990s. When salmon farming had reached its peak production

around the late 1990s, FR SS productivity had reached the historic low of the 1960s, but then continued to fall after 2000 to a historic nadir while farmed salmon production did not had in a major way during this nearly 10-year period.

***LMD: This comment is indicative of how the reviewer has failed to grasp the significance of the Connors long-term analysis, which looks simultaneously at several factors (and their interactions) that have been suggested by experts to be the cause of the decline (R. M. Peterman et al. 2010. Synthesis of evidence from a workshop on the decline of Fraser River sockeye. Pacific Salmon Commission, Vancouver BC). Looking at factors like fish farming one at a time (as in the Korman report) is much less informative, and impossible with the limited data in any case.***

Such associations lead to a simple, plausible and testable hypothesis to explain the decline in FR SS.

1. The entire 30-y decline was caused by the onset, growth and nearly 10-y steady production of farmed salmon.

However, the KTR and the CTR clearly point out the difficulty of testing this hypothesis because details on farming practices do not extend back far enough to be useful for statistical analyses. Given that the continued decline in FR SS productivity continued long after a plateau in farmed salmon production, one could argue for a time-delay in negative impacts. However, the 2010 FR SS productivity (and perhaps the FR pink salmon productivity in 2009) represents an unusually dramatic recovery (off the chart in Fig. 1), which would perhaps require equally dramatic changes in aquaculture practices rather than the fewer lice and possibly fewer disease outbreaks reported in the KTR. Moving forward, while there is an on-going need to secure data to perform long-term analyses as deemed necessary, such data and analyses will not help resolved the current issue.

Given the details on farming practices are limited in duration, perhaps a related and testable hypothesis is the following.

2. The 30-y decline reflected a repeat of the decline seen in the 1960's and unrelated to salmon farming practices followed by a secondary, near cataclysmic and unprecedented decline in FRSS productivity that was triggered by the sustained, peak salmon farming activity.

In this sense, salmon farming activities had advanced beyond a “tipping point” around 2000. However, the data contained in the KTR and the short-term analysis perform in the CTR do not lend support for this hypothesis.

Works specifically cited above.

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Hvidsten NA, Finstad B, Kroglund F, Johnsen BO, Strand R, Arnekleiv JV & Bjørn PA. 2007. Does increased abundance of sea lice influence survival of wild Atlantic salmon post-smolt? *J Fish Biol* 71: 1639–1648.

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Krkošek, M., Ford, J.S., Morton, A., Lele, S., Myers, R.A., Lewis, M.A., 2007. Declining wild salmon populations in relation to parasites from farm salmon. *Science* 318, 1772-1775

Krkošek, M., Morton, A., Volpe, J.P., Lewis, M.A. 2009. Sea lice and salmon population dynamics: effects of exposure time for migratory fish. *Proc. R. Soc. Lond. B Biol. Sci.* 276, 2819-2838.

Report Title: Scientific Research Project #5D – Impacts of salmon farms on the Fraser River sockeye salmon – by Dr. Larry Dill

Reviewer Name: John R. Post

Date: June 22, 2011

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

The most important point made by Dill in this review is that it is unlikely that there is a singular cause for the decline of Fraser River sockeye salmon. On the surface this sounds like a trivial point for us scientists, but the whole inquiry appears to be set up as a series of singular analyses dealing with a list of potential causes, largely in isolation. If in fact Dill is correct, the predicted outcome of the process is rejection of a laundry list of independent processes, many of which may indeed have some complex impact on sockeye population dynamics. The Connor (2011) analysis is suggestive along these lines, but is certainly not conclusive for statistical reasons.

A general review of the literature led Dill to conclude “the available evidence suggests that salmon farms can be deleterious to sympatric wild salmon, at least under some circumstances and in some places”. This is defensible given the literature, but it of course says nothing about Fraser River sockeye and BC fish farms. But it does point out that the appropriate null working model is one of impact that needs to be rejected. Statistical purists won't buy this, but this is the logical precautionary approach to environmental impact issues.

Although the objective of the review was to consider Fraser River sockeye, it would be very useful to know if there any information on time series of adult returns per spawner in other areas, without salmon farms, while on their seaward migration route? i.e. is this temporal pattern (Figure 1) general across stocks or particular for the Fraser populations? Along the same lines have all of the Fraser sockeye stocks shown the same pattern when disaggregated by spawning location and timing?

***LMD: These data are available in Peterman RM & Dorner B. 2011. Fraser River Sockeye Production Dynamics. Cohen Commission Technical Report, Vancouver, BC. Connors (2011) included other sockeye salmon populations as reference populations for the aquaculture variables considered in his analyses. The complete sockeye dataset considered in that report included 18 populations from the Fraser River as well as 8 others from Washington State and British Columbia and 5 from the Southeast and Yakutat regions of Alaska.***

The brief discussion of the Pacific Ocean Shelf Tracking (page 33) is very interesting and I am left wondering if this work can provide any more useful information on the spatial and temporal patterns of juvenile sockeye migrations

and their mortality patterns. Is there more of this available?

***LMD: Not to my knowledge. And its worth noting that the only sockeye tagged were from the Cultus and Sakinaw Lake stocks, not those with longer migrations to Fraser headwaters.***

**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?**

I suspect that there is extensive discussion of Figure 1 in other reports, but it merits consideration in this analysis of fish farm impacts. The observation is a relatively invariant adult returns per spawner through the late 60s to late 80s. Then substantial decline starting in the late 80s with a reduction in the rate of decline since the mid 90s. Are there features of the early fish farming industry from the late 80s to mid 90s that could have had this strong impact, and which appears to be somewhat ameliorated more recently?

***LMD: A good question but hard to answer as we have no fish health data prior to 2003.***

The assessment of disease transfer from escaped Atlantic salmon to wild fish appears to be overstated. The diseases discussed are all endemic in Pacific salmon and it appears that escapees are rare. I think that the key issue is in the potential to concentrate disease in fish farms which then act as an incubator and source of disease for juveniles that migrate near farms. The small number of escapees won't have this potential impact.

***LMD: Agreed. The point about biomagnification is made on pg. 24.***

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

See above in points 1 and 2.

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

In general, yes, with caveats discussed in other sections of this review.

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

To understand the potential impact of disease transmission from farmed to wild fish we need spatial and temporal data on disease prevalence and impacts (condition, growth and survival) on juvenile sockeye as they migrate through the gauntlet of fish farms.

***LMD: Agreed. This is covered in the section State of the Science: Knowledge Gaps***

To understand the potential impact of sea lice infection we need spatial and temporal data on sea lice prevalence and impacts (condition, growth and survival) on juvenile sockeye as they migrate through the gauntlet of fish farms.

***LMD: Agreed. This is covered in the section State of the Science: Knowledge Gaps***

Correlation, multiple regression and time series approaches can be useful in developing mechanistic hypotheses but they are data hungry and riddled with statistical caveats. The data series must be continued because the very limited data available at this point has insufficient power to identify an effect, if there is one. But be careful to conclude that there is no effect from the information that currently exists.

***LMD: Agreed. As stated in the report it is important to continue the collection of data, with periodic re-analyses.***

It would be useful to know if migrating juvenile sockeye are attracted to farms due to enhanced food, or lights, or due to their bathymetric characteristics. If so this could enhance disease or lice transmission and concentrate the negative impacts on the localized chemical environment.

***LMD: Agreed, and covered in the report.***

We need directed studies on the impact of lice on juvenile sockeye, in both controlled conditions and in the wild, to help extrapolate impacts to population level effects.

***LMD: Agreed, and covered in the report.***

The idea that lice enhance the transmission rate of various diseases warrants further research.

***LMD: Such research is underway, in both BC and Europe.***

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

The author should recognize the statistical caveats associated with the multiple regression result of Connor (2011) that concluded that fish farms and pink salmon impact survival. The best way to present this is that the work is suggestive of a logical mechanistic explanation that now needs mechanistically oriented research.

***LMD: Connors (2011) is careful in his report to make the distinction between correlation and causation and he addresses the uncertainty in his estimated effects in some detail. I have reiterated these cautions (pg. 16).***

## **APPENDIX 3 - INTERVIEWS CONDUCTED (in person, by phone or by e-mail)**

### **MEETINGS**

Alex Morton, Raincoast Conservation  
Rick Routledge, SFU  
John Reynolds, SFU  
Craig Orr, Watershed Watch  
Stan Proboszcz, Watershed Watch  
Tony Farrell, UBC  
John Volpe, UVic  
Stephen Cross, UVic  
Ben Koop, UVic  
David Lane, T Buck Suzuki Foundation  
Duane Barker, VIU  
Kristi Miller, DFO  
Stewart Johnson, DFO  
Simon Jones, DFO  
Kees Groot, DFO (ret)  
Andrew Thomson, DFO  
Ron Tanasichuk, DFO  
Dick Beamish, DFO  
Chris Neville, DFO  
Mike Foreman, IOS  
Dave Mackas, IOS  
Angelica Pena, IOS  
Marty Krokosek, University of Otago

### **TELEPHONE INTERVIEWS**

Mark Sheppard, DFO  
Pat Gallagher, SFU  
Susan Allan, UBC  
Michael Price, UVic and Raincoast Research  
Vic Palermo, DFO

### **E-MAIL INTERVIEWS**

Neil Frazer, U Hawaii  
Jody Erikson, Raincoast Research  
Brent Hargreaves, DFO  
Marc Trudel, DFO  
Sonja Saksida, BC Centre for Aquatic Health Science  
Ashley Park, UVic  
Larry Hammel, UPEI