EXECUTIVE SUMMARY

The Pacific Coast Wild Salmon Society, the Raincoast Research Society and Dr. Alexandra Morton (the 'Aquaculture Coalition') are pleased to put forward these submissions to the Cohen Inquiry. The Commission's task is threefold:

1. To review the cause of the 2009 sockeye collapse,
2. To understand the reason for the long-term declines in productivity since1992, and
3. To provide recommendations for the future sustainability of sockeye salmon.

The Commission has heard important evidence on a wide variety of threats to the well-being of the Fraser River sockeye. Each of these is legitimate, and we look forward to recommendations from the Commission on many of these topics to ensure future health of the wild sockeye. However many of these threats, while important to future sustainability, do not account for the 2009 collapse.

The submission of the Aquaculture Coalition is that the primary cause of the failure of the 2009 sockeye return was disease, and that salmon farms along the path of the migrating salmon played a significant role in the origin or amplification of that disease. Ocean conditions north of the salmon farms may have played a role in increasing mortality from that disease.

Disease is also the most likely cause of the long-term declines in salmon productivity since the early 1990's, when salmon farms expanded into the confined areas of the Discovery Islands and Johnstone Strait. We should not be surprised. Wild salmon are in decline wherever there are salmon farms worldwide.

Wild salmon declines due to disease is the best fit for the available evidence differentiating between the stocks that thrived and those that failed in 2007, and in distinctions between 2007 and 2008 returns.

Direct empirical evidence of disease causation is not feasible in salmon populations, and could not be expected here. However, there is compelling ecological evidence and evidence from other fish farming jurisdictions to show the inevitable role that fish farms play in the amplification of endemic diseases, and the evolution of new diseases. [See Part I-2]

We have asked the Commission to point out to the public that the Process the Commission adopted in this Inquiry did not allow the participants, including the Aquaculture Coalition, the opportunity to call evidence or prove the harms of fish farms. We were reliant on evidence called by the Commission counsel and on cross-examination. This was not a trial. [See Appendix A]

However, the evidence before the Commission did show compelling evidence of the true rate of disease in fish farms, which is much greater than the public has previously understood. Through the Commission's efforts, the databases produced by the Province and salmon farmers, when properly examined, show rates of *mortality* from disease on fish farms in the range of **2 million** fish per year (4-6% of the population). The rates of sick or infected fish are probably much

greater, but the evidence shows that no one knows how much higher, because no one is measuring it. The numbers of diseased fish in farms along the sockeye migratory route likely at times exceed the number of wild sockeye. The number of pathogen particles to which migrating sockeye may be exposed could be in the billions. [Part I-2(c) – (e)]

The Project 1 and Project 5 Reports, prepared by scientists selected by the Commission, were unanimous in identifying the failure of DFO science in respect of disease and wild salmon. There are simply no studies that have been done from which it is possible to justify the extraordinary ecological risk represented by diseases from fish farms on wild sockeye routes.

Although Dr. Conners found a clear statistical connection between increasing farm salmon production and wild sockeye salmon mortality, we question the utility of the project reports' focus on a search for statistical patterns, in the unpredictable world of disease epidemics, and given the few years of data produced. Instead, we encourage the Commission to focus on ecological science. We adopt the conclusions of Dr. Dill from SFU:

> "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." *Dr. L. Dill, Project 5D"*

and

> "…The findings of a long term analysis suggest that high standing stocks of fish and floating net pens in the Discovery Islands can negatively impact Fraser sockeye survival…"[1]

[The Project 1 and Project 5 Reports are discussed at Part I-3]

We discuss in detail the unprecedented evidence of diseases and clinical signs of diseases, known and unknown, that has become available as a result of the production of the disease databases, and we urge the Commission, despite the amount of work involved, to understand this evidence in detail. We point out that of the thousands of fish audited, many died of unknown or undiagnosed diseases – 60% of the diagnosis are 'open'. We note the limitations of a process by which only confirmed diagnosis are counted, and the diseases of individual fish ignored, counting only epidemics "at the population level" [Part I-3(a), (b)]

We highlight the evidence available showing the "classic lesions of ISA and the prevalence of symptoms of salmon leukemia (marine anemia) shown in the database. [Part I-3(d)-(f), and Appendix B]

The disease of salmon leukemia (marine anemia), which is a disease of 'confinement' and was the cause of fish farm epidemics just preceding the long-term decline in sockeye is also

[1] Ex. 1540, p. 15

examined in detail. As a case study, it demonstrates the approach of DFO to science when a new disease is discovered. Despite the high susceptibility of sockeye to salmon leukemia found in fish farms, DFO abandoned research into the effects of the disease on wild fish, once the disease ceased to be of economic concern to the aquaculture industry. We suggest the disease continues to exist, and may potentially be having continuing adverse effects on sockeye, and perhaps a continuing connection to the virus found by Dr. Miller. [Part I-4]

We submit that the work of Dr. Miller is extraordinarily important, and potentially the 'smoking gun' that could explain not only the cause of the 2009 decline, but also the abnormal early entry behaviors and pre-spawn mortality that has been occurring since the advent of fish farms in the wild sockeye migratory route. We also suggest that DFO's response to Dr. Miller's work is indicative of its current incapacity to respond effectively to new diseases, and an example of the conflict of interest that arises from DFO's promotional support of aquaculture. [Part I-5, 6].

In respect of conditions in the marine environment, we support the work of Dr. McKinnell and Dr. Welch, suggesting that marine conditions after the smolts had passed the fish farms may have been challenging in 2007, which potentially could have exacerbated the effects of any disease. We suggest that the evidence supportive of a theory in respect of Georgia Straight is much weaker and not credible. [Part I-7]

In Part I-8, we note a potentially important anomaly between 2007 and 2008, and the significance that Chinook farms in the Discovery Islands may have been fallow in 2008, but infected with marine anemia in 2007. [Part I-8]

In Part II we examine more fully the Regulatory Environment under which DFO has failed to meet its mandate to protect wild salmon from the impacts of aquaculture.

Of most importance is that 'Siting Decisions' that were made in the past under which existing fish farms got their licenses and locations, were made in the absence of consideration of the potential effects of disease on wild salmon. The research did not exist at that time. DFO's decision to 'grandfather' these sites is unwise. We urge the Commission to ensure that this dramatic oversight is corrected, and to recommend, at least, that the fish farms on the constrained areas of the wild sockeye migration route be reconsidered and removed. [Part II-1]

The Commission has an exceptional opportunity to impact DFO's regulation of aquaculture. We examined DFO's decisions to favour aquaculture as an industry, the lack of enforcement of section 35 and 36. [Part II-3]

We also suggest that the proposed regulatory mechanisms that DFO has and will institute, which turn on self-regulation, self-reporting and depend upon 'Fish Health Management Plans' will not be adequate to protect wild sockeye from disease, and are not an abdication of DFO's responsibility.

ISA is a disease of major significance in every other fish farm jurisdiction in the world, and will have unknown impacts on wild sockeye salmon. We submit that the mechanisms to protect BC from ISA instituted by DFO are inadequate and will or have already failed. We will be asking

the Commission to reopen to accept newly discovered evidence. We urge the Commission to recommend appropriate testing at all fish farms, now and in future.

We submit that the evidence establishes that DFO's role in promoting aquaculture has, in the last two decades, frequently undermined its role in the protection of wild sockeye from the aquaculture industry. We suggest that the political support of the Government of Canada for the aquaculture industry, whether or not it is misplaced, has no place in the mandate of DFO if wild sockeye are to survive. [Part II-7]

The Science Branch of DFO is in need of a substantial overhaul. The absence of science, through two decades of aquaculture, into the potential impacts of disease on wild salmon populations is one of the most glaring examples. Science dollars have overwhelmingly gone to support the industry. Science staff has lost its independence, and is regularly used to promote industry or to provide misinformation, designed to comfort the public. We examine the controversy over sea lice from fish farms in that context, and suggest that DFO has been predominantly involved in conducting 'rebuttal science', designed only to respond to public concern. [Part II-8]

DFO claims to administer its programs pursuant to the "precautionary principle". We submit that the evidence submitted to this Commission shows that that principle is honoured more in the breach. In fulfillment of its mandate, we ask that the Commission consider carefully the application of cautionary principle and the inevitable risk to wild sockeye of disease from the aquaculture industry. [Part II-9]

FINAL SUBMISSIONS OF THE AQUACULTURE COALITION

PART 1 - CAUSE OF DECLINE

1. The Cause Of The 2009 Collapse

Wild salmon are in exceptional decline wherever there are salmon farms worldwide (Ford and Myers, 2008).[1] *Tthe decline of the Fraser sockeye is not an unexpected outcome of siting industrial salmon farms in their narrow migratory channels. One of the two mechanisms of impact identified by Ford and Myers was pathogen amplification.*

The Aquaculture Coalition proposes the conclusion that pathogen amplification by fish farms is the primary driver of the sockeye declineIt is the best-fit variable to meet the criteria of timing, the specific stocks affected, the onset of the anomalous early entry and pre-spawn mortality, the viral indicators found by Dr. Miller (both genomic and physical) and the reversal of the decline in 2010 and 2011.

No one may ever be able to definitively *prove* the cause of the 2009 collapse of Fraser River sockeye. Scientific proof, after the fact, does not work that way. However, one can point to likely or probable causes, based on correlations. Some witnesses have referred to this as trying to solve a 'detective story'.

There are a number of facts – 'clues' – that must be accounted for in identifying the probable culprit:

(a) the Columbia stocks (which migrate outside Vancouver Island) and the Harrison stocks (which stay in Georgia Strait) did well in 2009. It was primarily the fish stocks that migrated through the inside passage (Johnson Strait and the Discovery Islands) which primarily were devastated;[2]

(b) the 2008 returns versus 2007 returns (something was wrong in 2007 that was different in 2008);

(c) Dr. Miller's work, which showed dramatic differences in the 2007 MRS (90%) versus the 2008 MRS (40%)[3]; and

(d) The long-term decline in productivity dates back to 1992 (the dramatic increase of aquaculture on the migration route also dates to the early 1990s).

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[1] Ex. 1487, Ford, Myers, "A Global Assessment of Salmon Aquaculture Impacts on Wild Salmonids"
[2] Dr. Welch October 25, 2010 Trancript, pp. 66-68
[3] Ex. 2, "Presentation of David Welch", slide 15

There is evidence before the Commission that suggests that a significant amount of mortality happened on the smolt out-migration in 2007, within 20 to 30 days of those smolts passing through Johnstone Strait.

There is also evidence, through Dr. Miller's work, that suggests that a significant amount of mortality -- sufficient to account for potentially all of the lost salmon in 2009 -- may have occurred on the inward migration, and through abnormal early entry and pre-spawn mortality, as the fish returned in 2009. This loss may have been caused by a viral disease, combined with the normal stress of the transformation from sea water to fresh water.

Disease offers the best explanation for all of the available evidence. It may be that unusual ocean conditions in 2007 exacerbated the impacts of this disease, but this is not an independent cause if disease was responsible for increasing the vulnerability of the salmon to those conditions.

The nature of the specific disease, and its cause or origin, are as yet undetermined. The work of Dr. Miller may provide further answers to the identity of the disease, but is unlikely, given the time that has passed, to be able to prove its origin.

Whether disease was *the* cause of the 2009 decline or a significant contributing factor should not change the nature of the Commission's recommendations. There is likely very little this Commission can recommend or DFO can do to change ocean conditions. Disease, however, is one over which recommendations and actions can have significant effect, if the disease arises from or is exacerbated by human activities.

If disease is the cause of the 2009 collapse, it is unprecedented in the known history of the Fraser River sockeye. In general, as the Commission has been told, in the natural world sockeye and disease agents maintain an equilibrium. An epidemic of this magnitude must have been caused by a new disease, or by a new strain, or an existing disease increased in virulence, amplitude and exposure.

The most obvious source for a new disease agent is fish farms which have been located directly within the path of the migratory sockeye salmon, and to which they are exposed at the most vulnerable stages of their life history. Fish farms, both in this country and elsewhere, are demonstrated incubators and reservoirs of disease. The concentration of fish farms in the confined areas of Johnstone Strait and the Discovery Islands is an abnormal risk factor for wild salmon- one within the control of DFO. No other significant causal agents have been identified during the Commission's hearings. **Whether or not the actual disease or the moment of disease transfer from the particular farm to a particular fish population can ever be identified, it would be irresponsible folly to ignore this obvious risk**.

2. Fish Farms Cause Disease

(a) The Ecological Approach – Fish Farms are an Inherent Risk

In the absence of direct empirical proof, the science available on risk is compelling. Aquaculture is a massive change in the ecosystem for the Fraser sockeye. Fish farms magnify endemic disease, increase exposure to disease for Fraser sockeye, and create the conditions for the emergence of new diseases.

The Commission has heard evidence that it may be impossible to ever prove, on a direct-observation, empirical scientific basis, that a particular disease in wild salmon was caused by one or more particular fish farms, even if true. It is difficult enough to identify mortality in wild sockeye in the open ocean[4] -- dead fish drop to the bottom or are eaten by predators. There are added problems with proving disease -- disease transfers in the ocean away from observation, salmon populations are difficult to segregate and follow.[5]

On top of this, it is clear that any and all disease outbreaks on fish farms are readily assumed by fish farmers to originate from wild stocks, and fish farmers are willing to identify any disease as "endemic" should it ever be identified in wild populations[6]. DFO appears to accept this. Even where it could be shown that a particular disease is impacting wild fish, and this could be correlated with a disease outbreak on one or more fish farms,past conduct suggests that industry veterinarians will immediately claim that the fish farm got the disease from wild stocks, and not the other way around. Given the current state of science, such a hypothesis will not be disproven.[7] Fish farm scientists, who readily accept the transfer of disease from wild stocks to farm fish, seem to believe that water flows in only one direction through open nets.

In the absence of any reasonable methods to obtain empirical or observable proof of specific disease transfer by fish farms, how does science evaluate risk? The inability to prove the actual connection does not mean there is no scientific evidence of risk. The scientific evidence, both from general ecological science, and from experiences in other countries, is compelling:

> "The rapid growth of aquaculture has been the source of anthropogenic change on a massive scale … not surprisingly, the consequence has been the emergence and spread of **an increasing array of new diseases**."
>
> Walker and Winton, Exhibit 1486.

> "Aquaculture can offer close to **ideal environments to the spread of infectious diseases.** Owing to high density monoculture of hosts, numerous possible routes of

[4] Ex. 1461, p4; Aug. 22, 2011 Transcript (Question from Commissioner to Fish Health panel), pp. 37:1-38:43, evidence of Dr. Kent, pg 11,
[5] Dr. Kent, Transcript, August 22, pg 11
[6] Eg. Dr. Sheppard Briefing Note re ISA – ISA will originate from wild stocks Ex. 1679 (Aug 1, 2007)
[7] Unless one is present to witness the first transfer of pathogen to the first fish on a disease outbreak – obviously an impossible scenario.

transmission and suboptimal protection by available vaccination for several viral diseases, viruses may thrive in modern salmon aquaculture."…
"The key to control viral epidemics is to block the transmission of infection." …

Rimstad, Exhibit 1482.

"**Viral diseases are a major problem** in Atlantic Salmon aquaculture….Farming fish in dense populations in the open sea **inevitably leads to outbreaks of infectious diseases**."
"Weak individuals within the farm population will pick up pathogens from the external environment and transmit the agent to healthy individuals."

"Without vaccines, Atlantic salmon farming would have been impossible due to bacterial diseases … . Today, **viruses represent the main challenge**."
"Outbreaks of these diseases must be due to **the appearance of high-virulent viral strains** …"

"It is now well accepted that **horizontal transmission is the main route of the spread of viral diseases in salmon farming**."
"Viral diseases will remain a **continuous problem in aquaculture in efforts to stop horizontal and vertical spread of viruses**, therefore need to be at the forefront of disease control strategies."

Robertsen, Exhibit 1483.

"An increasing number of scientists have recently raised concerns about the threat posed by human intervention on the **evolution of parasites and disease agents**. New parasites (including pathogens) keep **emerging** and parasites which previously were considered to be 'under control' are **reemerging sometimes in highly virulent forms**. This reemergence may be parasite evolution, driven by human activity, including ecological changes related to modern agricultural practices. **Intensive farming create the conditions for parasite growth and transmission drastically different from what parasites experience in wild host populations**"…

"We consider the case of the fish farming industry … and present evidence that supports the idea that **intensive farming conditions increase parasite virulence**."

Mennerat et al., Exhibit 1484.

"Microbial pathogens have preyed on fish for eons and have co-evolved with them. These cohabitants have, in a general way, established an overall equilibrium with their house in their natural habitat. … The artificial rearing of fishes has led to the **exacerbation of diseases that previously existed in wild populations.**"

Reno, Exhibit 1485.

These science reports, and many like them, cannot be ignored.

These and others suggest a consistent international experience that where there is a large-scale aquaculture industry, there are significant disease issues and impacts to wild stocks. In their

article "A Global Assessment of Salmon Aquaculture Impacts on Wild Salmonids" [Ex. 1487], Ford and Myers conclude after applying a meta-analysis of existing data that, that marine survival and abundance of salmonids in regions with salmon farming is reduced from those without (p. 1). The authors posit:

> "The estimated reduction in survival of wild salmonids is large, and would be expected to increase if aquaculture production increases." (p. 4)

This statement is supported by the evidence of the effects of salmon farming throughout the world[8]..

(b) The Risk is from Endemic and Exotic Diseases

There seemed to be an operating assumption – one that sometimes underlay the testimony or reports of scientists supportive of fish farms – that if a disease has ever been found in wild populations, it can be said to be endemic, and, the impact of fish farms on that presence, level and risk from the disease can be ignored or disregarded.

Thus, Dr. Kent and Dr. Noakes could minimize known diseases and Dr. Saksida could dismiss marine anemia as a threat.[9] This is the logic by which fish farmers can claim that so long as egg imports are carefully regulated, everything is fine – because all diseases can then be said to come from wild fish, and any disease that comes from wild fish is by definition no longer a threat.

Such logic ignores the literature above – it ignores the devastating role that fish farms can play in *amplification* of disease, as a *reservoir* of disease, and in *evolution* and alteration of *virulence* of existing diseases. Fish diseases, like human ones (e.g., Avian Flu) can constantly undergo the creation of new strains, which may have dramatic impacts.

It is absolutely self-evident that the location of fish farms represents a highly abnormal eco-system risk -- an "*anthropogenic change on a massive scale";* conditions for disease transmission "*drastically different"* from natural conditions. The extraordinary number of fish in a densely confined area of similar age and genetic makeup is an ideal breeding ground for disease. As noted in the evidence of a number of witnesses and reports, wild stocks in general live in equilibrium with disease. Their comparatively low densities greatly reduce transferability, and diseased individuals are quickly removed by predators. As wild fish travel to the open ocean, even where disease naturally exists, the concentration of pathogens will normally be low. When fish farms undergo disease outbreaks, many ready hosts exist in great numbers, free from predators, and diseases can readily explode into epidemics. Pathogen concentrations in the vicinity are very high, and cannot be compared to natural conditions.

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[8] Other evidence before the Commission (in addition to those articles cited above), show the same pattern of disease associated with fish farming. Ex. 1502 discusses the presence of ISA in all regions where there is large-scale Atlantic salmon aquaculture, In Ex. 1571, Costello reviews the consistent negative impact of aquaculture on sea lice for wild stocks throughout the world.

[9] Based on one study that found it in seven fish. Exh. 1796; Ex.1493, Eaton et al. "Biochemical and Histologenic Evidence of Plasmacytoid Leukemia…"

Fish farms also increase the virulence of disease for reasons discussed on the scientific literature, and the new diseases and new strains of existing diseases can readily evolve or "emerge". New diseases, 'emerging diseases', or more virulent strains of existing diseases can do untold damage to wild stocks, before any sufficient reactions from regulators can possibly occur.

(c) The Rate of Mortality

There are approximately 30 million Atlantic and Chinook salmon held in net pens in an average year on the BC Coast.[10] 3,000,000 (10%) of those fish die each year.[11] Most of that is from disease.

Dr. Korman collected the data (from BCSFA files), and collated it in his Spreadsheet (Exhibit 1544). Dr. Noakes published, as Table 8 in his report[12], a table taken directly from Dr. Korman's report. Dr. Korman wrongly assumed, in preparing this table and his report, that the "fresh silver" category was the "maximum number" of fish that may have died of disease[13]. He concluded therefore that mortality was *only 2% on average*. Dr. Noakes accepted that number, and then declared that number to be "quite low".[14] When pressed for a justification for this subjective opinion, Dr. Noakes stated "I'm not an expert in fish health."[15]

But that evidence was proven to be mistaken. An examination of the categories in the BCSFA database shows that disease is equally likely in the "Other" categories from Dr. Korman's data. The only distinction between "Old" and "Fresh Silvers" is the state of decay at the time they are collected.[16] The 'old' category in the spreadsheet totals 5,249,809 for the relevant period. The 'other' category also includes 'poor performance' (4,030,887), which can be readily associated with the disease. The environmental category (7,628,126) includes deaths from algae or low oxygen. It may also reasonable to assume that in the other categories, e.g., predators, handling/transport, the diseased fish will be overrepresented even from this cause of death.

Including just the 'old' and the 'poor performers' category doubles the number of mortalities properly attributable to disease, to approximately **4%**. Adding in the environmental category would increase the mortality rate from disease to almost **6%**. Dr. Korman agreed with this analysis in his cross-examination.[17]

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[10] Korman, Ex.Ex. 1543, Figure 3, p. 17
[11] Dr. Korman Report 5A, Figure 4, p. 18; Ex. 1544, Korman Spreadsheet 'Mortality Summary' Tab
[12] Technical Report 5C, Ex. 1536, p. 27
[13] Korman Report, p. 7
[14] Noakes Report, Ex. 1536, p. 25. We question how a subjective opinion like this can be supported. On cross-examination neither Dr. Noakes nor Dr. Korman could support this. August 29, 2011 Transcript, pp. 22, 24
[15] August 29, 2011 Transcript, p. 26, line 19
[16] The definition for the various categories is found at Ex. 1564, Supplemental Appendix to the Annual Report Fish Health Program, BCMAL, at p. 4.
[17] Transcript, August 29, 2011, p. 17-21

BCMAL Fish Health Plan Report indicates that fresh silvers should generally represent less than 1% of the population.[18]

- –Silvers": total number of fresh carcasses that still have silver skin/scales and have died most recently, due to: no apparent reason, or they may show signs of disease. These carcasses are likely most reflective of the robust living _production population' and they generally represent less than 1% of the dead group.

It is unlikely that any reasonable epidemiologist would suggest that a disease rate of 5-6% of a given population, on an annual basis, is 'healthy'. As noted in cross-examination, the Spanish flu, the greatest epidemic in modern human history had a mortality rate of 3% (and it lasted two and a half years).[19] By BCMAL expectations, the existing disease mortality rate on fish farms may be as much as five times that expected in a healthy population.[20]

Further, the mortality rate does not describe the full rate of very few diseases are 100% fatal. The rate of disease, for most disease is much higher. More fish get sick than die. In the rate of sickness the number of fish infected may be ten times higher or more than those that die, depending on the particular disease.

Some order of magnitude of this risk, in relation to passing sockeye salmon, is important. If an average sockeye return is in the range of 5 to 10 million fish, the 30 million farmed Atlantics and Pacifics would far exceed the total number of returning sockeye. If 20-30% of the farmed fish suffer from disease in a given year, diseased farmed salmon outnumber sockeye.

In that context, it is notable that the BCMAL audit data base[21] confirms that on a *random audit*, 95+% of the individual fish audited show signs of disease, known and unknown, even where the veternarians have not made a diagnosis of disease at the farm population level. (see Disease Databases)

There is no evidence before the Commission that would allow the conclusion that the rate of mortality from disease, whether it be 6%, or 4% or even 2% (which was proven wrong), is 'low'. There is no evidence before the Commission that would allow the conclusion that the rate of disease (including both mortalities and chronic disease) is within acceptable bounds.

This Commission should not conclude that the rate of disease on fish farms is either 'low' or acceptable. We submit that the weight of evidence shows the rate of disease to be grossly unacceptable at fish farms, given their location in the midst of the migratory runs of sockeye, in a wild environment that would otherwise have low pathogen levels.

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[18] Ex. 1564, p. 4
[19] Wikipedia, 1918 Flu pandemic, http://en.wikipedia.org/wiki/1918_flu_pandemic
[20] BCMAL Fish Health Report, Supplement, Ex. 1564
[21] Ex. 1549-217 (2864)

(d) The Rate of Disease on Fish Farms

No one knows the total rate of disease on fish farms[22].

That is because no one is measuring it. Fish farms measure and report only numbers of *dead* fish, ie.mortality. The Province audits only *dead* fish.

Why? Perhaps because mortality has a financial cost -- dead fish cannot be harvested. Fish with clinical or sub-clinical sickness, or with chronic diseases, can generate and transfer pathogens to wild fish as (or more) effectively than dead fish, but do not matter to fish farms unless they have significant impacts on growth. And the closer to harvest size, the less that matters to the fish farmer.

But, to the public and to the sockeye, it is the rate of disease that matters, not the rate of mortality. It is diseased fish that breed and transfer viral pathogens to the open waters.

Chronic Disease vs. Acute
As Dr. Stephen noted in his Report 1A

> "Most reports are of overt die-offs rather than the chronic or sub-clinical effects that influence the ability of fish to reproduce or survive. The most likely diseases to continue to spread within a population are those that infect fish but allow a fish to live with its cohorts for longer effective contact times. This stands in contrast to most historical fish disease research that has focused on rapidly lethal diseases and not on the dynamics of chronic, endemic disease"[23]

The amount of pathogen produced by a single farm with a disease outbreak may be 60 billion particles per hour.[24]

The greatest risk from disease comes from live infected fish, yet DFO does not measure or audit live disease.

Astonishingly, DFO (and the Province before it up to 2010) has never made any effort to study the rate of disease occurring in live fish within fish farms. The Audit program instituted by the Province (and now replicated by the Federal Government) takes only 'fresh silvers' – ie recently dead fish.

Not only does the regulator (Province and now, DFO) have no program to study the rate of disease in live fish, DFO apparently takes the position it has no right to do so under current regulation. When Dr. Miller needed to test live fish in fish farms in 2009 for SLV, and again in 2010 for parvovirus, she needed the permission of fish farms to do so. (She didn't get it in 2009,

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[22] Note Dr. Korman: Aug. 29, 2011 Transcript: "Q Now, Dr. Korman, you didn't measure how many fish were sick in fish farms, no way to do that? DR. KORMAN: Right. Correct
[23] Project 1A, Ex. 1454 p. 27
[24] Ex. 1529, Garver, "Risks of IHNV dispersion associated with aquaculture",, pg 8

and so it never occurred; in 2011, she may get it, but the fish farms must agree on the conditions and their role – this testing may never occur, or may be ineffective).

RECOMMENDATION: ***DFO institute a program to audit disease in live fish, and the federal government implement regulations to allow DFO to conduct the audits .***

(e) The Rate of Disease Outbreaks

Dr. Korman calculated, from BCSFA records, and based on Dr. Kent's 'high risk' categories of disease that there are approximately 30 'high-risk' Fish Health Events (disease outbreaks) per year:

> "Approximately 25% of the FHEs (30 events per year) were caused by bacterial and viral diseases that were classified as high risk to Fraser River sockeye."[25]

This is an extraordinary number, considering that there are only 121 farms licenced, and some evidence suggests only 70-80 active on a regular basis. That means either 1-in-4 or 1-in-3 fish farms experience a disease event every year.

Considering there are 10 fish farms in the Discovery Islands that sockeye must pass through, that means the odds are high that in any given migration it is inevitable that the sockeye will pass through at least one disease outbreak.

(f) What is a Safe Threshold for Disease

> *Q. (to D. Marmorek): Would you send your children to a school next to an explosives factory? Isn't risk a factor to be considered, even though you lack empirical evidence…*
>
> *A Well, I think there's pretty strong empirical evidence that explosives explode…*[26]

There is strong empirical evidence that fish farms harbor and amplify disease, and that there is no way to prevent the pathogens from leaving the net, and passing into open waters to exposed migrating salmon.

Thirty high risk events per year (among a population of 70-100 working farms) is not "low" or "reasonable", where such disease outbreaks can happen in the midst of the outward migration of highly susceptible juvenile smolts, or to returning adults undergoing the high stress of returning to fresh water. Millions of diseased fish, held every year in net pens powerless to prevent disease transfer, is a risk too high.

Even the best managed, most carefully regulated explosives factory does present an unacceptable risk, and for that reason they are sited outside of downtown areas, and away from schools.

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[25] Report 5A, Ex. 1543, pg 7
[26] D. Marmorek, Transcript Sept. 19, pg 84

Fish farms, for their own financial self-interest, do work hard to manage disease. But the evidence shows that disease can only be managed not prevented. No matter how well managed in the current regulatory environment, **disease outbreaks do occur** – and regularly. There seems to be no regulatory means to prevent it. And once disease occurs, there is no way to prevent it from coming into contact with wild sockeye salmon, so long as fish farms are sited within the migratory pathways.

What should be the reasonable threshold for disease? Dr. Stephen pointed out in his report and testimony[27] there is currently no accepted standard for the amount of pathogens in the environment against which he could measure.

Both Dr. Kent and Dr. Stephen, and Drs. Noakes and Dill, pointed out the lack of science in wild salmon, or in transmission studies, that could help to answer the question of how much disease transfer might be supportable in wild salmon. Fish farms have been in BC waters for 20+ years, and many millions have been expended in research dollars in designing better nets and more profitable farms, but no money investigating this critical question.

The onus must be on the proponents of fish farms to demonstrate some acceptable science, in light of the proven history of disease, showing a margin for pathogens safe to wild salmon.

Until that is done, the only supportable means to regulate against this unavoidable risk is through siting farms away from the vulnerable migrating sockeye.

3. THE COMMISSION REPORTS

(a) The Project 1 Reports

The reports by Dr. Kent and Dr. Stephen are helpful, but subject to the limitations of the available research and the scope of their work for the Commission.

Dr. Kent has not conducted research in British Columbia for over eleven years, and was completely reliant on the literature available. Given that DFO has done almost no research on diseases in wild salmon, and none at all in transmission of diseases from fish farms[28] to wild salmon, Dr. Kent's literature review was bound to produce negligible results. Dr. Kent confirmed that the absence of data does not mean the absence of disease:

> "The absence of data on pathogens and diseases in wild salmon in British Columbia is a reflection of the historical research focus on fish diseases, in both the Province and other

[27] Aug 22, 2011 Transcript, pp. 44-46 (Stephen)
[28] Dr. Richards, September 26, 2011 Transcript, pp. 62-70, 75:27-42, Dr. Kent and Dr. Johnson explain some of the reasons for the lack of research, August 22, 2011, p. 10-14

> regions. Most research on salmon and diseases has been directed towards those afflicting captive fish, either in government hatcheries or private fish farms."[29]

Dr. Kent noted the difficulties in his 'State of the Science' portion of the report:

> "It is also difficult to study the impacts of diseases on wild fishes, particularly in the marine environment… In recent years, this type of research has not been well supported as it is considered by some funding agencies to be merely survey work and not hypothesis driven."

The inadequacies of the available research precluded Dr. Kent from making any conclusions, on the basis that

> "… pathogens cannot be excluded at this time as adequate research on the impacts of disease on this population has not been conducted."[30]

Dr. Stephen similarly eliminated the inadequacies of the research:

> "The research emphasis on the pathophysiology and microbiology of cultured salmon and diseases has been insufficient to answer questions on how infection diseases can affect the distribution and abundance of salmon outside of fish culture settings…. Evidence based conclusions on the effects of diseases of enhancement facilities on Fraser River sockeye salmon are not currently possible."[31]

Dr. Stephen's observations apply with equal force to fish farms.

In the absence of available research, or direct involvement, Dr. Kent was limited to a basic survey of known salmon diseases, with a subjective assessment of the potential risk. While interesting, this survey has little value other than as a primer – it provides no empirical justification for risk, allows no conclusions in respect of the 2009 collapse, and has no value in relation to new or emerging diseases.

The most significant limitation of Dr. Kent's work is that he intentionally chose not to examine the potential impact of fish farms in causing or exacerbating diseases in the wild salmon population. As a result, he did not examine the extensive databases of diseases in fish farms, and he did not examine the available literature showing the prevalence of disease in fish farms in other places.[32]

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[29] Project 1 Report, Ex. 1449, Executive Summary
[30] Project 1 Report, Ex. 1449, p. 23-24
[31] Project 1A Report, Ex. 1454, p. 98
[32] Dr. Kent did not read or cite Ex.s 1482 – 1487, Rimstad et al., "Examples of Emerging Virus Diseases in Salmonid Aquaculture", Robertson et al., "Can We Get the Upper Hand on Viral Diseases?", Mennerat et al., "Intents of Farming – Evolutionary Implications for Parasites and Pathogens", Reno, "Factors Involved in the Dissemination of Disease in Fish Populations", Walker, Winton, "Emerging Viral Diseases of Fish and Shrimp", Ford, Meyers, "A Global Assessment of Salmon Aquaculture/Wild Salmonids". Given the titles of these papers,

In his Report, in answer to a reviewer's comment, it questions relating to fish farms as a cause of disease needed to be addressed, Dr. Kent responded, "Fish farms and sea lice are dealt with in more depth in another report."[33]

In testimony, Dr. Kent confirmed:

> "Q The question I am asking is whether your report didn't cover the problems from fish farms, regardless of the reason why.
> DR. KENT That's correct.
> Q It didn't, did it?
> DR. KENT It did not."[34] …
>
> Q So you didn't do this fish farms in your report because you felt they were being done in another time; is that fair to say?
>
> DR. KENT And this was following discussions with the Commission. When I had this review back, I discussed this with Dr. Levy about should I expand this, based on the limitations in my report and the time, and then following the discussions of Dr. Levy, that was the decision, to leave this for the fish farm issues."[35]

This was an unfortunate oversight. In the result, both Dr. Noakes and Dr. Korman, in Project 5, in addressing fish farms, made the decision to focus on the diseases identified by Dr. Kent (presumably without understanding that Dr. Kent had intended to leave fish farm diseases to them). Then that result is that no one (other than Dr. Dill) covered diseases from fish farms. No one focused on the extensive literature available in the science to show the inevitable results of disease causation from fish farms.

On the other hand, Dr. Stephen, who was asked to address hatcheries, did focus on the potential mechanisms by which hatcheries could cause disease. Although Dr. Stephen also found that research was severely limited, he was able to confirm that causation was reasonably plausible. Dr. Stephen's conclusions and observations on this point have as much or more relevance to fish farms, as they do to hatchery facilities. Fish farms, which sit in the middle of salmon migration routes, with open nets, would have a much greater likelihood of transmitting pathogens to wild salmon swimming past them.

There will be some who attempt to mistakenly (ab)use the absence of conclusions from Dr. Kent and Dr. Stephen as if it were an exoneration of fish farms. Nothing could be further from the truth. The Commission should be careful to distinguish the inability to draw conclusions based on a lack of research from any suggestion that harm from disease had been disproven. The

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and the journals, in which they were published, these papers would have been readily available on even a cursory literature search.

[33] Ex. 1449, p. 55-56

[34] August 23, 2011 Transcript, p. 27

[35] August 23, 2011 Transcript, p. 28

Commission, having created the Terms of Reference for Project 1, bears some responsibility to clarify that outcome.

The real value of Project 1 is to identify the total failure of the regulatory authorities in the Province and Canada to have done proper research in respect of disease in the wild salmon populations as part of their stewardship responsibility and failure to have done research into the transmission of pathogens by fish farms prior to citing such farms in the path of migratory wild sockeye. [See "Disease not Studied" and "The Absence of Science" in Part II]

Drs. Kent and Stephen were unanimous in concluding, as their major recommendation, that pathogen research in wild salmon populations must be urgently upgraded. The Commission should consider adopting and promoting these recommendations.

RECOMMENDATION: ***DFO should prioritize scientific research that impartially investigates the impacts of aquaculture on wild salmon stocks and the ocean ecosystems and aimed at ensuring the conservation of wild salmon and salmon habitat. Research into disease in wild salmon and aquaculture; and disease interactions between the two, be given highest priority.***

(b) The Project 5 Reports

> "**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."** *Dr. L. Dill, Project 5D*
>
> **"The results of this analysis suggest that increasing farmed salmon production…increases sockeye salmon mortality. In addition, the influence of aquaculture production on sockeye mortality was predicted to be greater when SST [Sea Surface Temperatures] anomalies are negative"** *Dr. B. Connors, Project 5B*

The Commission's process for the Project 5 Reports was unusual. Rather than seeking one neutral scientist to author a single Report (opposed by the BCSFA), Commission staff consciously identified a scientist from each 'side', and commissioned a Report from each. While that process seems fair, it must be recognized that the inevitable consequence of that choice is to produce the appearance of 'dueling' science.

In such circumstance, it might be tempting to conclude that "the science is controversial", or that in the absence of consensus, "no conclusion can be reached". Given that the Commission, through its process, has constructed the very scenario of opposing viewpoints, to do so would be an abdication of the Commission's responsibility.

It is a common scenario in trials that credible experts are called to put forward opposing viewpoints. The task of the trial judge is to weigh the evidence and the opinions, and if need be, the credibility of the expert. In respect of Project 5, the Commissioner may need to call upon these fact-finding skills.

It would be a mistake to attempt to combine the evidence, and search only for points of agreement – i.e., reduce the evidence to its lowest common denominator – as was done in part in the Project 6 Addendum Report. While there is value in realizing the common points of agreement, it would not justify rejecting the other important evidence simply because one expert rejected it, especially where that expert was chosen for his pre-existing bias.

The Statistical Approach

It is questionable as to why the Commission sought to employ, as a primary strategy, a search for statistical proof. That is not to blame Dr. Korman, Connors or Noakes, all of whom are primarily statisticians, and not experts in fish health. They did what they were asked to do. However, it must be noted that 'disease' and particularly 'epidemic disease' is not necessarily subject to statistically precise patterns. By its very nature, epidemic disease happens occasionally and often unpredictably. To expect a predictable pattern, based on slight changes in population or production, that could be exposed through statistical analysis would not be the first choice in looking for evidence.

In population disease, the normal course to find patterns would be to look to ecological science.

That is all the more true in this case, where only a few years of data were produced by fish farmers. Although they have been operating for 20+ years, only 5 years of usable data was available. In that circumstance, it is no surprise that any statistical correlation would be either difficult to find, or a very weak one.

It is all the more significant then, that Dr. Connors was able to find a statistical correlation.

Dr. Connors found a statistical connection between increases in farm salmon production and sockeye salmon mortality.[36]

In looking for empirical evidence through statistics, the Commission was looking in the wrong place. The correct approach was the one taken by Dr. Dill, the only ecologist called. Dr. Connors conclusion simply provides some evidentiary support for the predictive models of ecological science.

It is respectfully submitted that the evidence of Dr. Connors and Dr. Dill should be preferred over Dr. Noakes.[37]

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[36] Connors' Report, Executive Summary and p. 22

[37] Dr. Noakes was clearly biased. Dr. Noakes support of fish farms goes back many years. Dr. Noakes, as early as 2000, was prepared to author, along with Dr. Beamish and Dr. Kent, the paper "On the decline of Pacific salmon and speculative links to salmon farming in British Columbia" Exh 779 which, without evidence excused aquaculture.

The Ecological Approach

Dr. Dill's approach was based on ecology. Dr. Dill's approach is careful and measured, and appropriately considered benthic impacts, escapes, sea lice and other potential negative effects of fish farms, and was prepared to recognize that they were unlikely causes of population-level declines for sockeye. His conclusions about disease therefore deserve greater credibility. Dr. Dill described the risk of 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."[38]

In relation to the Discovery Island Fish Farms, Dr. Dill points out:

> "…The findings of a long term analysis suggest that high standing stocks of fish and floating net pens in the Discovery Islands can negatively impact Fraser sockeye survival…"[39]
>
> "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 farm fished 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 infected particles."[40]

Dr. Korman's approach

Dr. Korman's report and spreadsheets deserve comment. Dr. Korman provided a fair and useful accumulation of the data, and many useful charts. However, it should be noted there are three critical limitations in Dr. Korman's analysis, each of which he accepted in his testimony:

(a) he significantly underestimated mortality by counting only 'fresh silvers';

(b) he used only confirmed diagnoses of known diseases – ignoring the 'open' diagnosis where fish had died of unknown disease, constituting 60% of the diagnosis – thereby severely underestimating the amount of disease; and

(c) he counted only 'farm-level' disease diagnosis, and did not account for the very significant numbers of diseased individual fish.

Dr. Korman's report is therefore a very significant underestimation of the magnitude of existence of disease on fish farms. Dr. Korman prepared the data in good faith, and gave his evidence in a

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His views haven't changed. Also, evidence of his partiality can be found in the interchanges with Dr. Connors, and in fact, in his decision to file supplemental work critical of Dr. Connors –whose methodology was confirmed by Dr. Korman.

[38] Report 5D, Ex. 1540, p. 24

[39] Ex. 1540, p. 15

[40] Ex. 1540, p. 27

fair and balanced way, but the resulting tables and statistics must be reconsidered in light of the other evidence.

Dr. Noakes however relied on Dr. Korman's database, and further misconstrued it.[41] Dr. Noakes' statements that the majority of the audit cases had "no sign of disease" and that the "mortality rate on farms is quite low" cannot be sustained on a review of the actual data.[42]

Dr. Korman, and through him Dr. Noakes, relied on Dr. Kent in choosing the diseases to focus upon -- however, as noted above, Dr. Kent did not focus on fish farm diseases. This is an additional limitation to the approach taken by Dr. Noakes in particular.

The net result is that no one of Dr. Kent, Dr. Korman or Dr. Noakes examined the possibility of a new, unknown, or emerging new strain of disease.[43] Dr. Noakes' willingness to dismiss the risk of known disease as "minimal and likely undetectable" simply because those diseases are endemic,[44] shows a complete lack of awareness of the science referred to by Dr. Dill and represented by Exhibits 1482-1487. It also shows a willingness to jump to conclusions without evidence, which must be contrasted with Dr. Stephen. Dr. Noakes identified the absence of disease data for wild fish as being "a serious deficiency"[45], but whereas Dr. Stephen flagged that as "no conclusion possible", Dr. Noakes was prepared to conclude there was no impact. This conclusion says more about Dr. Noakes' credibility and partiality than it does the evidence.

The DFO workshop and the PSC workshop both concluded that disease was a likely or very likely cause of the decline of the wild salmon.[46] This scientific consensus is at odds with the work of both Dr. Kent and Dr. Noakes. If disease was a significant causative factor in the 2009 decline, what disease was it, and where did it come from? The work of Dr. Noakes provides no assistance in answering that question.

4. THE DISEASE DATABASES

The unprecedented amount of material provided to the Commission regarding fish health and disease in fish farms (particularly that in Exhibit 1549) is an opportunity, never before available to the public, to examine what is really happening with disease on fish farms.

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[41] It is clear that the statement made by Dr. Noakes at p. 25: "The vast majority of the audit cases tested negative with no sign of disease in the histopathological examinations," (emphasis added) is quite wrong. It is clear that Database 2864 (Ex. 1549) shows signs of disease in almost every fish.

[42] Dr. Noakes' comparison of the fresh silver mortality rate on farms to the juvenile fish in the wild is indicative of his bias.

[43] Dr. Korman notes, at p. 10, Ex. 1543, that the records do not show the presence of "new or novel high risk pathogens", but apparently did not connect that with the 60% of open diagnoses. Dr. Noakes discusses the genomic signature found by Dr. Miller (at p. 31-32) but seems to dismiss it as relevant to his paper.

[44] P 32-33 of Dr. Noakes report Exh. 1536. Dr. Korman's report at p.7 also refers to 'diseases are endemic'

[45] p. 34

[46] Ex. 203- Peterman, et al "Synthesis of Evidence from a Workshop on the Decline of FRS"; Ex. 1364, "Draft Summary Report: DFO Synthesis Workshop on the Decline of FRS, April 14-15, 2011"; Ex. 614, "Update on Science Review 2009 Fraser Sockeye"

The record should show that the Province and the BC Salmon Farmers Association fought the release of this information with great vigor. Far from being released 'voluntarily', it was only through the order of the Commissioner, following Rule 18 and Rule 19 applications. Even after being ordered produced by the Commissioner, again at the Hearings, counsel for the BCSFA, the Province and Canada fought to protect it from becoming an exhibit.

We now know why.

The public has been told on many occasions by both fish farm industry and regulators that disease is not a problem on fish farms. Even in testimony before this Commission, in the face of the detailed evidence available, some witnesses from both the industry and government persisted in this fallacy.

It has become clear that very, very few people ever had access to these databases, and apparently no-one in DFO. It was confined solely, it appears, to Dr. Marty and Dr. Sheppard, both acting on behalf of the Province, and perhaps Dr. Saksida, acting for industry. We know that the Science staff at DFO, including Dr. Miller, had no such access. They were required to rely on Dr. Marty and Dr. Sheppard's second-hand 'assurances' that the disease records showed no problems. Even after the databases had been released publicly, through the Commissioner's rulings, very few at DFO have read any of it – notably, none of the senior DFO staff – Dr. Richards, Ms. Dansereau, the Deputy Minister, Mr. Bevan, the Associate Deputy Minister, Ms. Farlinger, Regional Director General for the Pacific Region. [47]

This limited access to closely confined information must never be allowed to occur again.

It has become apparent that the only way in which anyone, including Dr. Marty and Dr. Sheppard, could contend that diseases were not present in fish farms is by a series of carefully contrived definitions, assumptions and practices, which combined to filter out any uncomfortable facts. These contrived practices included:

- Distinguishing between 'symptoms' or 'clinical signs' of disease, from an actual diagnosis of confirmed 'disease'
- Requiring full compliance with tight definitions for a single confirmed diagnosis – thereby over 60% of the diseased fish could be given an "open" diagnosis – even though quite dead, and filled with disease-symptoms constituting a potential cause of death – and then those 'open' diagnosis be <u>ignored</u> for statistical and data aggregation purposes, and never reported to the public or DFO
- Defining reportable disease as 'disease at a population level' or at the 'farm level' thereby being able to <u>ignore</u> for public purposes all diseases found in individual fish, even where such disease was from a confirmed diagnosis, and confirmed cause of death;[48]
- Transferring the responsibility to identify population diseases from the histopathologist, Dr. Marty, back to the provincial veterinarian, Dr. Sheppard, and then back to the fish

[47] Sept 26, 2011 Transcript p.71
[48] See Dr. Sheppard, Transcript, Aug 31, 2011, pg 85, line 17

farm veterinarian, 'who has knowledge of the situation on the farm',[49] so that even where serious disease were identified in a single sample or group of samples, it would be rare that any diagnosis was made.

(a) The "Open Diagnosis"

On review of the BCMAL disease databases, notably Ex. 1549-206 (BCP002850) and 1549-217 (BCP002864), which were the primary summary databases yheld Dr. Marty and Dr. Sheppard and provided by the Province, it becomes apparent that disease is highly prevalent in all the audits. How then could Dr. Korman and Dr. Noakes conclude that "the vast majority of the audit cases tested negative with no signs of disease"?[50] Quite clearly they did not look at the actual raw data, but relied upon the summary diagnoses by Dr. Marty and Dr. Sheppard. It is in this way that the fish farm industry is able to mislead the public by stating that there is little disease in the farms, when the audits show clinical signs of disease in almost all fish.

In Ex. 1549-206 (2850), a comparison between TAB 3-Histology, which shows clinical findings or references to disease in almost 75% of the audits, to TAB 18 FARMDX which shows an actual diagnosis in less than 40% of the cases. In TAB 18, the diagnosis is "OPEN" 152 times. We learned in testimony that an open diagnosis can be reached because there are two or more diseases found in a group of audited fish, or that symptoms may be indicative of a number of possible diseases, but not enough to state with certainty the particular disease. Thus the "open" diagnosis does not mean 'no disease', it means 'undiagnosed disease'. (Note: the finding "No significant findings" is used only 2 out of 583 times – the remainder is 'significant'). On an individual level, the equivalent is found in Exhibit 1549-217 (2864) under column J as 'none'. 'None' is used 55% of the lines, even though there are 'significant' lesions found that could be the cause of death (and the fish are dead). None is used wherever there are multiple possible causes or the actual cause of death is not determinable. Over 95% of the 2700 fish listed in that database show clinical findings or significant lesions.

As noted earlier, in Dr. Korman's compilation of these databases, at Exhibit 1544, the farm level diagnosis is shown as "Open" in 495 of 794 audits – i.e. **62% of the time**. This became his Figure 8.[51] Dr. Noakes relied on that as assuming the 'open' diagnosis as being benign[52] – which they are not. The BCMAL reports, reports only the #'s of incidents of 'diagnosed' disease.

Dr. Marty, firm in his diagnostic standards, confirmed to Dr. Miller:

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[49] Transcript Aug 31, 2011, pg 84-87, both Dr. Marty and Dr. Sheppard
[50] Report 5C, Noakes, Exh 1536, pg 25.
[51] Ex. 1544, pg 20
[52] Because Dr. Noakes looked only at those diseases identified by Dr. Kent as 'high-risk' and then only looked at the no. of confirmed diagnosis for those diseases in the Korman database. See Transcript, Aug. 29, 2011, pg 26, Line32-43 "What I relied upon…was the evaluation by Dr. Kent"

> ***"I am confident that some and perhaps many of the unknown causes are infectious diseases"*** [53]

It is clear that a new or emerging disease, without a diagnostic standard yet developed, will always show up under the "open" category. Similarly, salmon leukemia, which Dr. Marty "does not believe in" (see below) will go in the "open" category. If Dr. Miller's "parvovirus" is confirmed on fish farms, it will be seen to have been in the "open" category.

The most reasonable conclusion available to the Commission on the extent of disease confirmed in the BCMAL audits is that 62% of the time the cause of death is from an "unknown" disease.

(b) "Farm –level diagnosis" vs autopsy reports

Through the testimony of Drs. Sheppard and Marty we learned that the vet who does the autopsies on the farm salmon (Dr. Marty), is not the vet who gives the farm diagnosis (Dr. Sheppard). The farm diagnosis becomes the more public record and it was the farm diagnosis that Drs. Dill, Connors, Noakes and Kent used for their reports to the Commission on potential of disease to impact the Fraser sockeye.

The Aquaculture Coalition looked at the original autopsies and we argue this is essential because there is so much disagreement between vets on Salmon Leukemia, brain tumours and ISA virus. In our report, Exhibit 1976, we note that interstitial cell hyperplasia in the kidney is a reported in two scientific papers as a diagnostic of Salmon Leukemia. None of the vets involved with this disease, Kent Sheppard, Marty could explain why they no longer believe this lesion suggests Salmon Leukemia, they simply no longer diagnose it.

There is a similar disagreement over Infectious Salmon Anemia virus – is it exotic or not and has the proper testing occurred?

The seeming contradiction between Dr. Morton's's analysis that salmon farms are having significant impact on the Fraser sockeye and the analysis by other scientists who found no relationship is explained by differences between Dr. Marty's original autopsy reports and Dr. Sheppard's farm diagnoses. Only the Aquaculture Coalition examined the full extent of Dr. Marty's reports, including the raw data. This Commission must do the same.

(c) Database Indications of Salmon Leukemia (Marine Anemia)

Stephen et al (1996) give us the primary case definition by which marine anemia can be diagnosed:

"**hyperplasia of the interstitial cells of the caudal kidney."**[54]

[53] June 27, 2011 email to Dr. Miller, Ex. 1568, p.1
[54] Exh 1491 pg.421

In disease database 2850[55], the BCMAL audit records of histopathology of individual fish, the presence of interstitial cell hyperplasia in the kidney is carefully recorded for both Atlantic and Pacific salmon under the column "ISH", defined as:

> "Interstitial (hematopoietic) cell hyperplasia (kidney); ISH is evidence of increased demand for erythrocytes or white blood cells somewhere in the body. In Chinook salmon, this lesion is often associated with the clinical diagnosis of "Marine anemia"."

There is a dramatic difference in the prevalence and seriousness of interstitial cell hyperplasia shown in the audit results between Atlantic and Pacific salmon (Chinooks). Among the 447 Pacific salmon samples shown in the database, **291 (65%) are positive** for ISH.[56] Of these, 94 are rated '2' or '3' in severity. This compares to 175/2259 (7%) among atlantics, most rated 1.

Dr. Marty adopted a sorting methodology for the Atlantic salmon[57], but did not apply it to the the Pacific salmon tab. However, a comparison of the ISH column for the Atlantics with the ISH column in the Pacifics produces an obvious and dramatic difference. The same 'sorting' applied to this tab can be seen at Appendix B Table 1. There is no explanation on the record for why interstitial cell hyperplasia of the kidney is so much more severe and more common in the Chinook salmon than Atlantics other than the relative difference in susceptibility to Plasmacytoid Leukemia.

Dr. Marty later agreed that marine anemia would have to be determined by looking at the prevalence of the symptoms.[58]

Dr. Sheppard stated that BKD is "often concomitent" with marine anemia.[59] Notably, the diagnosis of Marine anemia, under provincial guidelines, requires the absence of BKD before a diagnosis of marine anemia can be made:[60]

> "A diagnosis of MA is considered in Pacific salmon populations if:
> the fish sampled have gross clinical signs of MA; histopathological legions of MA;
> the farm is experiencing population level losses; and severe BKD is not largely evident."
> (emphasis added)

In the ISH positive samples in Ex. 1549-217, there are a number of diagnosis of BKD (56) associated with the 291 that are positive. Many more have no diagnosis, or just "ISH" as the most significant lesion. In Ex. 1549-206 there are 70 diagnosis of BKD at the "farm level".

The significance of these findings is not that we can definitively state that these fish had marine anemia – but that they had some disease that caused interstitial cell hyperplasia, and no-one has identified what that is.

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[55] Exh 1549-217, Tab "Abbreviations" - ISH
[56] See Appendix B, Table 1 and Exh 1549-217, Tab "Atlantic" and "Pacific" Columns 'AT'
[57] Transcript Aug 31, pg 63 [Dr. Marty suggested this was evidence that ISH was associated with BKD].
[58] Transcript Aug 31,2011, pg 87,Line 17-21
[59] August 31, 2011, pg 90, line 6
[60] Ex. 1564, BCMAL Supplement to Fish Health Report 2009, p. 26

(d) Database Indications of Infectious Salmon Anemia Virus

It seems likely that it is a matter of <u>when</u> Infectious Salmon Anemia will strike in British Columbia, not if. The consequences can be catastrophic, including to wild stocks, and the risk associated with salmon farms on the Fraser migration route is not acceptable.

Infectious Salmon Anemia (ISA) is a disease of salmon that is present, and that has had devastating consequences for fish farming in all the major fish farming regions of the world (including Norway, Eastern Canada and the United States, Ireland, Scotland, Faroe Islands, and Chile).[61] Chile's salmon farming industry was almost wiped out from the disease in a matter of only a few years after the disease was first detected.[62]

We have recent evidence to suggest that ISA may already be present in BC waters, and we intend to ask the Commission to re-open the evidence to examine that possibility. Should ISA be confirmed, there is little doubt that the source will have been aquaculture facilities, and imports from other countries.

That there has not previously been a reported occurrence of ISA in British Columbia's coastal waters is not a comfort in light of its history of emergence wherever there is Atlantic salmon aquaculture. It seems likely that it is a matter of when the disease will strike in B.C., not if.

As Dr. MacWilliams noted, when ISA is found in Pacific salmon in British Columbia, it will be because of aquaculture (at a farm or by importation).[63]

And while Pacific salmon may be less susceptible to the disease generally, Pacific salmon species are nonetheless vulnerable, particularly in light of evidence that ISA mutates.[64] The amount of testing on sockeye salmon is quite simply inadequate to provide any assurance of safety. The impacts on wild salmon, if there is an outbreak are unknown, but potentially catastrophic.

As with other diseases, once there is an outbreak, the consequences can be devastating and very difficult to control. Certainly the regulatory and policy framework in place does not guard against ISA or its potential impact on wild salmon, in light of the fact that there are over one hundred fish farms on the migration route. The potential for ISA serves as an example of the need for a more/true precautionary approach by the department to fish farms and disease.

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[61] Ex. 1502, Vike et al, "ISA virus in Chile: evidence of vertical transmission", p. 1; Ex. 1464, Rolland et al, "Relative resistance of Pacific salmon to infectious salmon anaemia virus", p. 511

[62] Ex. 1502, ibid, and Ex. 1976, Morton, "What is Happening to the Fraser sockeye", p. 30; Ex. 1687, Preventive Fish Health Work

[63] Aug. 23, 2011 Transcript, (MacWilliams), p. 47:41-46

[64] Ex. 1464, Rolland et al, "Relative resistance of Pacific salmon to infectious salmon anaemia virus", p. 511

The only data we have access to regarding the presence of disease in fish farms is the Fish Health databases provided by the province.[65] The databases either 1. rely on reporting by industry of mortality in fish which resulted in a histopathological exam and report by a provincial veterinarian or 2. are data from fish health audits conducted by the province- which audits were voluntary by farms and, in terms of sampling, consisted of removing a few dead fish the farm for testing. PCR testing for ISA has been performed on a very limited number of occasions, given the 30 million fish in BC waters each year.

There is currently no formal testing program for ISA on BC fish farms[66]. There never has been. Such testing as has occurred has been sporadic, through the audit program on about four fish per facility per year.

Dr. Marty testified that the goal of testing for ISA that currently occurs

> "The audit program, the goal of that program is to audit the fish health events that are reported by industry. So we are not attempting to certify any individual farm free from disease."[67]

Dr. Sheppard testified "the program is designed with a confidence of 95 percent at a two percent prevalence".[68] Given the catastrophic potential consequences of an ISA outbreak, this does not suffice.

Prior to receiving the databases, Dr. Morton has raised her concerns regarding the potential for an ISA outbreak in British Columbia waters on numerous occasions. Though aware of the catastrophic effects of ISA in other areas of the world, the Minister and/or representatives of DFO have consistently disregarded those concerns, relying heavily on Canada's egg testing policy as sufficient protection. [Canada's position with respect to concerns about ISA is discussed further below (under "Egg Imports" in Part 2- Regulatory Environment").]

We submit those protections have been inadequate to provide any such assurance, and that, if ISA should be found, Canada's continuing willingness to have put wild salmon at risk is nothing short of criminal negligence.

In our submission, there is no sure way to protect against ISA coming to fish farms in British Columbia . It will come, and probably already has done so.

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[65] DFO has not separately regulated nor monitored disease on fish farms, nor had access to or reviewed the databases or audits. Since accepting and exercising their jurisdiction over farms starting in Dec. 2010, DFO purports to have a reporting and auditing system in place, though we were not provided with evidence of reporting nor audits occurring.

[66] Proper testing requires 60 fish per facility – that is not done. Discussed further in Part II. See Manual of Compliance; Ex. 1567, "International Response to Infectious Salmon Anemia- Prevention, Eradiction and Control", p. 26; see also, Ex. 1976, Morton, "What is Happening to the Fraser Sockeye", pp. 33- 37

[67] Aug. 31, 2011 Transcript, p. 55:30-32

[68] Aug 31, 2011 Transcript, p. 58: 5

With respect to histopathology, symptoms consistent with ISA are described **1,100 times** in the fish health databases.[69] We do not have, so cannot analyze databases for health/disease of wild salmon, as those do not exist.

In the BCMAL audit database, [70] the column HEM (Column AY) is defined:

> "Hemorrhage/congestion (interstitial, kidney); HEM probably is a nonspecific result of endothelial damage; HEM is often associated with VHSV and bacterial infections. Renal congestion and hemorrhage is one of the classic signs of infectious salmon anemia (ISA), but ISAV has never been isolated from fish in BC."

The column SSC (Column AF) is defined:

> "Sinusoidal congestion (liver); SSC is a nonspecific result of sinusoidal damage. In BC Atlantic salmon, sinusoidal congestion is an uncommon feature of infection with viral hemorrhagic septicemia virus (VHSV) and *Listonella anguillarum*. Sinusoidal congestion is one of the classic lesions associated with infectious salmon anemia virus (ISAV) infection, but ISAV has never been identified in British Columbia."

These columns produce the following table, adjusted to include only Region 3 results:

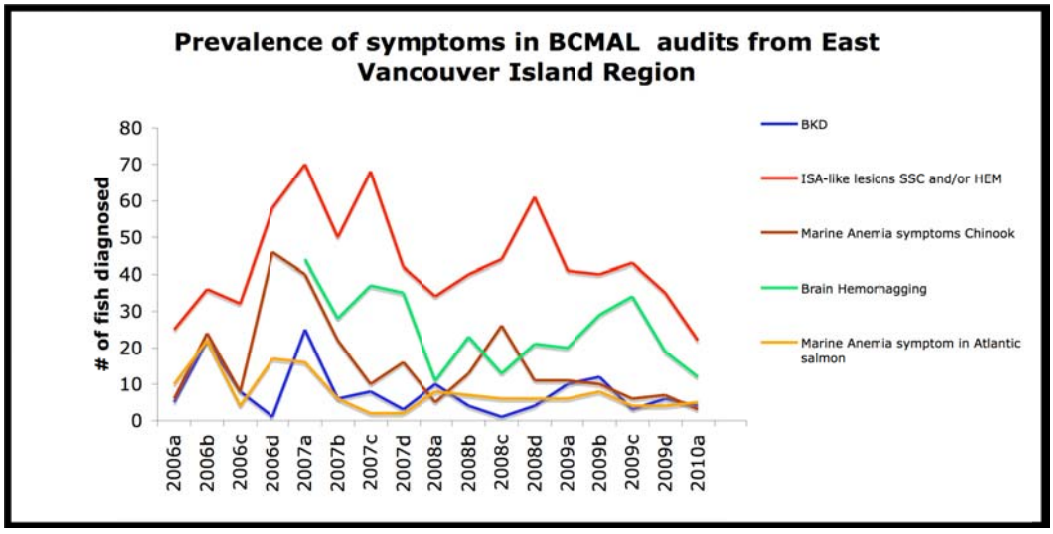

Figure 1: These data are the BCMAL audit data recorded in BCP002864. Of note is the suggestion of diagnostic pulses rising and falling in concert with each other. These do not represent prevalence only the symptoms found in farm salmon that were collected on the day of the audits, freshly dead and identified as "silvers" by the industry. Presumably, the methods are consistent across years. [71]

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[69] Ex. 1976, Morton, "What is Happening to the Fraser sockeye?", pp. 30-32
[70] Ex. 1549-217(2864)
[71] This chart - Exh1983 - is prepared from prevalence of symptoms found in Ex. 1549-217 in columns AF (SSC in liver) and AY (HEM kidney) for "ISA-like lesions" and column AT (ISH in kidney) for "Marine anemia symptoms" Atlantic and Pacific (Chinook) tabs. The numerical support is outlined at Exh 1983.

The numerical support is outlined at Ex. 1983. It should be noted that these charts are based upon random audits of fresh silvers. Only about 400 fish per year are examined in the audit, out of total mortalities in the range of 3 million fish mortalities reported by the fish farms. (see "The Rate of Mortality" above). Assuming these audited fish are representative of the 2 million plus fish that might die of disease each year, each audited fish may represent 5,000 fish with a similar disease or symptom. Considering only 400 fish per year are examined (and fewer than that for Region 3), the fact that the total number of ISH, HEM, SSC, brain hemorrages or BKD symptoms each year are in the range of 46 (2010a) to 195 (2007-first qtr) is significant.[72] Those numbers can be multiplied by 5000 to get a real world equivalent.

The fact that the symptoms seem to peak in the first quarter of 2007 may also be telling.

There is also a notable conjunction between the symptoms of ISA and the symptoms of ISH. We do not suggest these two diseases are the same, but it bears some scrutiny as to why their prevalence should happen in some apparent relationship.

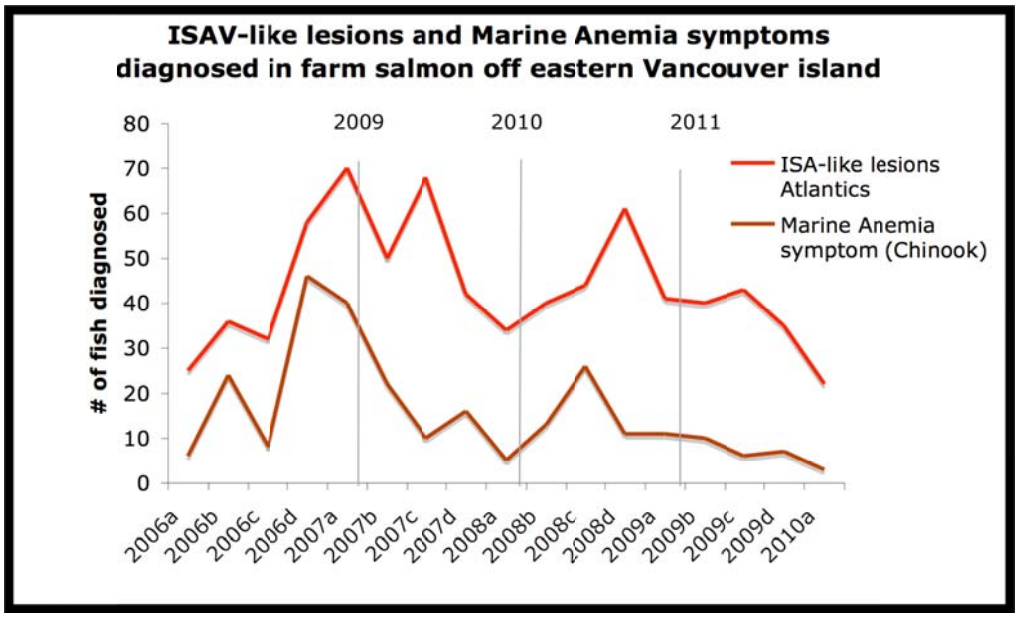

Figure 2: This graph is a subset of the same data. A spike inISAv-like lesions follows a spike in Marine Anemia symptoms in 2006, 2007 and 2008. The ISAv–like lesions are in Atlantic salmon, Marine Anemia in farmed Chinook. The Atlantics also have interstitial hyperplasia of the caudal kidney distinctive of Marine Anemia, though there is no record of confirmation of the disease. In areas of high salmon farm density, such as the Discovery Islands, salmon farm –origin pathogens were co-occurring and the Fraser sockeye were thus exposed to suites of potentially novel and amplified pathogens.The lines under "2009, 2010, 2011" mark where the Fraser sockeye that returned in those years passed these salmon farms as smolts..[73]

[72] See numerical table, Exh 1983
[73] Ex. 1983

There are numerous cases in the databases where Dr. Marty notes multiple classic diagnostic lesions for ISA virus. Given that this disease is notoriously difficult to detect these reports should have triggered the testing required as per Canada's Manual of Compliance to designate a population free of a suspect pathogen. We found no evidence the required sample size of 60 salmon were ever tested in a suspect farm. The letter from the CFIA responding to my reporting of BCMAL ISAv lesion diagnostics suggests the sum total of their response was to call the salmon farm vets. This does not meet the Manual of Compliance standard to clear BC farm salmon of the stigma of ISAv suspicion.

If a single PCR test was all that was required to detect all strains of Infectious Salmon Anemia virus the OIE would clearly state this. But they don't, they say, "There is no gold standard test for ISAV, and the confirmation of an infection depends on a combination of test results…This highly contagious disease can be insidious, with an initially low mortality rate."[74]

(e) Other Exotic Diseases

No one seems to know where Salmon Leukemia came from. On occasion, Atlantic salmon imports were screened for it, but not for the eggs imported before the first epidemics. Dr. Kent and others, who published on it, have treated it as an endemic virus, but with *very little* evidentiary support.

The literature from other places notes a host of other exotic or emerging new diseases, for which there is little preparation in B.C. These include ISA, Salmon Alphavirus, Heart and Skeletal Muscle Inflammation (HSMI), Cardiomyopathy, Pancreatic Disease (PD), Infectious Pancreatic Necrosis (IPNV) and others. [75]

However, there are four exotic viruses apparent in Dr. Marty's reports. He reports the lesions associated with Salmon Alphavirus, Heart and Skeletal Muscle Inflammation, Cardiomyopathy and a Chilean coho farm virus.[76] These are emerging pathogens, meaning the causative virus has only recently been identified. However, the diseases they cause have a much longer history among the North Atlantic Ocean fish farm industry (Norway, Scotland, N.B. etc), ie they were spreading and infecting farms before there was a known cause. Atlantic salmon eggs imported into BC were not screened for these new 'known' viruses even though they were prevalent in the North Atlantic.

By definition, egg imports cannot be screened for new diseases, before they are discovered, and before testing mechanisms have been designed. There is literally no defence for new diseases, other than a complete ban on egg imports from other countries.

As a result, the border is wide open to importation of exotic disease, a concern with a long history voiced by individuals in the provincial and federal government since 1985.

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[74] Morton report Exh. 1976
[75] See Journal articles, Ex. 1483,
[76] See Appendix B

Our only defense would be rapid response to signs of it appearing the farms, but these reports are not reaching even DFO's government scientists. The entire evaluation of whether these are a risk or not has rested (and still rests) on one man, Dr. Mark Sheppard (formerly with the Province; now with DFO). And there is no comfort to be found in the evidence of 'rapid response' to emerging diseases.

Further, as is discussed in Part II- Regulatory Environment below, Canada has failed to put in place adequate safeguards to protect against the introduction of exotic diseases.

(f) Ongoing critical testing

The Aquaculture Coalition would like to draw the Commission's attention to two disease projects that are underway and could have critical impact on our understanding of the potential for salmon farm impact on the Fraser sockeye.

1. We heard evidence that the salmon farming industry was going to discuss a farmed salmon testing protocol with Dr. Miller after the hearings. This is essential testing and it would be in the public interest for the Commission to follow up on this and request the results. Salmon Leukemia is the best lead we have to the demise of the Fraser sockeye;

2. Exhibit 1527 references a 'jaundice – related' condition that has been killing farmed Chinook salmon for 7 years. Because Dr. Marty has been reporting symptoms of a virus in Chilean Coho that could be a form of ISAv and because Exhibit 1686 is an email discussion between Dr. Saksida and Dr. Garver about whether this could be Salmon Leukemia the results from this project should be provided to Justice Cohen before making his evaluation of the potential impact of salmon farms on the Fraser sockeye.

The Aquaculture Coalition has also noted a series of recent actions by the salmon farming industry that appear to be a heightened response to the threat of an exotic virus, and specifically Infectious Salmon Anemia virus. The Norwegian companies have constructed their own disease management process that fades completely away from public scrutiny as they step outside the government audit process.

1. August 2009 – until last available record (July, 2010), Marine Harvest begins unprecedented requests to Dr. Marty for PCR tests for the exotic virus Infectious Salmon Anemia[77]

2. April 2010, the salmon farming companies halt government access to their dead fish and the provincial audit process ends[78]

[77] Exh 1549-318 and 322
[78] Ex. 1688

3. April 16, 2010 the Norwegian companies operating in BC sign an MOU without government to "manage viral fish diseases and to minimize spread…"

4. The companies stop Atlantic salmon egg imports in 2010, even though government allows it.

(g) Disease Information will be secret in future under DFO regime

It became clear through the hearings that farm salmon disease information was not available to DFO managers or scientists. As noted above, it appeared in this Commission that this significant body of information was only available to Drs. Gary Marty the BCMAL farm salmon pathologist and Mark Sheppard, who was making the farm diagnosis based on Marty's reports.

While DFO is responding to public concern by maintaining that salmon farms are not a threat to wild salmon, senior Pacific Coast management told the Commission they have never examined the salmon farm disease records.

None of this evidence was available to DFO scientists until this Commission had it produced. None of it was available to non-DFO scientists doing research.

Under the proposed new Licence, the only evidence required of disease will be only in the case of fish health events", and there a one word description of 'Diagnosis" (for the whole event will be sufficient.[79]

No information in respect of testing of individual fish would be available.

None of the information present in Exh 1549-217 would be producible by the fish farm to DFO under this regime.

There are no plans to make public audit data, as was done before the Commission. This will be the last time any such information will ever be seen. The public interest is in knowing what emerging symptoms or individual diseases are being found.

The pretext that DFO will be following a 'transparent' model for fish health information is completely false.

We are going backwards. DFO itself will never have this information as regulator.

5. Salmon Leukemia and DFO Lack of Response

The capacity and practice of DFO in response to a new disease in fish farms can be observed in relation to the plasmacytoid leukemia epidemics of 1988-1991.

[79] Exh 1594, Licence, and see Appendix VIII, pdf p. 35 Section C

Dr. Kent and Dr. Stephen both testified, and a number of their papers are in evidence.[80] At the time of this disease epidemic, Dr. Kent was a senior scientist with the Department of Fisheries and Oceans at the Pacific Biological Station.

- Dr. Kent, identifies this was an "apparently new disease", first recognized in the fall of 1988.[81]
- He identified the disease as a form of "plasmacytoid leukemia". He later named the disease agent "Salmon Leukemia Virus".
- His experiments, in 1990, showed that the disease was "infectious" and transmittable to other species including coho, sockeye and atlantic salmon. Sockeye were found to be "very susceptible". [82]
- He confirmed the disease was caused by a retrovirus (reverse transcriptase activity was detected in infected tissues).[83] Dr. Stephen conducted experiments with "purified virus"
- the disease was quite virulent ("mortality was 80% at one site")[84].

The study by Dr. Kent that confirmed that sockeye salmon were "very susceptible to PL" in the laboratory, was published in 1991. What actions were taken by DFO to protect the sockeye? The simple answer is "none". Dr. Kent, and later Dr. Stephen, continued to do some studies, but notably no studies were undertaken to determine the impact on wild sockeye, or the rate of transmission to sockeye. The operational or regulatory arm of DFO took no action whatsoever.

The risks to sockeye were apparently well recognized:

> Dr. Kent: "The occurrence of PL in sockeye and atlantic salmon further emphasizes the importance of this disease. The commercial fishery in BC relies heavily on wild sockeye salmon and many farms are starting to rear sockeye in net pens. The culture of atlantic salmon is already well established in BC. The economic significance of PL is still not fully realized."[85]

> Dr. Stephen "Evidence supporting the hypothesis that marine anemia is a spreading, infectious neoplastic disease could have profound regulatory effects on the salmon farming industry"[86]

Eventually, Dr. Kent did *one* study that found that the disease was now present in a small sample of wild Chinooks. That study was published in 1994[87]. The purpose of that study was described as follows:

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[80] Ex.s 1488 to 1494
[81] Ex. 1488 He believed the same disease might have been seen once before, at a fresh water hatchery in Washington State, but never in BC waters.
[82] Ex. 1489, p. 162.
[83] Ex. 1488, p. 5680.
[84] Ex. 1489, p.159
[85] Ex. 1489, p. 165.
[86] Ex. 1492, Stephens and Ribble (1995)

> "…whether SLV is present in wild or wild-caught populations of chinook salmon, whether it causes disease in them, and could any interaction between such fish and those in hatcheries or netpens contribute to an increase in PL"

The study found histological changes 'consistent with mild or early PL' in 7 chinooks. The study specifically advised that it "does not answer the question of the origin of SLV"[88]. It posited that the positive salmon could have come from netpens or hatcheries or obtained the SLV from netpens. It also stated that it might be "possible" that it was 'endogenous'[89], but that the virus would "be enhanced in the high density environment associated with aquaculture". The authors recommended the questions associated with wild stocks "warrant further attention".[90] In other words, whether the original disease in 1988 had come from wild fish, or whether the epidemics of 1988 to 1991 in fish farms had transferred the disease to wild chinook could not be determined. No subsequent study was undertaken to determine that. No study was undertaken at all on wild sockeye. Why not?

By the late 1990s, any further study in relation to this disease was abandoned. There was no funding, and Dr. Stephen and Dr. Kent moved on.

However, by 1998, Dr. Saksida was prepared to baldly state: "a survey performed by Stephen et al. found the disease to be an endemic problem"[91] – based on this one study that specifically did not determine that. That is, on the face of the study, an incorrect statement. There is a strange dichotomy between the amount of proof apparently required by DFO to comprehend a risk of disease transfer from fish farms to wild salmon, and the extraordinary low proof by which they will accept that disease is 'endemic' and thereby ignore any risk of disease thereby from fish farms. Such an extreme dichotomy can only amount to "willful blindness".

Once the disease had tapered off at the fish farms, and was no longer an economic problem to the aquaculture industry[92], it appears that it ceased to be of interest to DFO.

DFO's jurisdiction and obligation to protect the wild sockeye existed strongly in those years from 1988 through the 1990s, as it does today. Although aquaculture was regulated by the province, the responsibility for the wild fish remained with DFO. Could it be that it never occurred to *anyone* that this epidemic of fish farms could have adverse impacts on wild fish? Or is it that no one cared?

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[87] Exhibit 1493, Eaton (1994). The purpose of this study is described at pp. 147-8
[88] Exh. 1493, pg 150
[89] At pg 150, relying on a lab study by Kent 1993, the authors suggest that horizontal transmission 'occurs rarely'. This is highly illogical – if it's not likely to have gone from fish farms to wild salmon because of low horizontal transmission rates (even in a fish farm with epidemic levels of the disease), then why should it be so readily assumed that it came from wild fish that had extraordinary low incidents of the disease.
[90] Exh. 1493, pg 150
[91] Ex. 1796, (Introduction, para 1, p. 107)
[92] Apparently many farms had switched to atlantic salmon

(a) Time that DFO takes to React to New Diseases

What does the history of Salmon Leukemia tell us as to how DFO would react to a new disease that emerged in fish farms today? Clearly, DFO would "study the disease" for a number of years.

Salmon Leukemia and the Fraser sockeye timeline

1990 – 2010 farmed Chinook exhibiting "interstitial cell hyperplasia," (the leading diagnostic for Salmon Leukemia), placed across the Fraser sockeye migration route.

1988-1992 : Epidemics of 'Plasmacytoid Leukemia' devastate Chinook farms

1992 – Decline in Fraser sockeye productivity begins, with the notable exception of the Harrison run which Tucker et al (2009) suggest do not travel the eastern Vancouver Island shoreline where 75% of the salmon farms are sited.

1994- Dr. Kent publishes paper showing Plasmacytoid Leukemia found in wild chinook

1995 – Abnormal early entry behavior and pre-spawn mortality begin to be noticed

1995-1999: Dr. Kent and Dr. Stephen stop research on plasmacytoid leukemia

2006 - Genomic research suggests pre-spawn mortality and early entry are caused by a virus killing and/or weakening large numbers of Fraser sockeye.

2008 – 2010 The specific cellular pattern of the genomic profile and the physical condition of the Fraser sockeye leads DFO research scientist, Dr. Miller, to consider Salmon Leukemia virus and make a request to test farm salmon.

2009 - DFO Science responds by preventing Miller from attending closed scientific meetings on the Fraser sockeye crash, prevents her from discussing her hypothesis on timing with the Pacific Salmon Commission, prohibits her from speaking to the media and cuts her funding for sockeye research.

2010 – Farmed Chinook salmon are removed from the Fraser sockeye migration route in June 2007 and the first generation of Fraser sockeye that did not pass Chinook salmon farms return in historic abundance.

The approach to Dr. Miller's work, which has been on-going since 2007, without any action by DFO, other than further study is a further textbook example of what DFO is likely to do in the face of any new disease outbreak.

(b) The Nature of Marine Anemia

There seems little doubt that 'Marine Anemia' – Salmon Leukemia – is a disease. It certainly was fatal in the earlier epidemics. Yet Dr. Marty refuses to diagnose it, and 'does not believe in

it'.[93] Dr. Sheppard also testified that he "disagreed" with Dr. Kent and the cause of death for the 1988-1991 epidemics (although he was not there and could not say what that disease was).[94]

It is no wonder the disease is no longer reported by provincial reports, since those are the two individuals who were responsible for the reporting.

Despite Dr. Marty and Dr. Sheppard's reluctance, the Province's Fish Health Report, as of 2009, still recognized Marine Anemia (Salmon Leukemia) as a "disease of farmed Pacific salmon":[95]

> **Marine Anaemia (MA):** An endemic disease of farmed Pacific salmon characterized by marked gill pallor, enlarged kidneys and spleens, ascites and exophthalmia. The cause of this disease may include a retroviral infection and/or an intranuclear microsporidian, *Nucleospora salmonis*. Marked haemoblast proliferation in specific organs is the histopathological hallmark of the disease. Grossly MA can appear similar and concurrent to BKD. A diagnosis of MA is a considered in Pacific salmon populations if: the fish sampled have gross clinical signs of MA; histopathological lesions of MA; the farm is experiencing population-level losses, and severe BKD is not largely evident. Atlantic salmon do not appear to be afflicted by this form of marine anaemia.

There is obviously something wrong with the system when the contrary opinion of one veterinarian – Dr. Marty or Dr. Sheppard – can lead to a failure of diagnosis for all the years in which Dr. Marty has been in charge of the provincial histology. Other veterinarians continue to recognize the disease[96], and although Dr. Kent says that in his work in the US lately he finds the causative agent more often the microsporidium, he has not changed his mind on his original research. Dr Kent and Dr. Stephen's work clearly show the presence of a virus, that is infectious, and that can produce 'purified virus'.[97]

It is notable that even under provincial diagnostic test above, there would have been no diagnosis of 'marine anemia' whenever BKD is also present. An examination of the disease databases shows that BKD is often present in association with the symptoms of marine anemia. Dr. Kent and Dr. Stephen note in their papers that BKD is often associated with marine anemia.

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[93] Ex. 1568, June 27, 2011 email chain from Dr. Miller.In his response to Miller, Dr. Marty says "Marine anemia is probably better characterized as a syndrome, (a set of symptoms that appear together), rather than a specific disease. With a syndrome the cause is not necessarily known"

[94] Aug 31, p. 90

[95] BCMAL Fish Health Report, Supplement, Ex. 1564, p. 26

[96] Eg. Ex. 1686, Email from Garver to Traxler (Jan. 28, 2009)

[97] Ex. 1490, Eaton and Kent, "A Retrovirus in Chinook Salmon", p. 6498, and Exhibit 1493 Eaton, Kent et al 1994, pg148 "Analysis of this sample showed the presence of a virus – Note also in that study "The microsporidian Enterocytozoon salmonis was not found in any fish examined in this study". This substantially reduces the credibility of Dr. Sheppard's claims to the contrary.

A review of the disease databases shows there are over 70 confirmed diagnoses of BKD[98] at a farm level. Many of these are associated in the database with the symptoms of marine anemia.[99] How many of these are also salmon leukemia is unknown, since Dr. Marty and Dr. Sheppard do not believe in the disease.[100]

However, in the end it does not matter whether plasmacytoid leukemia (marine anemia) is a 'disease' (Kent/Stephens) or a 'syndrome' (Marty) – it is equally of concern if it is infective and it kills fish.

It is also highly relevant if associated with the early entry/PSM behavior or the MRS found by Dr. Miller.

(c) Is Salmon Leukemia still a problem- the 'disease of confinement'

> "The environmental conditions created by intensive aquaculture may have facilitated the emergence of marine anemia. Rearing systems used in seapen aquaculture represent a substantial change in the ecology of Chinook salmon. … If marine anemia is a "disease of confinement" then its discovery may simply be a reflection of the recent rapid growth of the British Columbia salmon farming industry"[101]

There is no reason to believe that salmon leukemia is any less prevalent (on Chinook farms) than it was in the 1990's. At that time the disease was present in most fish farms at about a 6% rate.[102] Much of the industry switched to atlantic salmon, but Chinook farms still exist.[103] It strongly appears from a review of the disease databases that the symptoms of the disease continue to occur in Chinook farms.

We know from the series of scientific papers written throughout the 1990s about the outbreak of Salmon Leukemia in Chinook salmon farms that once a site became infected, it remained infected even in subsequent generations of farm salmon.[104] The literature reports most of the

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[98] Ex. 1549-206 (2850), Tab 1Farm
[99] See Appendix B
[100] Aug. 30, 2011 Transcript, pp. 86-90
[101] Ex. 1491, Stephen, et al., "Descriptive Epidemiology of Marine Anemia in Seapen-Reared Salmon" (1996), p. 424
[102] Ibid, p. 423
[103] Ex. 1976, Morton "What is happening to the Fraser sockeye?", p. 16, quoting Stephen: "…the susceptibility of Atlantic salmon to experimental replication of marine anemia (Newbound and Kent, 1991) and the finding of marine anemia – like lesions in farmed Atlantics as well as in apparently wild stocks of Chinook suggest that we should not dismiss marine anemia…. Instead attempts should be made to synthesize new and existing information to develop potential intervention strategies not only to service the remaining Chinook producers in the province, but also in **preparation for the possibility of marine anemia becoming a problem for other farmed and wild species** " (Robert Craig Stephen, Thesis Spring 1995 Department of veterinary Microbiology U. Saskatchewan, Saskatoon, A Field Investigation of Marine Anemia in Farmer Salmon in British Columbia , National Library of Canada 0-612-23929-2).
[104] Ex. 1491 and 1492

farms were infected. We know that Dr. Stephen described the failures of fish farms to diagnose the disease during the period of his studies.[105]

We lose track of this situation for a few years, but databases provided to the Cohen Commission from the province of BC allow us to see this disease and the symptoms of this disease were still being diagnosed in 2003 onward.

A graph prepared by Dr. Morton from the prevalence of ISH in Ex.. 1549-217, using only the Chinook farms in Region 3 (the east coast of Vancouver Island – the migratory path of the sockeye), shows how common the disease/syndrome may be, and especially how it spiked in the last quarter of 2006 and first quarter 2007 – just before the 2007 smolts were outgoing through the farms).

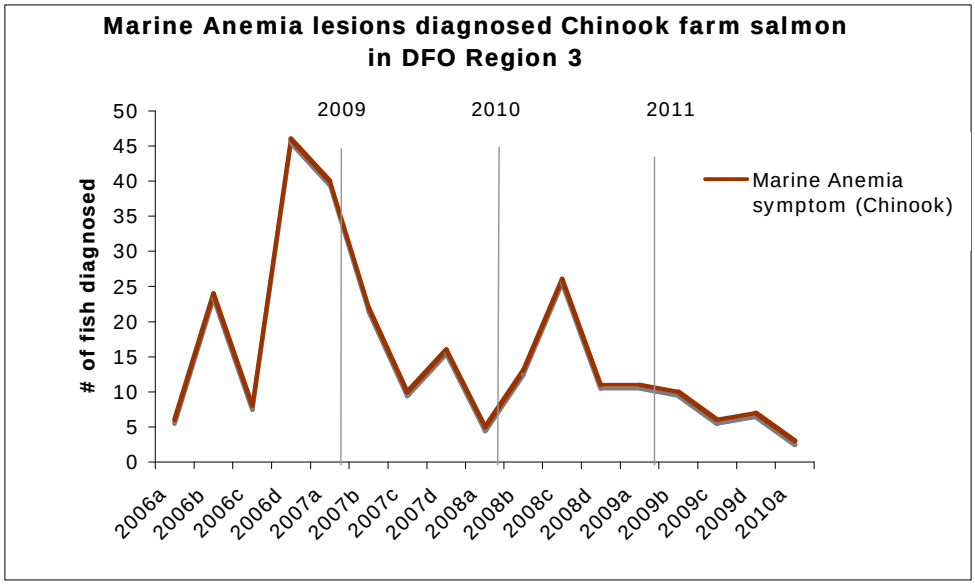

Figure 3: BCMAL audit data (BCP002864). Region "3" includes Sechelt Inlet, east Vancouver Island and central coast. There are no farm site designations so this includes data for a larger area than just the migratory route. The dates 2009, 2010, 2011 mark when these sockeye went to sea. The largest number of farm Chinook with Marine Anemia symptoms were found in the months when the Fraser sockeye that crashed were going to sea as smolts. Miller found the highest rate of Mortality Related Signature, >90%, in this generation.

(d) Chronic Marine Anemia in Fish Farms?

It is also possible that 'Chronic marine anemia' continues to exist in fish farms – ie. symptoms of the disease that are not sufficiently fatal to produce a diagnosis of a disease epidemic, but

[105] Ex. 1491, pg. 422 "marine anemia had not been diagnosed in 15 of the 23 farms we visited. We later found cases in all but 1 of the 23 farms"

nevertheless infect fish who give off pathogens to the open water. If, as Dr. Miller's work suggests, the cause of death for fish with the immune suppressing genomic signature/syndrome arises primarily during the stress of the transition to fresh water, which farmed fish do not experience as adults, then farmed fish would not die from it before they were harvested. However, the disease may infect migrating salmon, and become acute or fatal in migrating sockeye salmon at a much greater rate, on the return home. Chronic, non-lethal diseases are not measured in fish farms or the BC audit of 'fresh silvers'

The spike in mortality in Chinook farms in 2007 (see Figure 4 below) and the presence of a marine anemia outbreak in the Discovery Islands during the 2007 out-migration is a potential concern, and possible cause. [See "8. Chinook Farms, Disease and the Discovery Islands, below]

6. Early Entry And Pre-Spawn Mortality

Pre-spawn mortality and the associated behavior of 'early-entry' have significant correlations with the long-term decline of Fraser sockeye productivity and with the work of Dr. Miller.

Beginning in the mid- 1990s fisheries scientists began discovering escalating pre-spawn mortality (PSM) afflicting the Fraser sockeye. This phenomenon was also associated with abnormal early entry. Beginning in the mid-1990s some stocks, particularly the late-run stocks, began entering the Fraser River 4 -6 weeks earlier.[106] With respect to late-run stocks, early entry is a phenomenon that cannot be explained by adaption to changing conditions- early entry puts the salmon into the Fraser River when river temperatures are at their highest, instead of the cooler temperatures associated with late-entry. With higher temperatures, sockeye have to expend more energy to reach the spawning grounds and are exposed to heightened pathogen levels.[107]

Early entry and PSM were subject to a Technical Report prepared by Drs. Hinch and Martins (Technical Report 9). In that report, the authors discuss the known and the unanswered questions of early entry and PSM and make clear that these phenomena are having an inexplicable, devastating impact on Fraser sockeye. Statements from the report include:

> "…These studies have demonstrated that the earlier migrants each year suffer the highest en route and pre-spawn mortality. In years of the most extremely early entry, total freshwater mortality has exceeded 90%..." (p. 38)
>
> "From the start, this unprecedented phenomenon was a scientific mystery and presented a huge challenge to the management of the salmon resource…" (p. 38)

[106] March 8, 2011 Transcript (Hinch), p. 63:6-35

[107] For overview, see Hinch et al, Technical Report 9, "A Review of Potential Climate Change Effects onSurvival of Fraser River Sockeye Salmon and an Analysis of Interannual Trends in En Route Loss and Pre-spawn Mortality", pp. 35-40

> ""The causes of the shortening or complete elimination of the estuarine holding period are not well understood but a picture is emerging which illustrates complex links between physiology, environment and behavior. ..because the physiological changes that initiate reproductive maturation appear to occur prior to fish reaching the coast during their homeward migration (Miller et al. 2009; Patterson & Hills 2009) the estuarine behaviorual change may have its roots in the open ocean. Early entering fish are also physiologically stressed. Their gene array prfiles reveal immune suppression an stress responses (Miller et al, 2009, 2011)…"(p. 40)
>
> "…adopting a radically different behavior that is seemingly non-adaptive (eg. entering the Fraser River early and dying before spawning) would likely not occur without some physiological basis…"(p. 40)
>
> "**Although sensitive to the time period evaluated, the available data suggest that en route loss may be a critical contributing factor to decreasing trends in abundance for some Fraser River sockeye salmon stocks**, in particular, those that do not cope well with warming rivers…"(p. 50)

Before the Commission, Dr. Hinch gave the following testimony:

> Q So if we were to combine these two numbers, en
> 32 route loss and pre-spawn mortality, we're in
> 33 numbers that exceed 70 percent?
> 34 DR. HINCH: Yes.
> 35 Q And that would make this problem the single
> 36 greatest problem in terms of loss of salmon of any
> 37 that you're aware of, I would suggest?
> 38 DR. HINCH: Any that I'm aware of.
> …
> 39 Q This might be the single greatest causative factor
> 40 we have to look at?
> 41 DR. HINCH: Yes. For -- again, for a group of -- for
> 42 those particular group of stocks that are affected
> 43 by en route loss.[108]
> …
> 34 Q So that would be highly predictive of pre-spawn
> 35 mortality then?
> 36 DR. HINCH: It -- yes. It was certainly associated
> 37 with pre-spawn mortality at that level.
> 38 Q So this purported virus [Miller virus], if it in fact exists --
> 39 DR. HINCH: Mm-hmm.
> 40 Q -- goes a very substantial way towards explaining

[108] March 6, 2011 Transcript, pp. 64-65

> 41 the early -- or to explaining the whole of the en
> 42 route loss?
> 43 DR. HINCH: It could….[109]

Drs. Hinch and Martin briefly describe the major research initiatives, the first by the Pacific Salmon Commission in 2001, followed by some funded research by DFO.[110] The research of Dr. Kristi Miller began as part of these initiatives.[111] She was tasked with investigating whether biological predictors of PSM could be identified such that fisheries managers could account for the estimated PSM in setting seasonal fisheries allocations and escapements. While there are still questions that remained unanswered, Dr. Miller's research indicates a biological explanation for early entry and declines in survival and abundance of Fraser sockeye.[112]

7. Dr. Miller's work

(a) Causation

Dr. Miller testified that as many as 28 million salmon in 2008[113] could have been lost through the virus she believes she has discovered, and that the losses could have been "three or four times more" in the 2007 smolt/ 2009 return year. That is over 100 million fish. If she is correct, that in itself accounts for the 2009 collapse that is the primary question before this Commission:

> "Q. So in 2007, you found a much heavier prevalence of the MRS in the smolts than you did in the 2008 smolts?
> DR. MILLER: That's correct. It was a small sample size, because that's all that was available to us, but most of the fish that we sampled in the ocean at the end of June contained this signature in 2007, whereas it was less than 40 percent in 2008. We have actually, since, amplified parvovirus out of these same fish and we see the same phenomena.
> 37 Q The same phenomena was --
> 38 DR. MILLER: We see a much higher prevalence in 2007
> 39 than we do in 2008.
> 40 Q And if, in fact, the mortality is related as we just discussed, that would seem to indicate to me that the impacts in the 2007 smolts or the 2009 fish, would be much heavier than that of the 2008 smolts, 2010 fish?
> 45 DR. MILLER: Yes, potentially.
> 46 Q So we could be talking about many, many millions of fish here?

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[109] March 6, 2011 Transcript, p. 73
[110] In addition to the research identified in the Technical Report, pertinent Exhibits include: Ex. 1806, 1809, 1811, and 557.
[111] Aug. 24, 2011 Transcript, p. 42:11-44:39 (Miller)
[112] Dr. Hinch is a co-author of the Miller *Science* paper [Ex. 558]and agreed that the authors posit a virus associated with the biological indicators of early entry. If he had had more time to draft Technical Report 9, he would have included discussion of the virus hypothesis as the cause. March 8, 2011 Transcript, pp. 70:15-72:6.
[113] Ex. 1512, and Transcript, Aug. 24, 2011 Transcript, p. 94: 10-23

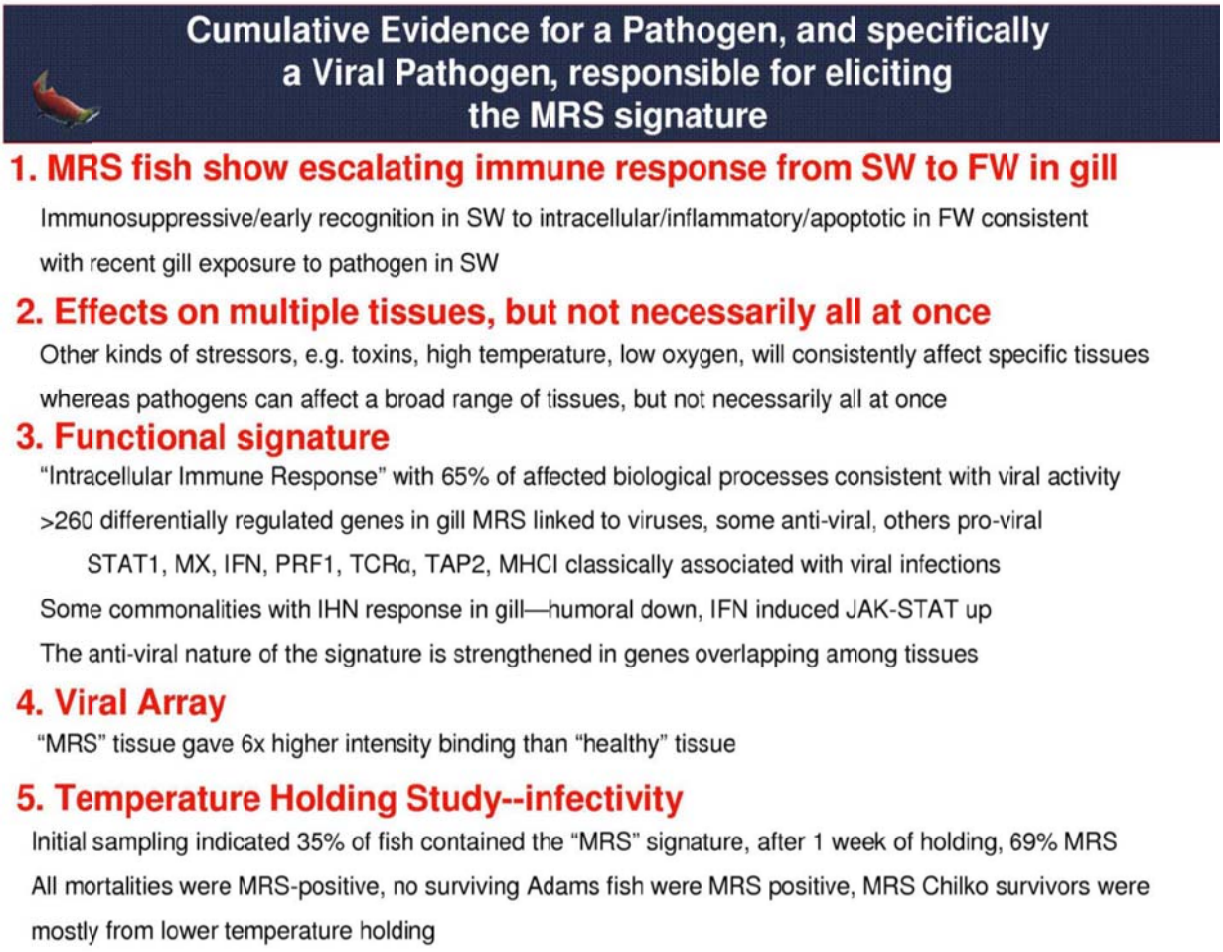
> DR. MILLER: I did a calculation somewhere 1 in one of these talks, but yes, we're talking in the order of, I can't remember what it was, three or four times more fish, in the least, between those different years. We're talking millions of fish, yes.
> 7 Q And so is it fair to suggest that this particular MRS, if it turned out to be the virus and if it turns out to have the mortality that you've speculated about, really could be a very, very significant explanation for the 2009 decline?
> 12 DR. MILLER: If we can demonstrate that this virus causes disease and has -- and mortality of fish in the early marine environment under certain circumstances, it doesn't necessarily have to be
> every year, I certainly expect that the role of the environment will be a strong one, but if we demonstrate that when fish are entering the ocean and they become stressed in the ocean and they
> carry a high load of this virus, that we see significantly enhanced mortality, they're certainly given the prevalence rates of fish that we see in certain years with this parvovirus there is certainly the potential that this virus could have a major impact on salmon declines.
> 26 Q And if, in fact, that's the case, using the terminology that we heard yesterday, this, in fact, may be the smoking gun for the 2009 declines?
> 30 DR. MILLER: It could be the smoking gun".[114]

Dr. Miller has determined that in 2007 90% of the smolts showed the MRS; in 2008 it was 40%.[115]

(b) Why it's a virus

In Dr. Miller's words:

[114] August 24, 2011 Transcript, pp. 94-95
[115] Ex. 1521, p. 7



Dr. Miller testified that she fully expects that she will identify the full sequence of the virus.[116]

(c) Parvovirus v. Salmon Leukemia

Dr. Miller was searching for a predictor for the early-entry and PSM syndrome that has been infecting the sockeye since the early 1990's. What she found is a potential new disease.

Dr. Miller has not confirmed the nature of the virus she has found -- or even confirmed conclusively that it is a virus (although the sequencing work done makes it highly likely[117]). For its value to the question before the Commission, it doesn't matter at this point whether the new virus is parvovirus or salmon leukemia, or some other disease agent. It is clear the MRS signature is associated with 'early entry'. It is clear that behavior goes back to 1995, which

[116] Aug. 24, 2011 Transcript, pp. 89:41-91:47
[117] Aug. 24, 2011 Transcript, pp. 87-92

began with the generation which, as smolts, were first exposed to the dramatic increase of fish farms along their migratory route.

Dr. Miller has not ruled out salmon leukemia. Rather she has no funding to pursue that.

It is notable that salmon leukemia was never sequenced by Dr. Kent or Stephen in the 1990's. Although it was proven to be a virus, and an infectious disease, the nature of this virus has not been confirmed. It remains plausible that the original virus studied by Dr. Kent, and named by him as Salmon Leukemia Virus is in fact the same or similar virus to that being pursued by Dr. Miller. It is also plausible that

Correlation may not be causation, but in the absence of any other change-agent of note, to which sockeye have been consistently exposed since the mid-1990's, it should give rise to significant questions – and a protective reaction based on the precautionary principle.

Identifying the virus as the cause of the MRS will not solve the problem. It will be necessary to determine the original cause of the virus, and hopefully, lead to actions to minimize or eliminate further exposures.

Dr. Miller is presently unwilling to conclude that the new virus is related to fish farms. She did so speculate initially, but faced significant backlash within the Department. Her uncertainty now stems primarily from the fact that the MRS is found in smolts coming out of the river. However, if the virus is transmitted vertically (from the parent to the eggs) or from returning salmon to outgoing smolts, that causation is still feasible[118]. How it is transmitted and where it occurs now does not necessarily identify its original causation. Given that the early entry behavior stems back to the early 1990's, and early entry is correlated with the MRS, it seems reasonably likely that this disease, when it is identified, will be found to have been present and infecting some fish since the early 1990's. Its origin dates back to that point, and causation likely traceable to some new factor at that time. That factor may well be fish farms.

Obviously more research is necessary.

(d) The connection to early entry

See Appendix C and "6. Early Entry and Pre-Spawn Mortality".[119]

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[118] There is an interesting correlation from the fact that the Harrison sockeye is the only stock whose smolts do not mix with returning adults, and the Harrison is doing well. Coho and Chinooks, whose smolts and returning adults also mix, are doing poorly over the long-run; chum, which do not so mix, are doing well. Sept. 7, 2011 Transcript (Morton), p. 71; Tucker et al, "Seasonal Stock-Specific Migrations of Juvenile Sockeye Salmon along the West Coast of North America: Implications for Growth" (2009)- This Tucker article should be Ex. 1818 (see Sept. 7, 2011 Transcript, p. 97)

[119] Ex. 1524, p. 4

(e) The non-disclosure of this information- What DFO's reaction to Miller's work tells us about DFO?

The evidence shows that senior officials in DFO were aware of the information generated by Dr. Miller's research, and the hypothesis that the Fraser sockeye were suffering from a virus, at least by mid-2008.[120] The public was not advised; and, there is some evidence that it has been ill-advised. [121]

In drafting the memos to the Minister regarding the potential causes of the poor returns in 2009, reference to "brain lesions" in the title was removed and the language suggesting the seriousness of the potential disease issue was watered down for the final version.[122]

Parliament was not informed. The speaking notes prepared for speeches to be given to the House regarding the cause(s) of the 2009 poor return made no mention of disease (though there is considerable discussion of why sea lice is not the cause).[123]

The information only became public through a Globe and Mail story on Nov. 3rd, 2010. Dr. Richards offered various reasons why DFO did not publicize the virus research, including because the DFO structure required the Minister to know first; that the research was ongoing, and, the Commission of Inquiry was being conducted.[124] None of these explains why DFO has known for years a virus likely has has a major impact on Fraser stocks, but kept that information to itself, despite intense public interest that a Commission of Inquiry was called. This Commission was called in November 2009. If this information had been made public, it is very possible this Commission would have been unnecessary, (and $29 million could have been saved).

Efforts were made internally to persuade Dr. Miller not to come forward with the information, and to 'tone down' or alter her views[125], including to take out references to possible links to aquaculture, including correlating the symptoms and timing with aquaculture . Drafts of the Briefing Note to the Minister in 2009 show an express deletion of a paragraph outlining such links. [126] Dr. Miller stated that there were "some concerns" in the department about linking the genomic signature/virus with fish farms.[127] This seems an understatement.

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[120] March 17, 2011 Transcript (Richards), pp. 49- 60; see also Ex. 627, 628, 613G, 557, 635, 637
[121] For example, letter from Minister Shea to Morton, March 3, 2011 mis-states how long the department has been aware of the virus hypothesis [Ex. 636].
[122] Ex. 615, 616A, 616B and 617; March 17, 2011 Transcript (Richards)
[123] Ex. 661, Ex. 622A, March 17, 2011 Transcript (Richards), pp. 66-70
[124] March 17, 2011 Transcript (Richards), pp.27-28, 66-73
[125] See, for example, Ex. 1458, MacWilliams, Update on Science Review, 2009 Fraser Sockeye
[126] See Ex. 1523, 1524 and 1528; Aug. 24, 2011 Transcript (Miller), pp. 93-100
[127] Aug. 24, 2011 Transcript, p. 99:12- 100:17

(f) The need for further funding and further research

Dr. Miller's work has not been well-funded.

It has also clearly not been popular within the DFO bureaucracy. Whether that unpopularity stems from the potential connections to fish farms, or her initial speculations around such possibility is a matter of opinion.

The failure to have funded the initial requests for tests within fish farms is unexplained, and we contend, inexplicable.[128]

RECOMMENDATION: ***DFO should prioritize comprehensive research into the potential parvovirus and/or salmon leukemia virus, its origins and a strategy to mitigate or eliminate it and conduct genomic profiling on all Atlantic salmon farms.***

8. The Marine Environment

(a) Increased impact of disease in a warm environment

> "Should the young sockeye be infected at sublethal 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 incur hundreds or even thousands of kilometres 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)."[129]

(b) The Georgia Strait and Queen Charlotte Sound

The evidence suggesting the conditions in the Georgia Strait were the cause of the 2009 decline are not compelling.

This evidence is based entirely on Dr. Beamish's studies. It is respectfully submitted that those studies do not support the conclusions suggested. The studies are based on far too little data, and are not scientifically credible.

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[128] Ex. 639, Miller, "Proposed 2010 DFO Funded Research relating to sockeye declines"
[129] Dr. Dill, Project 5kD, Ex. 1540, p. 31

It is also not consistent with other known facts. The tagging work of Dr. Welch suggests that mortality occurred at least 20 to 30 days after passing the fish farms in Johnson Strait[130]. The Harrison stocks, which spent far longer in Georgia Strait, did well.

Dr. Beamish's reliance upon sampling from the July troll surveys to estimate sockeye abundance is indefensible. The troll survey was never designed as a measure of relative abundance for rapidly migrating species like sockeye.[131] The troll samples were taken at the wrong time of year – after the sockeye runs had left.[132] The few fish taken were too small a sample size.[133] Most importantly, the data presented, even on this inadequate sample, was contradictory.[134] It would appear that Dr. Beamish set out to prove a hypothesis and selected data that supported it, ignoring the rest. The same comment may apply to the portion of the study, attributed to Dr. Thomson (who did not testify) in respect of the theory put forward concerning variations in wind and run-off, which seems to have been adapted from what was available.[135]

There were other examples where Dr. Beamish was prepared to 'explain away' evidence that didn't support the hypothesis. Thus, he could dismiss the inconvenient fact that sockeye stomach contents were consistent with other years on the basis that "those sample sizes were pretty small".[136] But from those same small samples he was prepared to draw conclusions that the fish

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[130] Dr. Welch October 25, 2010 pg 66-68 Q: "So that the Fraser River problem in 2009 appears to be isolated to those stocks that were running up Johnstone Strait.
DR. WELCH: That's correct…"Our results show that most of the mortality happened after they passed the north end of Vancouver Island.
Q. Now, you have suggested in a submission you made to the Commission that much of the mortality may have occurred in the 20- to 30-day range after leaving Johnstone Strait; that's right?
DR. WELCH: That was my submission, yes.

[131] Ex. 1329, Beamish 2001, p. 5, "We do not believe that the June/July survey is a measure of relative abundance among years as most juvenile sockeye leave the Strait of Georgia before July." Ex. 1336, Beamish 2000, p. 4, "A comparison of pink and sockeye estimates among years was not made because these species tend to be highly migratory…".

[132] Therefore, it could be expected that the remaining fish sampled were unhealthy, late bloomers, or somehow other unrepresentative.

[133] With standard deviation with this number of samples would be far greater than the difference in size found. This paper would be unlikely to pass Peer review on that ground alone.

[134] For instance, Table 1, Ex. 1303 (Thomson et al, "Anomalous Ocean Conditions May Explain the Recent Extreme Variability in FRSS Returns"): there were more fish caught in Hecate Strait in 2007 (400) than 2008 (278) and 2009 (188). The average catch per set was higher in 2007 (14 versus 13.5 in 2008). Table 2, Ex. 1303, p. 54, the weight of fish from Georgia Strait was higher in 2007 (12.7) than 2008 (11.9). Table 4, Ex. 1309, p. 33, once adjusted for Harrison sockeye, the 'adjusted' length of sockeye smolts was also longer in 2007 (109.9) versus 2008 (106) [and note that both were higher than 2006]. Figure 7, Ex. 1309, p. 44, shows the length of sockeye was not unusual in 2007. Figure 9, Ex. 1309, p. 46, the percentage of empty stomachs for sockeye (last box) not substantially different from 2006, and lower than 2004.

[135] Transcript, July 7, 2011, p. 89 – 90, Dr. Beamish 'challenged' Dr. Thomson to explain poor returns by wind and salinity conditions in the Strait of Georgia. In fact, the email at Ex. 1334 demonstrates that Dr. Beamish had formed a theory based on the mistaken belief very strong winds come about when the facts changed, he changed his theory accordingly to produce the same result. Clearly Dr. Beamish started with the conclusion – poor food production – and then adapted the meteorological information to explain it (regardless of what the winds actually were.

[136] July 7, 2011, p. 97: 6-13

were "small" and "in poor condition".[137] The most telling example was Dr. Beamish's willingness in his paper to rely on apparently smaller sizes in the troll survey for 2007 sockeye as indicating "poor condition"[138], but then when confronted by evidence that the 2008 fish were actually smaller, at first suggesting they were essentially the same size[139]. But when Dr. Beamish was convinced the data showed the fish to be smaller in 2008, he promptly drew the conclusion that the reason the fish in 2008 were *smaller* was "a result of the large abundances of lots more juvenile fish in the Strait of Georgia".[140] Dr. Beamish's willingness to switch gears on a dime from smaller size in 2007 means 'poor condition' to smaller size in 2008 means 'large abundance' was breathtaking. It is certainly not the sign of credible science. This evidence must be rejected.[141] The real heart of Dr. Beamish's theory in respect of Georiga Strait is simply that Coho and Chinook did poorly in 2007. While that may be true, it does not follow that this is an explanation for the poor returns by sockeye in 2009. Coho have been doing poorly for years, and the 2007 experience was not an order of magnitude difference (and certainly not a twenty-fold difference as might explain the sockeye collapse in 2009).[142]

With respect to Chinook populations, Dr. Beamish predicted that the Chinook returns in 2010 and 2011 would show "exceptionally low marine survival".[143]

In respect to meterological conditions being somehow different in 2007, that portion of the theory is also doubtful. Dr. McKinnell and the team behind Technical Report 4 looked for and was unable to confirm such exceptional conditions.[144] The evidence is stronger in respect of unusual marine conditions in Queen Charlotte Strait. We accept the evidence of Dr. McKinnell.

To the extent that ocean conditions north of the fish farms cause added stress, any disease being experienced by the fish would likely have greater mortality. It is unlikely that ocean conditions account for the full magnitude of the 2009 declines. It is likely the temperature or other adverse ocean conditions are likely to exacerbate the impacts of any disease, and cause mortality among diseased fish in preference to healthy ones.

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[137] See Ex. 1309, p. 17:340-342, and p. 2 (abstract) at l. 23. Also see Transcript July 7, 2011, p. 99: 26-32, where Dr. Beamish denies drawing "major conclusions from that".
[138] Ex. 1309, p. 17
[139] July 7, 2011 Transcript, pp. 101:4, 37-47, and see p. 102: 20-29
[140] July 7, 2011, p. 102: 35-39
[141] Dr. Beamish has shown a significant bias in favour of aquaculture. In 2000, Dr. Beamish, along with Dr. Noakes and Dr. Kent, wrote the paper, Ex. 779, Noakes, Beamish and Kent, "On the Decline of Pacific salmon and speculative links to salmon farming in British Columbia." In 2011, after his testimony, Dr. Beamish wrote a paper for the BC Salmon Farms Association, "assessing the impact of salmon farming on Pacific salmon at the population level in British Columbia", Ex. 1984. This paper, written after Dr. Beamish was free of cross-examination, and not at the request of the Commission or Canada, shows how clearly Dr. Beamich has affiliated with the salmon farming industry. That paper can be given no weight.
[142] Ex. 1309, Figure 12, p. 49.
[143] Ex. 1309, p. 14, based on his theory. That has not come to pass. The Chinook returns for 2010 and 2011 have not been exceptionally low.
[144] Dr. McKinnell and Dr. Welch were also reluctant to accept Dr. Beamish's conclusions on salmon survival, Transcript July 8, 2011, p. 29-30, Dr. Welch, July 7, 2011, p. 73-77. Dr. McKinnell discussed Georgia Strait, July 7, 2011 at p. 37-38. He was not prepared to use the word "extreme" for Georgia Strait. Report 4 shows little difference, Ex. 1291, p. 89, p. 103, p. 137.

(c) Need for increased vigilance with Climate Change

There is little this Commission can do to stop global climate change. The evidence is clear that as temperatures rise, salmon will become increasingly stressed.

This makes it all the more urgent that those activities we can control that add risk.

9. Chinook Farms, Disease and the Discovery Islands

As noted above, on review of the Disease Databases, certain diseases, and symptoms of diseases, are disproportionately prevalent amount Chinook farms.

One example is Plasmacytoid Leukemia (marine anemia). The epidemics described by Dr. Kent and Dr. Stephen in 1988-1991 appears to have been exclusively found in the farms growing Chinook salmon.[145] Mortality at Chinook farms was as high as 80%.[146] Dr. Stephen found the disease at "all but one" of all the 23 Chinook farms visited in 1991-1992.[147] The experiments conducted by Dr. Kent indicated that Plasmacytoid Leukemia was transmittable, between species to both Atlantic salmon and sockeye salmon. He found that "Sockeye Salmon are very susceptible to PL"[148], but that Atlantic salmon were far more resistant than Chinook or Sockeye – "only two of 22 exposed of Atlantic salmon developed PL".[149]

As noted previously the database shows far more "ISH" for Chinooks than for atlantics (291 positive of 447 chinooks examined).[150]

It can also be seen in an analysis of other databases that diseases in Chinook (Pacific) salmon are far more common. In the MAL Fish stocking database BCP002850 (Exhibit 1549-206) the 'population level' epidemics are commonly associated with Pacific (Chinook) salmon farms, particularly in and around the period of 2007.

This leads reasonably to a suggestion that farms stocked with Chinooks may potentially have different diseases than Atlantic farms, and may have effects from disease transmission on wild sockeye that are markedly different.

The lumping together of all farms by Dr. Korman in his data (relied upon by Dr. Noakes) suffers from this very significant weakness. Combining in the larger number of Atlantic farms may very well 'mask' a substantial trend in disease offence. Because DFO has not studied these issues, at any time, in any depth, we cannot know for certain.

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[145] Ex. 1488-1493.
[146] Ex. 1489, (Newbound and Kent, 1991), p. 1(159).
[147] Ex. 1491, (Stephen, 1996), p. 422. Note that Dr. Stephen indicates in that paper, at p. 433, that mortality attributed to marine anemia during their surveys ranged from "2.5% to 11%".
[148] Ex. 1489 at p. 162.
[149] Ex. 1489 at p. 165.
[150] Exh 1549-217, and see Appendix B

However, there are a couple of facts, readily demonstrable with the aid of the databases, that raise a significant distinction between 2007 and 2008:

(a) Chinook farms were present in 2007 in the Wild Salmon Narrows (Discovery Islands), but were not present in 2008; and

(b) marine anemia was present in at least one of those farms – Conville Bay.

The stocking database produced by Dr. Korman, with some effort, identifies which farms were stocked with which fish, at which times. At Appendix B [we highlight the evidence showing that the Conville Bay site was stocked in late 2006 through to June 2007 with Chinook Salmon]. That site was fully harvested in June 2007, and was fallow thereafter.[151]

An examination of Histopathology FHAS 2006-2010 database[152] then shows that the same farm was audited in December 2006, and "marine anemia" is present in 23 of 24 fish that were audited. Dr. Marty refused to acknowledge this as a marine anemia epidemic, as Dr. Marty does not believe in marine anemia as a diagnosis.

Dr. Miller's evidence, produced graphically on a slide[153], showed the incidence of the 'mortality related syndrome' was significantly different in 2008 (40%) than 2007 (90%). It was this difference alone that potentially could explain a substantial loss of fish in 2007, and the greater numbers of returns in 2008.

Does this correlation between the absence of fish of Chinook farms in 2008, (and the consequence freedom from marine anemia) prove any causation in relation to the MRS in the 2009 declines? We are told – correlation is not causation. In that case the answer is there is no sufficient proof to draw that scientific conclusion. However, a correlation of this magnitude should trigger questions.

Given the greater amount of disease present in Chinook farms over Atlantic farms, it also raises questions as to why Chinook farms should be allowed, at all, in the combined passages of the wild Salmon narrows. Apparently, according to Claire Backman, those of some Chinook farms have returned to the area. It is essential that these disease factories are removed.

The farm salmon stocking database created by Josh Korman revealed that all Chinook salmon farms were removed from the sockeye migration route in June 2007 and remained absent through 2010. This means the 1st and 2nd generations of Fraser sockeye to go to sea without exposure to Chinook farm effluent returned in healthy abundance in 2010 and 2011. On the stand Clare Backman of Marine Harvest said there were Chinook farms still on the Fraser sockeye route in this time period, but those data was not provided in Josh Korman's database.

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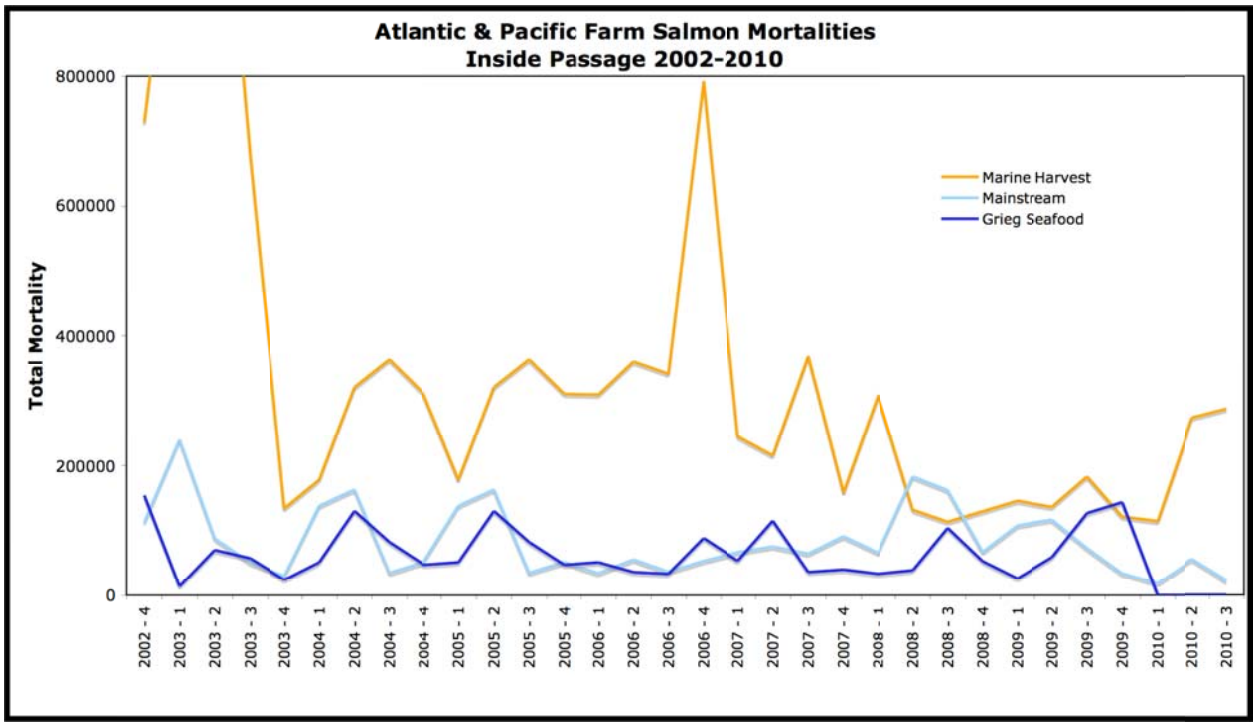
[151] The evidence of Claire Backman notwithstanding – his evidence was complete here say, and cannot stand against the actual databases as database records.
[152] Ex. 1678 (BCP0002864)
[153] Ex. 1521, p, 7

There is also the evidence of a remarkable spike in mortality in Marine Harvest farms in the Inside Passage, just prior to the removal of the Chinook farms, and just prior to the smolt migrations of the sockeye runs which collapsed in 2009 (see Figure 4 below).



Ex. 1985 (corrected)- Prepared from Dr. Korman spreadsheets. While BCMAL has been posting diseases diagnosed in farm salmon in aggregated form on their website, the above graphs suggest there are significant trends in the symptoms diagnosed of diseases that pose large and unknown threat to Fraser sockeye salmon. There is no record of adequate testing to confirm if these symptoms resulted in disease. That high mortality in the farm salmon belonging to one company, and peaks in two lethal salmon viruses and the outmigration of the sockeye run that crashed all coincide in time and place highlights the need for further investigation.

This is a remarkable pattern of collapse and rebound around presence/absence of a single variable – Chinook salmon farms. There is no other variable revealed by this inquiry that fits the available science and these critical changes in Fraser sockeye returns. Unfortunately, this remains only a pattern because Fisheries and Oceans Canada has not taken identified the causative agent for Salmon Leukemia or the mortality related signature. There has been no test of genomic profile of farm Chinook salmon exhibiting interstitial cell hyperplasia of the kidney to see if it matches the profile in the sockeye or not. In addition, there is a test being used by the salmon farming industry to look for Salmon Leukemia in their imported eggs called a Gram Stain test, but there is no evidence this test has ever been applied to the Fraser sockeye.

PART 2 - THE REGULATORY ENVIRONMENT

DFO's regulation of aquaculture has not been grounded in adequate scientific assessment of the risk of aquaculture to wild salmon stocks nor has it applied a precautionary approach. In this respect, it has not fulfilled its mandate to conserve the fisheries of Canada.

Since 1988, the Department of Fisheries and Oceans has largely failed to exercise its jurisdiction to regulate aquaculture as part of the fisheries of Canada and agreed to and acquiesced in the province regulating and managing the industry via an unconstitutional legislative regime.

Although even pursuant to the 1988 MOU, Canada retained responsibility under the Fisheries Act to protect wild fish, the evidence shows that Canada did not do so, except in an advisory and promotional role. Almost all regulation and management, including siting and monitoring, was abdicated to the Province. DFO did maintain a public relations role for aquaculture, maintaining that aquaculture was not harmful to wild salmon. The science over that time appears to have been dedicated to establishing to media that critics are wrong ('rebuttal science').

In particular, there is no evidence before the Commission, or in the larger database, that shows Canada ever played a role in the regulation of disease impacts, even though there is evidence, throughout that period of disease outbreaks on many occasions. There is no evidence that there was ever DFO staff with operational responsibility to regulate the disease impact of fish farms.

The failure of the Province to appropriately protect wild salmon from the effects of aquaculture and disease can be understood in the context of the constitutional division of responsibilities, and the assumption that this would come within DFO's purview, "relieving" the Province of the obligation to protect impacts outside the farms.

After Mr. Justice Hinkson's decision – two years later – it is clear that DFO has implemented a very sparse regulation. What has been seen so far of that regime does not suggest a new rigour or approach- rather maintenance of status quo, and, in some important respects, purported reduction in regulation. Shortfalls include: grandfathering past licence/siting decisions (for over 100 farms on the migration route) that were made without taking into account the risk of disease from aquaculture, gap in scientific data and research in B.C., failure to account for international experience of impact (particularly disease) impact of aquaculture on wild stocks, sparse and discretionary regulatory regime, inadequate reporting requirements, failure to ensure that fish farms will be subjected to rigorous assessment of their potential impacts to wild salmon and their habitat, and an inappropriate emphasis on promotion of the aquaculture industry at the expense of regulation and the health of wild salmon.

1. Siting

Past siting decisions for existing fish farms were made in the complete absence of consideration of disease impacts on wild salmon and in particular without consideration of the potential risks within the Fraser River Sockeye migration routes.

The single, most important recommendation that this Commission could make would be to recommend removal of fish farms from the Fraser River Sockeye migration route.

Based on all the evidence from the international experience, scientific and expert opinion before the Commission, there can be little question that salmon farms act as mechanisms for the amplification and transfer of disease and increase the risk of disease to wild salmon. That there is little data establishing empirical *proof* that disease transfer is occurring in British Columbia is only because no science has been done. [See section "The Absence of Research"] This risk must be accounted for in siting fish farms. It has not been.

The decisions to site fish farms in close concentrations in the confined channels of the Discovery Islands and Johnston Strait is egregious, and can only be explained by the complete failure of DFO to have considered migrating sockeye salmon as part of and at the time of approving site locations. Whether this oversight was the result of willful blindness, negligence or simple ignorance with respect to potential risks is not necessary to determine. The simple fact is that wild salmon impacts were not considered at the time of the original siting decisions (including in s. 35 of the *Fisheries Act* or CEAA assessments), and most importantly, were not considered at all by DFO in the decision to 'grandfather' these licences in December 2010.[154]

Significant evidentiary findings that can be made in this respect:

- DFO did not consider disease risk or impacts upon wild Sockeye at the time of the original site decisions (nor did the Province).
- Had DFO considered it, there was no scientific basis upon which they could have evaluated the risk (as they had not done the research).

Absent change recommended by this Commission DFO does not address disease risks through siting, but rather relies almost entirely on licence conditions, and specifically the Fish Health Management Plans (FHMP), to minimize risk [see section "The Inadequacy of Fish Health Management Plan to Protect Wild Salmon"]. This policy is <u>not</u> sufficient.[155]

(a) Disease not studied; not a criteria in siting

The hearings definitely showed that disease has not been a focus of DFO's research. The witnesses were in broad agreement that DFO has not done sufficient research into disease in wild salmon; nor has it studied disease impacts from fish farms on wild salmon. The Project 1 and Project 5 experts, and several DFO panelists, agreed that the data was not available to look at the interrelationship between fish farms and wild salmon for disease.

David Marmorek confirmed that the data on disease impacts did not exist (though studies could readily be done)[156]. Dr. Richards, Regional Director of Science, confirmed that disease has not

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[154] See Transcript September 22, 2011, pp. 80-83.
[155] Witnesses referred to "adaptive management" (Hoyseth, Sept. 1, 2011 Transcript, p. 58:1-6) and "area-based management plans" (Farlinger, Sept. 22, 2011 Transcript, p. 80) as approached DFO will strive to use in the future.
[156] Sept. 19, 2011 Transcript p. 77:9-45

been a focus; and that more work needs to be done.[157] Deputy Minister Claire Dansereau testified that the advice from DFO Science has always been that there is no "threat that we are completely aware of" for disease from fish farms to wild salmon. [158]

Thus siting decisions have been made without research being done, and, with the operating assumption that there is no or low risk of disease impacts from fish farms. Indeed, each of the processes through which DFO purports to evaluate the impacts of a proposed fish farm fail to identify disease as a criteria or risk, either on an individual farm basis, or as a cumulative effect.

It is true that DFO has begun to do science studies, in 2011, which might someday produce disease risk information.[159] What is obvious is that such science did not exist at the time of siting decisions (for over 100 farms on the Fraser sockeye migration route) nor could be considered at the time of the licence/site grandfathering decisions in December 2010.

RECOMMENDATION: ***Research into disease in wild salmon and aquaculture; and disease interactions between the two, be given highest priority within DFO; and, the federal government provide sufficient funding to ensure this research is carried out in a timely and scientifically-supportable way.***

(b) Siting Criteria

The Province had no mandate or responsibility for the protection of the wild sockeye. There is no evidence that the Province ever considered such impacts. The focus of the Province in respect of disease was to reduce or minimize the amount of disease within fish farms (which has an economic impact) and where disease occurs to minimize its transfer to other fish farms within provincial jurisdiction. For those purposes the Fish Health Management Plan might be appropriate. The Province relied on referrals to DFO of siting decisions to meet any responsibility to protect wild sockeye. Unfortunately, it does not appear that DFO ever fulfilled that mandate.

The siting criteria that have been used by DFO in the past, and that have been adopted for their regulation going forward, were not developed with a strong scientific basis.[160] They were developed after the Salmon Aquaculture Review in 1997, at a time when DFO had not done any research on disease or pathogen impacts from fish farms on wild salmon.

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[157] Sept. 26, 2011 Transcript, pp. 63-72

[158] September 22, 2011 Transcript, p. 78, "and our science has always been – the advice that we have always received from our scientists has always been that there is no threat at this point, or there is no threat that we are completely aware of..."

[159] Dr. Richards, September 22, 2011, p. 79 "But we do have some tools that I think we would be able to use to start to look at some of those questions". Also, September 28, 2011, p. 102 "…we have undertaken some studies, starting in 2009, to look more generally at the overall status of health of juvenile salmon…"

[160] The siting criteria are set out at page 45 of PPR 20- Aquaculture Regulation in British Columbia and Ex. 1589. On Aug. 30, 3011, A. Thomson confirmed that DFO is continuing to use those same siting criteria in licencing decisions going forward. (Transcript, p. 16:31- 17:4); as did G. Last (Aug. 30, 2011 Transcript, p. 69-70)

Location on a salmon migration route is not a criterion in the list. Mr. Swerdager and Gavin Last both testified that they were not aware of any aquaculture application that was rejected by DFO because it was located on a salmon migration route[161]; and, clearly, the number of salmon farms located on the Fraser migration route show that this was not a factor that was given any serious consideration when siting farms. Moreover, fish farms are densely situated in the confined areas of the salmon migration route, particularly the Discovery Islands, where they can pose higher risk of pathogen transfer to wild salmon.[162]

The criteria require that a farm should be at least 1 km from a local stream, however, this was not based on science and is not relevant to potential disease or cumulative impacts to wild sockeye. It is illogical that salmon farms should be sited more than 1km from a local stream – containing a single salmon run – but be placed directly in the path of major sockeye migrations of millions of juvenile smolts (just 15 days out of the Fraser River), when they are most vulnerable to disease, and in the path of the millions of returning adult sockeye.

There is no explanation in the evidence that would justify the failure to address wild sockeye migrations in the siting criteria. The only conclusion is that no-one turned their minds to it, until this Commission.

The siting criteria remain inadequate to protect wild salmon. Without a strong recommendation from this Commission, those sites along the migration path will remain, never having been assessed for impacts to migrating Fraser sockeye.

RECOMMENDATION: ***DFO should identify proximity to a Fraser sockeye migration route as a priority siting criterion; and, siting criteria should prohibit farms in close proximity to migrating salmon until DFO has conducted sound, supportable, peer-reviewed research into disease interactions between wild salmon and aquaculture operations.***

(c) CEAA

Aside from the siting criteria for locating fish farms, there were two other avenues by which impacts and risk to wild salmon from aquaculture could be, but weren't, fully evaluated. One is DFO's responsibility to administer the main protection of fish habitat and fish provisions of the Fisheries Act (sections 35 and 36). The second is through an environmental assessment pursuant to the *Canadian Environmental Assessment Act*. Neither of these processes has addressed the risk from disease or cumulative impacts from aquaculture on wild salmon. Instead they have been focused on the arguably relatively low-risk aspect of fish farming- the deposition of waste materials from farms.[163]

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[161] Aug. 30, 2011 Transcript, p. 71:8-18

[162] Ex. 1628, "Fish Farming on and around Vancouver Island and Coastal BC", Ex. 1563 "Map of Salmon Farms and Migration Routes" (Living Oceans); Aug. 30, 2011 Transcript, p. 67:27-34, 68:14-30 (Fleming), pp. 69:24-70 (Last)

[163] Rebecca Reid confirmed this to be the approach during her testimony on April 5, 2011 (Transcript, pp. 27-29)

A CEAA environmental assessment can be triggered for a fish farm when an authorization for a fish farm is required pursuant to section 35(2) of the *Fisheries Act* (to cause a harmful alteration, disruption or destruction of fish habitat) or a permit (to interfere with navigation) under the *Navigable Waters Protection Act* is required. In the former, DFO would be the responsible authority for the assessment; for the latter, Transport Canada is the responsible authority. As is discussed further below, because of DFO's policy toward assessing section 35 impacts, a section 35 authorization was rarely deemed required by the department and those fish farms that did undergo an environmental assessment did so because a NWPA permit was required.

On occasion through the Hearings, various witnesses relied on the fact that fin fish farms had undergone CEAA assessments as supporting the assumption that disease risks had been evaluated (on an individual or cumulative level). Aside from vague assertions that DFO must have looked at these issues, the evidence does not support that.

Rather, two CEAA assessments in evidence, for Dunsterville and Conville Bay (Exhibits 1629 and 1630) demonstrate how the assessment process took place, and, how the risk to wild salmon (particularly of disease) from fish farms was dealt with. The documents show that there has been essentially no examination of the fact that wild sockeye salmon will be migrating directly past these farms. In fact, in the description of the "Biophysical Environment" at page 4 there is no mention of wild sockeye or the migration route at all. This and other aspects of the assessment suggest that it is highly unlikely that wild salmon stocks were ever seriously considered.

The mitigation measures listed (at para. 32, page 5 of Exhibit 1630) refer to the regulations, particularly:

> "Fish Health Management Plan, which forms part of the provincial aquaculture licence…Atlantic Salmon Importation Policy
>
> *Fish Health Protection Regulations...*"

Disease is addressed only once in the Table 1 of activities associated with the project (at p. 9). In that Table, under the column "Potential Project – Environment Interaction" the bullet listed is "Potential Introduction and/or Transmission of Disease and/or Parasites from Farmed Fish Could Impact Wild Fish Populations", and under the heading "Significance" the finding is "Low". Under the column heading "Mitigation Measures" the justification analysis is clear – the first paragraph refers to the Atlantic Salmon Importation Policy (i.e. species imported from outside Canada are presumed to be free of disease by compliance with that Policy). The second paragraph also refers to fish transfer policies. The third paragraph refers to the Fish Health Management Plan as "mitigation to address issues of fish health for farm fish and takes into account interactions with wild fish". The reasoning seems to be that a Fish Health Management Plan will 'minimize' disease transmission to wild fish (that is reasoning or an assumption that needs to be questioned). The fourth paragraph relates to biosecurity measures – which are

designed to avoid transmission to other *fish farms*, and can have no measurable impact in mitigation of harm to wild fish swimming past the net cages.[164]

Similarly, in Table 2, dealing with cumulative impacts of multiple farms, disease transfer risk is again rated as "Low" (page 16 of Exhibit 1630). Under 'Comments' it states:

> "pathogens that originate in salmon farms at renewal sites in Hoskyn and Okisollo Channels are not likely to have significant cumulative adverse effects on migratory salmon. Uncertainty exists with respect to the migratory pattern of salmon along the Channel/Inlets in the area, and on effects associated with groups of salmon migrating past multiple farm sites a short distance apart (potential IHN reservoir locations). Measures outlined in the company's Fish Health Management Plans reduce likelihood of transmission and effects on wild fish populations."

The statements that FHMP's 'reduce likelihood of transmission and effects on wild fish populations' is completely unsupported, and on the evidence available to the Commission, completely unsupportable. On the evidence, Fish Health Management Plans may reduce the frequency of disease within fish farms, but once disease strike, there is nothing in the FHMP's, or within the existing power of fish farm operators to prevent the transmission of pathogens through the water columns, though the nets to wild fish. There is certainly nothing that can be said to reduce the "effects on wild fish populations".

The CEAA assessments in as Exhibits show no indications that the magnitude of the disease risk was ever studies empirically or referred to any scientist in the Aquatic Health Branch. Moreover, we know from the testimony from Ms. Farlinger and Ms. Richards on September 22 and 26, 2011, as well as the testimony of other witnesses, that no studies had been done at DFO into cumulative adverse effects on these farms, and in fact there was no one on the science staff who was responsible for vetting these applications or giving these opinions. Thus, the statement that "uncertainty exists" is absolutely correct. The statement that FHMP's 'reduce likelihood of transmission and effects' is completely unsupportable, tending towards false.

While we do not have all the CEAA assessments in evidence, Ex. 1629 and 1630 demonstrate the view of the department regarding risk to wild salmon. That view has been confirmed by other evidence, including the testimony of witnesses as well as documentary evidence regarding the state of knowledge of the department. In this regard, in terms of input from DFO, both these assessments (which were conducted with Transport Canada as the responsible authority) relied on the advice of DFO regarding potential impacts set out by form letter signed by Andrew Thomson and approving 97 farms.[165] Each of the 97 letters advised Transport Canada that DFO deemed the farm to pose no risk of significant impact to fish or fish habitat and set out mitigation measures (essentially requiring regulatory compliance). These letters again show absolutely no evidence that DFO considered the risk from fish farms to wild salmon on an individual or

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[164] Sept. 1, 2011 Transcript (A. Thomson), pp. 51:45- 52:45
[165] See page 3 of Ex. 1629 and 1630 under "Responses Received…"

cumulative scale or that Transport Canada could do so as part of the environmental assessment.[166]

(d) Section 35- HADD

Section 35 of the *Fisheries Act* prohibits unauthorized harmful alteration, disruption or destruction of fish habitat. The section potentially provides a tool by which the effects of fish farms on wild salmon, in particular through impacts to their environment, can be assessed and standards enforced by DFO.

However, the Department's approach to evaluating impacts to fish habitat is narrow, has been criticized, and does not accord with the definition of fish habitat in the *Fisheries Act* or the Habitat Management Policy. The department's approach does not consider cumulative impacts, impacts of disease, sea lice or pathogens, or far afield impacts. It does not account for the loss of use of fish habitat measured by ocean space on the migration route, including feeding grounds, available to Fraser River sockeye. And, it is not in keeping with the science from Canada or internationally.[167]

The Department has in place an "Interim Guide to the Application of Section 35 of the Fisheries Act to Marine Salmonid Cage Aquaculture". This policy guides DFO's assessment of the environmental impacts of fish farms and whether a s. 35 authorization is necessary. The policy restricts the assessment of the impacts of farms to "benthic impacts" using a Depomod (deposition modeling) method or model.[168] Rebecca Reid (former Regional Director of Habitat Management; now Regional Director, Fisheries Management) agreed that DFO's policy is restricted to benthic impacts according to the Depomod model.[169] Benthic impacts are impacts or deposition on the sea floor. As such, so long as a fish farm will output waste, etc that is predicted to result in under a certain level of deposition, it is deemed to not cause a harmful alteration of fish habitat and no authorization is required (or CEAA triggered). On a later panel, Kerra Hoyseth (head biologist OHEB…) confirmed the focus of the habitat program for aquaculture has been benthic impacts.[170]

Disease is <u>not</u> part of what DFO has considered in applying its regulatory jurisdiction regarding impacts to wild salmon/fish habitat in the past. It does not factor in to site evaluations pursuant to s. 35 applications or licence applications. Ms. Reid testified that the Depomod model used to evaluate fish farm sites does not include consideration of pathogen transfer.[171]

As a matter of law and fact, there seems to be no reason why the placement of a fish farm, within the ocean environment, if an abnormal source of disease outbreaks and potential viral pathogen

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[166] Ex. 663- DFO Letters dated July 19, 2005
[167] April 5, 2011 Transcript, pp. 26:7- 29:2; *Fisheries Act*, section 34(1); Ex. 260 (Habitat Management Policy), p. 6
[168] Ex. 670 (Interim Guide to the Application of Section 35 of the Fisheries Act to Marine Salmonid Cage Aquaculture), p. 3
[169] April 5, 2011 Transcript, pp. 26:9-29:2
[170] Sept. 1, 2011 Transcript, pp. 11, 21-24, 31-33
[171] April 5, 2011 Transcript, pp. 28:26- 29:2

transmission, could not be seen as an alteration of 'fish habitat'. Moreover, the department has restricted its approach to HADD to regulating the kind of impact that each of the Project 5 experts determined would not be a significant threat to the health of wild salmon[172], reiterating that the department is not using effectively what could and should be a strong evaluative and enforcement tool.

Ms. Reid confirmed that the Department's approach means that generally, so long as a fish farm will not generate more than a standardized amount of waste in the water, "Letters of Advice" are issued.[173] According to the Department, a Letter of Advice is not an authorization pursuant to section 35 (and does not trigger a CEAA assessment.[174]

(e) No consideration of cumulative effects

DFO has also not undertaken an evaluation of the cumulative effect of a large number of farms on the salmon migration route generally, or in particular with respect to the cumulative effect of pathogen transmission/amplification/impact on wild salmon.

DFO lacks baseline data or ecosystem or cumulative impacts assessment in the marine environment generally. Mr. Carter testified that DFO lacks and needs baseline data on ecosystem health for the purposes of habitat monitoring. DFO plans to address the effects of projects more broadly, including on ecosystem health- but the department's efforts continue to be focused on project compliance monitoring and the broader evaluations remain goals, not realities. [175]

Mr. Carter's statements specific to habitat monitoring were echoed by DFO panelists:

- Ms. Reid agreed that the Depomod model did not take into account cumulative impacts.[176]
- Mr. LeBlanc stated that DFO's approach to habitat did not facilitate consideration of cumulative effects or ecosystem level habitat changes [April 5, 2011 Transcript, p. 46].
- Mr. Chamut stated that DFO's knowledge of the marine phase of the Fraser sockeye migration route was a "black hole".[177]
- Fish health scientists comments (including Kent, Johnson, Dill, Saksida[178]); and
- Sue Farlinger and Laura Richards[179]

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[172] Sept. 19, 2011 Transcript (Marmorek), p. 75
[173] April 5, 2011 Transcript, p. 28:8-21
[174] For example, for the 97 farms subject to the letters of July 19, 2005, DFO had determined that there was no HADD (and no CEAA trigger), so the farms were assessed by Transport Canada. In Ex. 1718-19, there is reference to 80 farms without authorizations.
[175] April 6, 2011 Transcript, p. 12, 19, 48, 53-54, 57, 63
[176] April 5, 2011 Transcript, pp. 28:26- 29:2
[177] Nov. 30, 2010 Transcript, pp. 119:26- 120:7
[178] Aug. 22, 2011 Transcript, p. 22, 60; Sept. 6, 2011 Transcript, p. 57
[179] Sept. 26, 2011 Transcript, pp. 63- 72, 77-79

(f) Looking forward- the evaluation of risk to wild salmon is still inadequate

There is no evidence of significant improvement of the approach of DFO with respect to cumulative impacts, disease or aquaculture generally in the marine environment since it assumed jurisdiction in December 2010. Importantly, DFO has made the unsupportable decision to <u>not</u> review existing licences for impacts or the appropriateness of site locations.

Though Ms. Reid testified that the new DFO licence would consider those impacts, subsequent witnesses established that for the 120+ fish farms that already exist on the Fraser River migration route, no such evaluation will be undertaken.

During the Aquaculture Compliance panel on Sept. 1, Kerra Hoyseth (head biologist…) confirmed that DFO would not be re-evaluating whether farms that had licences before Dec. 18, 2010 caused harmful impacts to fish habitat. She said:

> 36 Q Right. Okay. So there's monitoring thresholds,
> 37 but there's not sort of a re-evaluation of whether
> 38 or not it would a harmful -- that having the farm
> 39 there at all would be an impact to the fish
> 40 habitat. The farm is allowed to stay; is that
> 41 right?
> 42 MS. HOYSETH: Yes, it's correct.
> 43 Q Okay.
> 44 MS. HOYSETH: Our policy does not give us the
> 45 flexibility to go back and re-evaluate.[180]

Sue Farlinger testified that DFO has no intention of readdressing site selection for new sites: "We are not going back and reviewing each farm as if it were a new site".[181]

Ms. Farlinger further testified "that since 2003 the Department has relied upon the screening criteria "and developed it into – *with the help of Laura's folks and the circulation modeling*[182] and other things – a far more structured system for analyzing sites for the location of fish farms…". However that evidence does not accord with the remainder of the evidence. The siting criteria date back before 2001. They were not based upon a scientific basis. There has been no research into siting criteria. There has been no research into disease transfer to wild fish.

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[180] Sept. 1, 2011 Transcript, p. 58
[181] Sept. 22, 2011 Transcript, p. 82:7-9
[182] September 22, 2011, p. 79, Ms. Farlinger's evidence on this point must be viewed with some caution. The reference to "circulation modeling" follows upon the evidence of Laura Richards that a circulation modeling study is *underway* – i.e. future tense – there is certainly no evidence that such studies were in existence or relied upon and applied in 2003 or in any past siting decisions (except perhaps in respect of localized denthic impacts). The reference to 'with the help of Laura's folks' must also be closely examined given Laura Richards testimony that there was <u>no one in her Department</u> who had regulatory responsibility to give such advice (September 26, 2011, p. 73-74). There is no indication in the organizational charts of any individual with such responsibility prior to the changeover to federal responsibility in 2010, nor is there any indication in the Ex.s or the Ringtail database of any individual giving such advice, or even being asked for such advice in relation to any fish farm application.

'Laura's folks' have not been involved with siting decisions. The circulation modeling was not done earlier – it is only beginning now.

Moreover, DFO has taken the position, and several witnesses testified to the effect that in the future, there will be no s. 35 authorizations issued. Impacts will purportedly be evaluated and enforced through conditions of licence.[183] There will also be, as a consequence, no CEAA assessments in respect of future siting decisions. The legality of this approach is questionable;[184] but more significant for present purposes, from a scientific and a conservation perspective, it is of serious concern to exempt farms from section 35 and any consequent CEAA.

2. Failure to Regulate Impacts of Aquaculture

DFO has regulated without accounting for the environmental impact of aquaculture operations, and in particular, disease.

As noted above, for over two decades, DFO essentially abdicated its jurisdiction to regulate aquaculture as part of the fisheries of Canada and allowed the province to take responsibility. During that time, DFO deferred to the decisions and practices of the province, for both siting and regulation. DFO was left with some regulatory power (pursuant to sections 35 and 36 of the *Fisheries Act*) which applied to aquaculture. Yet, the record shows that DFO has taken a very minimal approach to using those habitat and fish protection provisions. Moreover, the evidence before the Commission suggests that, while changed in form, the regulatory regime going forward will not in any significant way expand the substance of DFO's regulation of impacts from fish farms; and, in fact may carry forward much of the former approach and/or restrict it further. This section reviews past and future regulation (with the exception of points relevant to evaluation of impacts and siting, which is discussed above).

(a) WSP- decision to treat as "any other activity"

Early documents relating to the development of the Wild Salmon Policy suggest that, originally, the department conceived of the policy, amongst other things, addressing four areas of significant impact to wild salmon (including through operational guidelines), one of which was aquaculture. Over time, however, the department made a decision to not specifically focus on aquaculture as a significant risk and/or focus of the wild salmon policy, but rather to treat aquaculture as akin to any other human activities that may pose a risk to wild salmon. This

[183] For example, see Aug. 30, 2011 Transcript, p. 31:13-23 (Swerdfager)

[184] Section 35(2) states that no person contravenes the s. 35(1) prohibition against HADD by means or under conditions authorized by the Minister. *The Canadian Environmental Assessment Act* requires an environmental assessment where there is a s. 35(2) *Fisheries Act* authorization. It seems untenable that an aquaculture licence can authorize an activity such that a fish farm is not in contravention of s. 35(1) but not be an authorization for the purpose of triggering CEAA.

decision regarding how (little) to treat aquaculture in the WSP was made conscious of the preference of the Province (and industry) that this be the case.[185]

Apparently in light of strong concerns expressed by First Nations and others with respect to aquaculture and the failure to address it in the Wild Salmon Policy, the department ultimately included a page in the WSP highlighting the department's efforts at regulating aquaculture and assuring the public that aquaculture would be regulated in keeping with the Policy.[186]

While the Department is satisfied that its approach to aquaculture (i.e. approach of recognizing no special status or peculiar relationship with or impact on wild salmon for aquaculture) is the right one, other stakeholders, including First Nations and ENGOs have not shared the same level of confidence.[187]

(b) Failure to exercise s. 35 power in an effective way:

In practice, DFO has not utilized its powers under the habitat protection provisions of the *Fisheries Act* in a way that recognizes, measures or controls risk of harm from fish farms. The shortcomings in the ways DFO applied its mandate for evaluation of impacts from fish farms are outlined above. This has had consequences for monitoring and enforcement, including:

Kerra Hoyseth, former lead biologist for the assessment of fish farms at OHEB and current lead biologist at the Aquaculture Environmental Operations branch in charge of contributing to licencing decisions described the record for habitat management as a strong foundation.[188] (]. Yet the evidence suggests otherwise. The concerns expressed in Audits and the evidence of other witnesses aside, the evidence regarding enforcement and management entered during the panel suggests a failure to adequately regulate.

Specifically, given the limited approach to assessment of impacts, DFO rarely found or issued a HADD authorizations. DFO then took the position that if it had not issued a HADD, then it had no basis to enforce habitat standards on a fish farm. **As impacts such as disease and cumulative effects were not evaluated, they were not subject to enforcement measures**. Enforcement has been minimal even with respect to benthic impacts.

According to Ms. Hoyseth, over the last decade, the monitoring program appears to have consisted of a benthic audit conducted by industry once a year with some field visits by DFO staff, mainly to check off basic benthic and operational parameters compliance.[189] This is a very minimal level of monitoring, in terms of content and frequency; and, does not provide a

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[185] Nov. 29, 2010 Transcript, p. 17; Nov. 30, 2010 Transcript, pp. 54-55, 57-60, 65-69; Ex. 83, p. 39; Ex. 78, pp. 11-15; Ex. 113; Ex. 116; Ex. 1913

[186] Nov. 30, 2010 Transcript, p. 60-3, 74; Ex. 8 (Canada's Policy for Conservation of Wild Pacific Salmon), p. 31; Ex. 114 (Email from Pat Chamut, April 7, 2004); Ex. 115

[187] Nov. 30, 2010 Transcript, pp. 70-74; Ex. 94

[188] Sept. 1, 2011 Transcript, p. 24:25-31

[189] Sept. 1, 2011 Transcript, pp. 10:46- 14:43

sufficient basis to ensure that fish farms are managing their impacts on wild salmon and their habitat.[190]

As noted above, there is no applicable regulation or condition of licence that DFO can enforce to prevent the occurrence of disease or transmission of disease from farms to wild salmon. Pathogens and disease are beyond the scope of s. 35 as interpreted by DFO. Andrew Thomson confirmed that there has never been a charge laid for any disease related event.[191]

With respect to HADDs and s. 35, Mr. Carter (of the Habitat Monitoring Unit in the Oceans Habitat Enhancement Branch) testified that compliance monitoring is limited to those projects which have obtained letters of advice or s. 35 authorizations.[192] With respect to aquaculture, it seems the aquaculture office/DFO[193] has taken an even more restrictive approach, and will only consider enforcement action where a s. 35 authorization has been issued.

In an email string between various DFO regarding whether to pursue enforcement of a farm exceeding provincial limits for benthic impacts but without a federal authorization, Ms. Hoyseth advised:

> …For farms that do not have Authorizations, we generally do not even know if a farm has exceeded thresholds under the FAWCR [the former provincial Finfish Aquaculture Waste Control Regulation, which was struck down by the BCSC as unconstitutional], as the industry has no requirement to report those to us. We do not have any licence or permit to legally bind the farms to meet any DFO monitoring or reporting requirements. …
>
> So, in this situation, it would be consistent with past action to let MoE [provincial Ministry of Environment] deal with this under the FAWCR….
>
> I think the only way DFO could move forward on this from a compliance standpoint would be to consider doing a HADD investigation, which I would suggest is not appropriate given they are lawfully operating their farm at themomemt, as are up to 80 farms in BC without Authorizations. How could we call this impact a HADD, when we aren't calling the impact from 80 other farms a HADD? This site may not have shown evidence of a HADD I the past, (if we are considering 1300 umol of sulphide a HADD threshold), but the majority of un-Authorized farms would show this level of impact…[194]

As noted above, DFO's policy going forward is that s. 35 authorizations will not be issued. When considered in light of DFO's past practice of not enforcing impacts unless an authorization

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[190] Of note, the two complaints by members of the public for Cyrus Rocks and Cecil Islands revealed non-compliance by fish farms and impacts to fish habitat that was <u>not</u> caught by government monitoring program. See Ex. 1718, 1719, 1720, 1721 and 1722; and Sept. 1 transcript, p. 61:38-46
[191] Sept. 1, 2011 Transcript, p. 21:4-15, p. 78:37-47
[192] April 6, 2011 Transcript, p. 57:23-29
[193] Mr. Carter's testified that the Habitat Monitoring Unit does not monitor aquaculture sites- his understanding is that that is done through the aquaculture office. April 6, 2011 Transcript, p. 81
[194] Ex. 1718

was issued, there is real concern that there will continue to be no/minimal monitoring or enforcement with respect to impacts from fish farms. It remains to be seen whether the pro forma conditions of licence, to the extent that they are relevant to this topic, will be interpreted by conservation officers in the future to be conditions that they will in fact enforce.

DFO has never pursued enforcement through to conviction of any fish farm operation; and, it is not clear whether they have charged a farm at all.[195] On the other hand, there is considerable evidence that they will overlook potential infractions or causes for concern:

a) Cyrus Rocks- members of the public and ENGOs raised concerns that resulted in a finding by DFO that the fish farm was generating (unacceptably) high levels of benthic impact; but, because to pursue enforcement against this one farm would mean having to pursue enforcement of up to 80 other non-Authorized farms, DFO recommended against enforcement. (Ex. 1718-1719)

b) Cecil Island- concerns from A. Morton and the Namgis First Nation of oily bubbles and potentially dead fish at the Cecil Island fish farm spurred both DFO and the province to investigate. DFO investigators witnessed bubbling, but nothing non-compliant (including after checking the farm records). The province found a pipe full of rotting fish. DFO representative (lead habitat biologist) Kerra Hoyseth later reported to Ms. Morton that the concerns regarding bubbles were not substantiated and they found no indication of non-compliance.[196] [on its face, a pipe full of dead fish would appear to contravene the FAWCR which regulates fish mortalities and should present an issue for s. 35 and 36 of the Fisheries Act; but this was not the conclusion of DFO.]

c) Exhibits 1723- 1725- are other emails demonstrating DFO knowledge of and acceptance of large scale non-compliance by fish farms of reporting and other regulatory requirements.

(c) Section 36- not applied by DFO to fish farms

Section 36 of the *Fisheries Act* prohibits the deposition of substances deleterious to fish. There can be no exemption except by regulation passed by the Governor-in-Council. There is little question that the several 1000 pounds of waste and feed that are generated by fish farms everyday and that enter the environment, as well as the chemicals and pharmaceuticals used to attempt to control disease and control the environment of the fish come within the definition of a deleterious substance. Further, the discharge of disease pathogens from the numerous disease outbreaks on fish farms could clearly constitute 'deleterious substances'. This represents a very powerful tool for the prevention of disease.

Nonetheless, the federal government does not apply the section to fish farms; and, its plans with respect to the future are unclear. There has, to the best of our knowledge, there has been no

[195] Sept. 1, 2011 Transcript, p. 21:4-15, 78:37-47 (Thomson and Atagi)
[196] Ex. 1720-22 and Sept. 1, 2011 Transcript

branch of DFO nor individual who even had the responsibility for such investigation or regulation in relation to disease and there has never been a charge laid, nor even an investigation, warning or letter issued, in relation to the many disease outbreaks.

RECOMMENDATION: ***DFO vigorously and diligently apply its regulatory and enforcement powers, including pursuant to sections 35 and 36 of the Fisheries Act, to aquaculture operations such that fish farms will be held to standards for impact to fish and fish habitat set out in statute, supported by science, and expected by Canadians.***

3. The inadequacy of control mechanisms

Norway, the methods of protection from disease *to farmed fish* are "is maintaining appropriate distances between fish farms" and "a ban on transportation of fish to and from infected farms".[197]

> *Effective vaccinations are available for only a limited number of serious fish viral diseases, leaving expensive compulsory stamping-out eradication as the official approach. If fish health authorities do not have an efficient risk management procedure, outbreaks may become epizootics. Rimstad, Exhibit 1482*

> *"In Norwegian aquaculture, viral diseases are mainly controlled by efforts to stop the transmission of viruses from initial outbreak area." Robertsen 1483*

Norway also has licences and fish health management plans for farms, similar to those in BC, but, disease still occurs. These same methods are followed in BC. These methods perhaps can protect farm fish (though there is still disease in farmed fish, despite these methods), but they cannot protect wild fish.

What Norway has done that BC has not is establish zones (fjords) where no fish farms are permitted. BC has *no* plan to establish zones where no fish farms will be permitted, even though several witnesses agreed this would assist in reducing risk from disease to wild sockeye.[198] Essentially, DFO does not use the most effective tool available to protect the Fraser sockeye from risk of disease and pathogens from fish farms- that is, by siting farms away from the migration route; and, in particular away from confined areas of that route.

Looking into the future, DFO intends to maintain all fish farms on the migration route that currently have licences (over 100 farms) and does not intend to re-evaluate the impact of those sites. DFO is placing great emphasis on the conditions of licence and specifically the Fish Health Management Plans to protect wild salmon. As is discussed below, neither of these is up to the task of preventing fish farms from changing disease dynamics for wild fish and increasing the risk of disease to Fraser River sockeye. Nor can we be confident that Canada's egg import policy

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[197] Ex. 1483 p. 128.
[198] Aug. 30, 2011 Transcript (Fleming and Last), pp. 67-70; Ex. 1803 "Protection, Restoration and Enhancement of Salmon Habitat"

guards against the introduction of exotic diseases. ***A more pro-active and precautionary approach is necessary.*** Such an approach starts with removing farms from at least the narrow passages of the Fraser sockeye migration route and should encompass removing all farms from the migration routes of wild salmon.

(a) DFO's New Aquaculture Licence- conditions of licence do not protect against disease

We repeatedly heard that the conditions of licence would be the primary (only?) tool by which DFO would regulate aquaculture, and its impacts on wild salmon, in the future. Those licence conditions do not provide sufficient protection to wild salmon, particularly against the impact of disease and the effect of the 100+ fish farms on the Fraser migration route.

The template for the 2010 DFO finfish aquaculture licence is in evidence as exhibit 1594. It was referred to many times as including a "comprehensive" and "complete" set of conditions for the regulation of fish farms.[199]

There are no conditions of licence that prohibit or prevent the transmission of disease to wild salmon, as confirmed by Andrew Thomson in cross-examination::

> 29 Q Is there a condition of licence that makes it
> 30 illegal for a fish farm to have pathogens on their
> 31 fish?
> 32 MR. THOMSON: No. We have conditions of licence that
> 33 require them having a Fish Health Management Plan,
> 34 which -- and that they must follow the Fish Health
> 35 Management Plan, which would, of course, the
> 36 design of which is to limit pathogens being on the
> 37 farm site.
> 38 Q Right. But it's intended to limit, but it doesn't
> 39 prohibit.
> 40 MR. THOMSON: There is no condition of licence that
> 41 prohibits pathogens on a farm site, no.
> 42 Q No. Nor the transmission from fish in the farm to
> 43 those that swim by.
> 44 MR. THOMSON: No, there is no specific condition of
> 45 licence that has that, no.[200]

Similarly, Dr. McKenzie testified:

> DR. McKENZIE: The Fish Health Management Plan doesn't
> 11 specify you can or cannot have a disease finding.
> 12 It's a process, it's a way of managing to mitigate
> 13 disease. So it would be hard to be in
> 14 contravention of that.[201]

[199] See for example, the testimony of A. Thomson, Aug. 30, 2011 Transcript, p. 32:27-37; 32:41-33:16
[200] September 1, 2011 Transcript, p. 52

With respect to disease, the licence includes the following:

- **Section 5- Fish Health Management Plan** (p. 8 and Appendix 5)- the FHMP is discussed below but generally at most reduces impacts of disease to farms, but does not prevent transfer to wild salmon;
- **Section 7- Fish Health Record Keeping** (p. 10-11 and App. VII, Fish Health Report)- In the past, reporting by fish farms of fish health (and the ability of the government to audit fish health) has been considered to be voluntary by fish farmers.[202] DFO maintains that now fish health and disease record keeping is mandatory and will be public. However, so far, they have not enforced the reporting requirements and reporting that has happened has not been made public.[203] Additionally, the amount of information that is to be recorded, and that which we can expect to be public, is not sufficient to keep DFO, and certainly not the public and independent researchers, informed and able to assess disease risks and impacts. [The amount of information that will be publicly disclosed is not nearly as comprehensive as that which was produced to the Commission as the fish health databases.]
- **Section 8- Fish Health Event Response** (p. 11)-This is the only section that sets out requirements for how a fish farm should respond to the outbreak of disease on a farm. The section gives wide latitude to the fish farm to determine how it will respond. There is nothing that requires fish farmers to take immediate action to protect wild salmon from the possibility of encountering disease from farms. There are no clear standards that can be enforced by DFO.

The sum of these provisions does not suffice to protect wild salmon from disease.

RECOMMENDATION: ***Aquaculture licences should include clear prohibitions on the spread of pathogens to wild salmon, with penalties for transfer of disease to wild salmon. DFO should prepare contingency plans for immediate action in the event of disease outbreaks, including a requirement to cull or withdraw fish from the ocean environment forthwith.***

(b) The Inadequacy of Fish Health Management Plans to Protect Wild Salmon

The evidence shows that the approach of DFO to protecting wild salmon from disease at salmon farms is almost entirely reliant on Fish Health Management Plans. The FHMPs are not an adequate to this task and are not a substitute for proper, science-based, precautionary siting decisions.

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[201] Aug. 31, 2011 Transcript, p.91
[202] Ex. 1682, Email string between A. Champagne and others ending April 30, 2010; PPR 20- Aquaculture Regulation in British Columbia, pp. 40-41
[203] Ex. 1725, Email string including M. McNabb ending June 21, 2011

In the past and now under DFO regulation, both DFO and the province appear to have relied substantially on the efficacy of the FHMP for addressing disease risk from fish farms to wild salmon.[204] As noted above, the CEAA assessments (Exhibits 1629 and 1630) reveal no scientific evaluation of the risk from disease but relied on the FHMPs. There was no other mechanism for including disease risk into siting decisions.

DFO continues to place all the emphasis on the FHMP for addressing disease risks from farms going forward. Numerous witnesses signaled to the FHMP as the guardian against disease impacts from fish farms. As an example, Sue Farlinger stated:

> "…the management of any impacts that may occur is basically focused on how the Fish Health Plan and the waste monitoring for the farm is managed."[205]

Andrew Thomson similarly referred to the FHMP, as quoted above.

Dr. McKenzie, a vet for the fish farms agreed that FHMP's manage risk, they do not prohibit disease:

> "DR. McKENZIE: But there's nothing that could prohibit disease.
> 43 Q No.
> 44 DR. McKENZIE: There's no piece of paper that could do
> 45 that. So what the Fish Health Management Plans do
> 46 is they take all the effort possible to mitigate
> 47 risk[206].

Similarly, DFO Pacific's head veterinarian, Dr. Mark Sheppard agreed that the FHMP is intended to reduce disease, but it cannot prevent it[207]. The Fish Health databases show that disease does in facto occur, despite the FHMPs.[208]

The FHMP was developed by the Province, as part of its purported jurisdiction to regulate property and civil rights, as a requirement for fish farms. It is consistent with and aimed at the commercial interest in maximizing fish health (for sale) and minimizing the impact of disease within farms. It does not extend into preventing the transfer of disease from farms to wild salmon. It is not an answer to protection of wild salmon from this significant risk. At present, the only reasonable means by which the risk can be vitiated, is to remove the farms from the migration route of the Fraser River sockeye salmon.

The concept of the proposed 'Area Management Plans', raised by senior DFO officials in the final days as a justification to ignore risks of siting decision directly on the migratory route is

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[204] The egg import policy was a tool aimed at preventing importation of diseased eggs; but, was and is not aimed at addressing the ongoing risk arising from the outbreak of disease in fish farms on wild salmon on an ongoing basis (for the life of the fish in the farm).
[205] Transcript Sept. 22, 2011, p. 83
[206] Aug 31, 2011, p.91
[207] Aug. 31, 2011 Transcript, p. 89:5-14
[208] See also, Aug. 31, 2011 Transcript (Mackenzie), p. 90:35-91:3

completely inadequate. There is no detail or explanation given as to how such plans could protect the migrating sockeye from disease transmission; and, as has been seen with the WSP, large-scale, lofty plans often are very difficult for the department to put into practice and implement in an effective way.

RECOMMENDATION: ***DFO recognize at a regulatory level that neither the conditions of licence, nor specifically the FHMP, can prevent the transfer of disease from fish farms to wild salmon. In recognition of this, DFO adopt other measures to protect wild salmon from this disease risk, particularly by siting farms away from Fraser sockeye migration routes.***

(c) Egg import regulation- ISA

As discussed above (in Part I), ISA is a disease with catastrophic effects. Based on international experience with ISA emerging where there is Atlantic salmon aquaculture, it seems only a matter of time before ISA is found in British Columbia. As is discussed below, for many years DFO has been aware of the impact of ISA in other regions. The federal government has dismissed concerns regarding the potential for it to appear in British Columbia

The department relies heavily on Canada's policy for importation of salmon eggs, at times without full knowledge of what its practices are.[209] Correspondence by Dr. Morton caused email chains between various DFO scientists and managers asking what the practices were. The re-assurances from the department have also contained errors or misleading information. For example:

- In her Dec. 20. 2010 email response to Dr. Morton, Dr. Richards advised that introductions of eggs were "closely tracked" by the Introduction and Transfers Committee, whose records stated that from 1995-2001, all eggs imported into BC came from Washington State [Ex. 1933, p. 2]. In fact, in 1995, eggs were imported from Ireland.[210]
- In the same response, Dr. Richards advised that from 2004-2009, all imports of eggs came from a single company in Iceland, a country where ISA has never been found and no reports of any clinical signs that might indicate viruses in their stock have been reported. She then went on to describe the screening of eggs and smolts, which have never found any signs of infectious agents. [Ex. 1933, p. 2] However, the documents disclosed to the Commission as a result of the Rule 18 and 19 requests by the Aquaculture Coalition and Conservation Coalition reveal that, in fact, Canada has permitted imports from the Icelandic facility despite it not meeting Canada's testing standards and not having certification, which permission to import Dr. Richards herself

[209] Ex. 1933, Email exchange between L. Richards, A. Morton and others ending Dec. 20, 2010; Ex. 1934, Email exchange between K. Garver, A. Thomson and others ending Oct. 23, 2009, Ex. 1830, Email exchange between J. Power, E. Porter and others ending Dec. 1, 2009; Ex. 1679, "Briefing Note to Minister" (Aug. 1, 2007)
[210] Ex. 1976, p. 36, table from DFO webpage http://www.pac.dfo-mpo.gc.ca/aquaculture/reporting-rapports/egg-oeuf-eng.htm; Updated version of table/webpage included in Ex. 1597

recommended.[211] Moreover, those same records show that, at least on one occasion, a broodstock from that facility was destroyed in B.C. seemingly because of concern regarding the presence of a virus.[212]

- In 2007, Dr. Sheppard, then Aquatic Animal Health Veterinarian, now lead DFO veterinarian for the Pacific Region, briefed the Minister regarding the ISA outbreak in Chile. Dr. Sheppard advised that "The most likely source for ISA in BC is from migrating wild fishes from other regions of the Pacific Ocean as there is no importation of live Atlantic salmon or eggs to BC."[213] This is false on the record of egg importations. Moreover, it is not consistent with the evidence that correlates fish farms and the introduction of ISA into a region worldwide. As noted above, Dr. MacWilliams testified to the Commission that if ISA is found in BC, it will be because of aquaculture.

Indeed, the egg importation and testing standards on their face and in practice are not a fail-safe way to prevent the transmission of ISA to British Columbia. And, the government does not have in place a system to sufficiently and proactively monitor the presence of diseases like ISA once smolts are introduced into marine fish farms. The Fish Health Certificate for egg imports does not include ISA test results.[214] Canada's testing of smolts allows for pooling of fish and does not meet the common standard of testing (5%) fish for the presence of ISA. Canada currently relies on self-reporting by industry of disease or audits of only a few fish taken from a farm at a time for ongoing monitoring. This is not sufficient. [215]

(d) Danger of introduction of exotic diseases

More generally, the evidence and legislative and policy framework demonstrates that the federal government has failed to install (and has even removed) lines of defense against exotic fish diseases entering this country.

- The Fish Health Certificate required for egg import is not adding screening requirement for new pathogens as they are identified and has never included ISA virus, even as it went global in 2007.

- There is no visible requirement for salmon farmers to report exotic diseases in their farms, including infectious salmon anemia.

- No mechanism exists to forward BCMAL reporting of classic lesions associated with exotic diseases to a review committee that should be on surveillance for emerging epidemiological patterns.

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[211] Ex. 1683, 2004 Fish Health 1, PDF pp. 2-5- "Briefing Note for the Regional Director General- Request to Import Atlantic Salmon Eggs from Iceland" (Oct. 3, 2003)
[212] Ex. 1684, 2004 Fish Health 2
[213] Ex. 1679, "Briefing Note for Minister" (Aug. 1, 2007)
[214] Ex. 1566, "Fish Health Protection Regulations, Manual of Compliance", p. 46
[215] Ex. 1566, Manual of Compliance; Ex. 1567, "International Response to Infectious Salmon Anemia- Prevention, Eradiction and Control", p. 26; Aug. 29, 2011 transcript, p. 56:10-57:46 (Korman); see also, Ex. 1976, Morton, "What is Happening to the Fraser Sockeye", pp. 33- 37

- Dr. Richards waived the Canadian Fish Health Protection Regulations in 2004 on request from the Norwegian salmon farm companies to allow eggs from a specific hatchery that does not meet Canadian requirements. Since that time all Atlantic eggs have come from that hatchery, including an entire shipment that was destroyed after hatching in BC due to viral issues not revealed.

- Disinfection of imported eggs is only a guideline and not mandated by Canada.

RECOMMENDATION*: No further importation of eggs be permitted.*

4. Split Mandate

There is no separation between promotion of aquaculture and regulation of aquaculture within DFO. The focus on promotion has been at the expense of regulation and wild salmon (impacts overlooked or not investigated).

(a) Same people promoting and regulating

The departmental structure vis-à-vis aquaculture inter-mingles promotion of aquaculture with its regulation; and, appears to favour promotion. There is both a perception of conflict and evidence to suggest that in fact there is conflict.

The Director General of Aquaculture Management (DGAM) is the highest ranking person within DFO charged exclusively with managing aquaculture. This position is currently held by Trevor Swerdfager.[216] [testimony Nov. 2]

The job description entered as evidence (Ex. 33) for the DGAM emphasizes the responsibility to promote aquaculture, including working with industry and removing regulatory impediments to industry. There is no mention of a responsibility to manage risks of aquaculture to wild salmon.[217]

During the two decades when DFO largely left regulation of aquaculture to the province, DFO did little to exercise its jurisdiction to protect wild salmon or fish habitat. Persons within the department deferred to the province with respect to habitat management, siting, and regulation. DFO did during that time facilitate and promote aquaculture.

Following the *Morton* decision, DFO has agreed to exercise its jurisdiction over aquaculture, which should include managing impacts to wild salmon. Many of the same higher-level members

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[216] Nov. 2, 2010 Transcript, p. 3:19-46 (Dansereau)

[217] Ex. 33; Nov. 2, 2010 Transcript, pp. 3:19- 19:31; While Ms. Dansereau testified that this responsibility to protect wild salmon was implied, including by the insertion of the word sustainable, nonetheless, the job description emphasizes promotion; and, with respect, though the department has for over a decade maintained that its policy is based on "sustainable aquaculture", DFO's commitment to or interpretation of that policy has and continues to be open to question [see, for example, the criticism by the Auditor General found in Ex. 188 and quoted below].

of the department in aquaculture continue to have prominent roles (for example, Andrew Thomson).

Mr. Swerdfager was the lead DFO representative in charge of public consultations and review during the development of the new Pacific Aquaculture Regulations (PAR). His job description includes the following:

> Fosters the streamlining and harmonizing of legal and policy frameworks to facilitate the growth of, and minimize impediments to, the sustainable development of aquaculture.[218]

The new regulations in fact "stream-line" licencing and regulation (and thus are in keeping with the above description). They achieve this by being extremely sparse and removing any set regulatory standards and the potential for a s. 35 *Fisheries Act* authorization and consequent CEAA review.

More specific to managing the potential risk to disease, during the final panel (DFO Priorities), Sue Farlinger advised that she was not aware of any person in DFO whose role was to respond to outbreaks of disease on fish farms.[219]

(b) Promotion/protection strategy with industry

The Commission has before it evidence that strongly suggests that at a policy level, DFO favours promotion of the aquaculture industry over strong regulation or protection of wild salmon.

Evidence was admitted showing coordination between industry and the department, at times at the expense of the public interest. Before testifying before the Commission in March, Laura Richards was briefed by members of the Department. The preparation of briefing information for Richards refers to a meeting on March 14th, 2011 (3 days before Richards testified) at which, amongst other things, Dr. Miller presented the latest development/hypothesis in her research, that being parvovirus. BCSFA, the main aquaculture industry group, was present at the meeting. BCSFA was privy to Dr. Milller's theory and part of a day of briefings of senior management/ an upcoming witness.[220] Note that the latest theory was not disclosed by Dr. Richards to the Commission during her testimony, nor to the public, until information relating to the internal DFO meeting in April, 2011 was disclosed to the participants.

Another example of coordination between industry and the department, at the expense of the public, is evident in the briefing note to the Director General Habitat Management from May, 2005. The memo addresses a communications strategy regarding DFO's habitat management with respect to aquaculture. Although the department's approach had recently (2004) been the subject of very serious concerns and criticisms by the Auditor General, the memo sets out the

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[218] Ex. 33; referred to on Nov. 2, 2011 at p. 7
[219] Sept. 26, 2011 Transcript, pp. 72:39-75:26
[220] Ex. 1526, Email from J. Stewart (March 14, 2011)

department's strategy to convince the public that it was doing a good job managing impacts to habitat. It refers to a meeting with and communications strategy coordinated with industry.[221]

The evidence with respect to the Department's minimal approach to evaluation, monitoring and enforcement is described elsewhere, but is pertinent here to the extent it demonstrates a trend toward protection of the industry over protection of wild salmon. Moreover, the evidence introduced during the compliance panel reveals a practice of shielding farms from public concern. In two separate incidences relating to two separate farms, First Nations, ENGOs, members of the public contacted DFO regarding concerns about environmental impacts and/or regulatory violations. In both cases, non-compliance was subsequently detected by DFO but in neither case did DFO pursue enforcement, nor inform the public of the non-compliance. In one, DFO expressed wonder why the public would be interested or concerned; and, there is no evidence that DFO disclosed their finding of non-compliance.

Additionally, as is discussed further in the next section, DFO has utilized its science function to promote the aquaculture industry far more than it has to protect wild salmon.

The end result is the impression that DFO prioritizes promotion of the industry. It does so at risk to wild salmon and the public, and, in failure of their mandate to preserve and protect the fisheries of Canada.

RECOMMENDATION: ***The federal government separate regulation from promotion of aquaculture. DFO's primary function ought to be the regulation of aquaculture; and, any personnel focused on promoting industry ought not to be involved in licencing or regulatory decisions.***

5. The Role of Science

(a) The Absence of science

The department has done very little research into the impact of fish farms on wild salmon, particularly in the area of disease transfer or cumulative impacts. In addition to evidence cited elsewhere in these submissions, the evidence shows that despite Miller's disease research being a leading hypothesis for the decline of salmon/2009 return and reasons to consider links to farm, the department has not researched any potential link between fish farms and the Miller research. [see Ex. 1936, a media release document that states that the department has not considered the link between the Miller virus/genomic signature and fish farms and Ex. 636 letter from the Minister to A. Morton dated March 3, 2011 stating the same; this despite Miller early hypothesizing a potential link to fish farms/SLV and her requests for funding, including in April, 2010- Ex. 639]. (As is cited elsewhere in these submissions), the experts and DFO witnesses who testified regarding the potential impacts of fish farms and/or disease were fairly united in

[221] Ex. 661, "Briefing Note for Director General of Habitat Management, Meeting with the BCSFA re: Public Confidence in Aquaculture"

agreement that research into disease in wild fish and potential interrelationships between disease in farmed and wild fish has been lacking and ought to be given greater attention.

In this regard, the author of the Cumulative Impacts report, which compiled the opinion and conclusions from all the other technical reports, David Marmorek testified that studies with respect to disease transfer would not be particularly difficult to conduct:

> 9 A That's correct. We do recommend that we actually
> 10 get some disease data.
> 11 Q Yes.
> 12 A And actually, so did Noakes and Dill.[222]
> …
> 21 So I think you can gather information on
> 22 disease and make sensible decisions, if you get
> 23 the data.
> 24 Q So it's possible to design studies that would show
> 25 these links, if they're there?
> 26 A I think so.
> 27 Q And that's not really rocket science, is it?
> 28 That's a pretty obvious way to do it, isn't it?
> 29 A It is good fishery science. ...[223]

There is no supportable reason why DFO has not done more and better research with respect to disease impacts of aquaculture on wild salmon. There is an abundance of international literature establishing the link, independent scientists in BC have emphasized the potential for fish farms to increase and change pathogens for wild fish, the department has long been aware of the need to do this research.

The limits to DFO's assessment of the impacts of aquaculture on fish and fish habitat have and continue to be the subject of criticism in reports by the Auditor General (including as recently as 2009). In the 2004 report, the Auditor General stated:

> The three audits [conducted in 1997, 1999 and 2000] also found significant gaps in the scientific knowledge about the potential effects of salmon aquaculture. Fisheries and Oceans Canada's Aquaculture Policy Framework expresses a strong commitment to developing a sustainable aquaculture industry in Canada. But when assessing applications for aquaculture sites, the Department needs to apply more credible, science-based criteria to ensure that approved sites are properly located. It has had difficulty assessing the cumulative effects of salmon aquaculture on wild salmon stocks. And it has to determine how to control the deposit of deleterious substances by salmon aquaculture operations. Wild salmon and habitat remain susceptible to the effects on salmon aquaculture. [Forward]
>
> 5.1 Overall, we are not satisfied with the progress made by Fisheries and Oceans Canada in responding to the recommendations we made in the three previous audits in

[222] Sept. 19, 2011 Transcript, p. 76
[223] Sept. 19, 2011 Transcript, p. 77

> 1997, 1999, and 2000. While many stocks are abundant, some Atlantic and Pacific salmon stocks are in trouble. We continued to identify significant gaps in managing risks.
>
> …
>
> • There are shortcomings in information on salmon stocks and habitat and scientific knowledge on the potential environmental effects of salmon aquaculture in aquatic ecosystems.
>
> • There are weaknesses in regulatory approvals, enforcement, and monitoring of salmon aquaculture operations. This includes approving aquaculture site applications, assessing cumulative effects, and monitoring salmon aquaculture operations to prevent harmful destruction of habitat.
>
> …
>
> 5.75 Our current audit found that, while some research has been undertaken or is ongoing, significant gaps still exist with respect to the needed research on the potential effects of salmon aquaculture in aquatic ecosystems and on wild salmon stocks. The Department, through its state of knowledge initiative, identified significant gaps in knowledge about far-field environmental effects of finfish aquaculture and the use of chemicals on finfish aquaculture in Canada. We also observed that sufficient knowledge of the risks and potential effects of salmon aquaculture on wild salmon does not exist in several areas such as diseases, sea lice, and escapes of farmed salmon from aquaculture sites.[224]

When asked about the finding of the Office of the Auditor General in the 2004 Report, Mr. LeBlanc acknowledged :

> MR. LeBLANC: I can't comment on that section. I do
> 42 realize that we have science gaps in many things
> 43 that we do, many of the activities that we
> 44 regulate. It is an ongoing challenge in terms of
> 45 having scientifically sound information and
> 46 knowledge to be able to support our decision, and
> 47 this is just one of the examples of the lack of
> 1 knowledge and information we may have about the
> 2 interaction of aquaculture or an industry sector
> 3 with fish and fish habitat. [April 5, 2011 Transcript, p. 24-25]

(b) Science- to support industry

That there has not been a focus on studying disease impacts of fish farms on wild salmon reflects the structure and priorities of DFO. The job description for the Division Head, Aquaculture lists

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[224] Ex. 88 (Report of the Commissioner of the Environment and Sustainable Development to the House of Commons, ch. 5 Department of Fisheries and Oceans- Salmon Stocks, Habitat, and Aquaculture), pp. ii, 1 and 15

first planning, developing and managing scientific research … in support of the aquaculture industry" first.[225] Studying interactions with and impact to wild fish is second. Dr. Laura Richards and Claire Dansereau agreed that research from Science has been "client" oriented, and, research into wild salmon, and impacts to wild salmon have not been a focus and needs more work.[226]

Although it is difficult on the documents disclosed by Canada as part of this inquiry to discern the amount of funding dedicated to aquaculture research, it seems clear that the vast majority of the funding has gone directly to projects that are in the corporate interests of aquaculture operators as opposed to projects that have as their primary purpose understanding or preventing risks of impact to wild salmon. This is demonstrated by the kinds of priorities and projects funded by the main funding programs for aquaculture research, particularly the AIMAP and PARR.[227]

(c) Science- mis-information

Additionally, DFO appears to dedicate departmental research and resources to protecting farms from public criticism.

Laura Richards, Regional Director of Science testified that she had only been involved in writing speeches or speaking notes for Parliamentary debate on one occasion. That was in the midst of political and public attention to the low return in 2009. On that single occasion, the speaking notes dedicated several pages to positing why sea lice from fish farms could not be the cause of the decline. Only a few lines were dedicated to the department's view of what the cause could be (stated to be marine conditions), with no mention of disease at all (though this was a, if not the, leading hypothesis in the department at the time). The emphasis on diverting attention away from the role of fish farms and failure to advise regarding disease (which would presumably bring attention to how DFO was responding to disease) calls into question the department's priorities.[228]

In August, 2009 Pacific Region Director General ADM Paul Sprout wrote a letter to the editor published in the Globe and Mail asserting: " Sea lice from fish farms are not the explanation for this year's extremely poor marine survival of Fraser River sockeye."[229] This letter to the editor was drafted and published *before* the department had considered the various hypotheses and evidence with respect to the decline, including the role of sea lice and in the absence of evidence.[230] There is no evidence that the department followed up this move with any subsequent editorial regarding the scientific hypotheses for the decline, in particular disease. On the contrary, the evidence

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[225] Ex. 33, Doc. 32
[226] Sept. 26, 2011 Transcript, pp. 63- 72
[227] Ex.s 1729- 1732
[228] March 17, 2011 Transcript, p. 27:4-36 and Ex. 626
[229] Ex. 60
[230] Ex. 643, Email string including T. Davis and others ending Aug. 19, 2009 confirms that DFO did not have the evidence for sea lice at the time of the letter. See also Ex. 644. The workshops into the causes of the 2009 decline did not happen for another month after the letter to the editor was written.

suggests a reluctance by DFO to provide information to the public or media regarding the Miller research or the disease hypothesis.[231]

As with the sea lice letter, DFO's research and communications strategy around aquaculture consistently includes assertions about the lack of risk associated with fish farms. On its webpage, DFO refers to suggestions or publications regarding risk "myths" or "surges of mis-information"):

- Ex. 61 is the DFO webpage "Facts about Sea Lice", which includes statements that:

> "There continues to be surges of (mis)information circulated about sea lice and its impact on aquaculture and wild salmon stocks in British Columbia.
>
> Are fish farms in the Broughton Archipelago causing a dramatic increase in sea lice levels?
>
> No….
>
> What does DFO research suggest in the debate on the potential impact of sea lice from salmon farms on wild salmon stocks?
>
> Since 2003, DFO and others (e.g.: th Pacific salmon Forum), have conducted extensive field and laboratory research in the potential origins of sea lice and their relationship to the health of wild salmon populations in the Broughton Archipelago.
>
> DFO research has not been able to demonstrate a link between the levels of sea lice that may be present on the farms and the number of wild adult pink salmon returning to the Broughton Archipelago. …
>
> Is it true that DFO research supports claims that sea lice from salmon farms is broadly infecting and harming wild pink salmon stocks in the Broughton Archipelago?
>
> No, this is not true….
>
> Is it true that sea lice are killing juvenile sockeye salmon?
>
> No. Sea lice from salmon farms cannot singularly explain the extremely poor marine survival of Fraser River sockeye…
>
> With the assortment of opinions on sea lice, what should the public believe?
>
> There are different and dissenting opinions on the status of sea lice in the Broughton Archipelago. Many researchers and environmental organizations believe that salmon farms are causing poor returns of adult wild salmon stocks, and that if salmon farms were eliminated, the sea lice problem would be too. This is just not factual. Much research exists that lays to rest the all-encompassing claim that sea lice is putting all wild salmon at risk…."

- Ex. 62 is the DFO webpage "Myths and Realities about Salmon Farming", which includes statements that:

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[231] March 17, 2011 Transcript, pp. 65-70; See discussion in Part I re: Miller Research

> Myth #3: No one is supervising salmon farms.
>
> > Reality: DFO, other federal departments, and provincial governments monitor fish farms. It is mandatory…undergo a lengthy environmental assessment…Habitat Officers routinely review sites in order to prevent harmful alteration, disruption or destruction of the oceans and freshwater habitat. In fact, the Fisheries act is one of the strongest pieces of environmental legislation in Canada.
>
> Myth #10: Farmed salmon spread disease to wild salmon.
>
> > Reality: Wild salmon have built up natural tolerances to the diseases and parasites with which they have naturally co-existed for centuries…
>
> Myth #11: Sea lice from farmed salmon are destroying pink salmon stocks in BC.
>
> > Reality: There is no confirmed evidence that this is the case….
>
> Myth #13: The Science on the aquaculture issue seems confusing.
>
> > Reality: Our scientific research is rigorously peer-reviewed and reported openly by being posted on our Web Sites….

Mr. Bevan testified that the two DFO webpages [Ex. 61 and 62] are meant to communicate DFO knowledge to the public and that it is consistent with DFO knowledge on the subject.[232] With respect, these webpages provide mis-information to the public, or mis-communicate to the public, by overstating concerns or other research regarding risks from fish farms as well as the extent of DFO knowledge, research and management. The effect is to promote farmed fish, supporting public confidence in the industry and DFO's management thereof, potentially at the peril of wild salmon.

What makes this approach particularly suspect is absence of, or contrary to, real data or research generated or obtained by DFO regarding risks, particularly disease and fish farms. Without data from British Columbia, and with a consistent international experience regarding disease impacts for fish farms on wild stocks, DFO's approach looks like it is favouring promotion and protection of the industry over regulation of aquaculture and protection of wild fish.

(d) Sea Lice and 'Rebuttal Science'

There is little doubt that sea lice, left unchecked as they have been at times in the past, can constitute a major adverse impact on wild fish in the vicinity of fish farms. We do not know the magnitude of those impacts on wild Fraser sockeye; however that does not mean that there are no adverse effects – it is highly likely that there are some. The majority of the research that has been done has focused on pink salmon, particularly in the Broughton area.

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[232] Nov. 4, 2010 Transcript, pp. 125:7-126:7

In relation to the issues before this Commission, the seriousness of potential impacts from disease from fish farms is likely much more significant than direct mortality from sea lice, in relation to the 2009 decline. In this regard, we accept the evidence of Dr. Dill.[233]

That does not mean that sea lice have had no impact. The evidence is clear that sea lice can be a disease transmission vector, and that sea lice can weaken fish and make them more susceptible to disease. If the Commissioner determines that the cause of the long term decline in sockeye, or the cause of the 2009 decline is 'death from a thousand cuts', then sea lice are clearly one of those cuts.

In relation to the long term decline of sockeye (and recommendations for future sustainability), the potential for harm from sea lice plays a larger role. Although the pesticide SLICE is currently being used to temper sea lice numbers on fish farms, it is highly likely that sea lice resistance to this pesticide will develop, as it has in other regions where it is used (including the east coast of Canada and Norway). The argument put forward by that Pacific sea lice are somehow genetically different is not compelling. Over time, more and more SLICE will be used (with uncertain impacts to the external environment), and a greater proportion of sea lice will survive to infect wild fish. The solution is not to create new, more potent pesticides, but rather to move fish farms to locations where they cannot harm wild fish.

The real significance of the sea lice evidence, in the context of this Commission, is to demonstrate the pattern of DFO response to a potential pathogen threat to wild salmon – i.e., how DFO decides to balance its mandate to protect wild salmon with its desire to promote the aquaculture industry. That response, in the sea lice context, can be seen as:

- denial;
- delay and inaction;
- response – finally provoked by media and political attention;
- 'rebuttal science' – studies designed to critique the science identifying issues with and negative impacts of sea lice, and enable further delay in action[234];
- media disinformation / reassurance (as demonstrated in the above sub-section);
- partial acceptance, accompanied by claims that the problem has been solved
- further years of inaction or partial response (under the guise of further studies);
- coordination (conspiracy) and cooperation with industry throughout.

There is no better example of the 'rebuttal science' approach than the sea lice controversy. Extraordinary attempts by DFO to find excuses to discredit strong, peer reviewed science is the best evidence available of the overwhelming bias within DFO caused by the political agenda to protect the aquaculture industry.

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[233] Project 5D Report (Dr. Dill), pp. 29-30

[234] Over the last decade (subsequent to initial research by independent scientists reporting adverse effects from sea lice in the Broughton Archipelago), DFO scientists have published numerous such papers on sea lice. See Ex. 1764, 1765, 1766, 1767, 1769, 1770, 1771, 1772, 1773, 1774, and 1775.

What has been achieved with this 'ping pong match', an extraordinary effort in expense by DFO. It has only been to diminish the credibility of scientists. The sea lice science is said to be "controversial" – but the controversy only arises because of the effort and ingenuity of DFO scientists who spend time publishing rebuttals, and rebuttals to rebuttals, and to, instead of replicating the original studies showing the threat, instead design studies to examine the variables seen to be potential weaknesses. How else does one explain three studies on sticklebacks?

RECOMMENDATIONS:

- ***DFO prioritize scientific research that impartially investigates the impacts of aquaculture on wild salmon stocks and aimed at ensuring the conservation of wild salmon and salmon habitat.***
- ***DFO stop orienting its science toward improving aquaculture's image and specifically from rebutting independent science that raises concerns about the impact of aquaculture.***
- ***DFO stop funding research which has as a clear objective assisting industry in increasing output and financial return (by technological advance or otherwise). This research is in the corporate interest of aquaculture companies and the cost ought to be borne by them alone.***

6. Precautionary principle and risk-based management

> "*and our science has always been – the advice that we have always received from our scientists has always been that there is no threat at this point, or there is no threat that we are completely aware of...*"
> [Deputy Minister Claire Dansereau[235]]

This statement by the Deputy Minister exposes the approach DFO has taken to aquaculture. This approach is directly contrary to the precautionary principle and the risk-based approach by which DFO purports to manage aquaculture and fisheries generally. According to the Department's own policy on the use of precaution, scientific uncertainty shall not be used to postpone decisions where there is a risk of serious or irreversible harm.[236]

Specifically:

- DFO has done little to no scientific research into potential risks to wild salmon from fish farms with respect to disease, despite correlations in timing between the decline of wild salmon and the presence of fish farms and disease in farms (specifically marine anemia), and, despite independent scientific opinion and experience from around the world suggesting or reporting negative impacts from aquaculture on the health of wild stocks;

[235] Sept. 26, 2011 Transcript p. 78:26-40
[236] Ex. 51, "A Framework for th e Application of Precaution in Science-Based Decision-Making About Risk", p. 2

- In the absence of absolute certainty that there is a risk (or perhaps in denial of the evidence suggesting that there is) posed by aquaculture to wild salmon, DFO has proceeded on the basis that aquaculture poses no disease risk to wild salmon;
- DFO has not assessed the cumulative effect of farms on wild salmon migration and health and thereofre cannot assess any risk in that respect;
- DFO has and continues to promote the aquaculture industry and its expansion, for economic gain. [There is no other reason to introduce millions of foreign fish into BC coastal waters than for financial interests. There is no reason at all to place the farms on the Fraser sockeye migration route.]
- With the assumption of regulation of aquaculture, DFO decided that past risk-assessment and siting decisions based on those assessments were not going to be reviewed again.

This means that over 100 farms located on the wild salmon migration route are permitted to remain into the future, despite the short-comings of earlier research, the growing evidence of the correlation between aquaculture and disease in wild stocks, and the fact that a leading hypothesis for the decline of the Fraser sockeye, and the poor 2009 returns, is disease. ***At a minimum, DFO must now recognize that there is a potential issue with disease that requires further study and precautionary measures.***

To allow the farms to remain is not good risk-based management and certainly does not accord with a precautionary approach. Moreover, to allow new farms or expanded operations in the marine waters of BC, and specifically on the Fraser migration route, before better, reliable research is undertaken would be a further affront to these guiding principles.

The department's current approach does not, and cannot protect wild salmon from the potential harms from aquaculture. This approach is unacceptable to the Aquaculture Coalition, and, to the overwhelming number of persons in this province who have expressed their concern, including by written submissions to this Inquiry, about how the department is regulating the industry.

RECOMMENDATIONS:

The Aquaculture Coalition strongly urges the Commission to recommend that:

- ***DFO re-visit its decision to grandfather existing farms on the migration route;***
- ***Those farms be removed until further, supportable research is undertaken and no new farms be sited on the migration route until that research is completed; and,***
- ***DFO regulate aquaculture truly using a precautionary approach that puts wild salmon first.***

APPENDIX A:

THE PROCESS WAS INADEQUATE TO THE TASK

Many members of the public looked to this Commission to be an inquiry into the aquaculture industry and its harm to wild fish. The mandate of the Commission, and its choice of process, failed to do that. The process provided only a glimpse into the harm caused by aquaculture. This was not a fair fight, like a trial, where both sides are given an equal chance. The Aquaculture Coalition was consigned to a role where it fought not just with one hand behind its back, but with both – there was no right to call witnesses or fair chance to produce evidence and no proper rights of cross-examination.

Despite the fact that disease was rated by DFO and most scientific workshops on the decline of Fraser sockeye as a 'likely to very likely' contributing cause of the 2009 decline, and despite the correlation between the long term declines of Fraser stocks and the expansion of aquactulture into the sockeye migration route in the early 1990s, this Commission allocated one day to the topic 'fish farms and disease'.[237]). One.[238] And on that day, the Commission called four expert witnesses, none of whom was independent; none of whom was prepared to give evidence against the aquaculture industry. The Aquaculture Coalition was given 30 minutes – to cross-examine four expert witnesses – 7 ½ minutes per witness, all hostile -- in relation to thousands of pages of disease database, regulatory background, and 20 years of history.[239]

As a participant, the Aquaculture Coalition was not allowed to call any witnesses. Only the Commission counsel could call witnesses.[240] Dr. Morton eventually appeared as a witness by the Commission and in the 'Perspectives' panel. She was not permitted by Commission Counsel to give expert evidence.

The Aquaculture Coalition was advised that no independent expert reports could be produced by participants, only witnesses called by commission counsel were to provide expert testimony in the Aquaculture hearings. However, when the fish farmers (BCSFA) produced (one-sided)

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[237] The Fish Health panel, Aug. 31, 2011

[238] August 31, 2011

[239] Any lawyer will recognize that such opportunity is sadly deficient. All we can say in respect of the fairness of this process is that the other participants labored under the same limitations. Cross-examination, constrained in this way by time, is alien to our system of justice. Any skilled bureaucrat or expert witness can easily obfuscate and avoid dealing with the point through verbiage for many minutes, if they know that the cross-examination time is so limited. Many did. Further, the obligation to give notice – one week ahead – of any documents to be used in cross-examination; which requires that cross-examination be planned and outlined before the witness has even given evidence, and the witness has full notice of the points to be raised in cross-examination, weakens the value of cross-examination.

[240] The commission counsel did consult with participants about witnesses. Through this process, witnesses like Dr. Dill and Dr. Fleming were eventually produced by the Commission. But, as Commission witnesses, their testimony was determined by commission counsel rather than a proponent, as would occur in a trial. And those witnesses were far outnumbered by government and industry witnesses. Dr. Dill, in his preparation of his report, was directed by Commission counsel, not by a participant, as would be the case in a trial.

expert reports, they were ultimately accepted by the Commission and entered as exhibits – without any rights of participants to cross-examine the authors.[241]

The ability to call evidence for the Aquaculture coalition was limited to *cross-examination* of witnesses chosen by the commission. The rules on cross-examination, the very brief time allocations, meant that cross-examination had very limited value. Counsel for the government or industry objected when documents were put to witnesses who did not author them or had no knowledge of them. So with little control over witnesses, and so little time, much potential relevant evidence could not be produced.

In a trial, two sides face off with an equal ability to prove facts, to call witnesses, to produce experts and expert reports, and to test the evidence of the other side through cross-examination. This is a system, developed over centuries under British and Canadian law, to form a reliable search for truth. That one side could call no witnesses, could produce no expert reports, and be limited to mere minutes of cross-examination per expert against them is a recipe for failure -- if an objective search for truth is the desired outcome.

Thus, in this proceeding, the public interest organizations that formed the Aquaculture Coalition were not given a chance to prove anything in respect of the harms from fish farming, *and the Commission Counsel did not undertake that role*. This was not a fair fight, like a trial, where both sides are given an equal chance. The Aquaculture Coalition was given a very limited role, and limited funding. More importantly, however, the nature of the process meant that the Aquaculture Coalition was consigned to a role where it fought not with one hand behind its back, but with both – there was no right to call witnesses or fair chance to produce evidence and no proper rights of cross-examination.

None of this is intended as criticism of the Commission, the Commissioner, or Commission Counsel, who acted throughout with professionalism, integrity and impartiality. It is to point out that this limited fact-finding ability was inherent in the design of the process, and the breadth of the mandate given to the Commission within the time allowed. The Commission had an impressively broad task, and on policy and many of the topics covered may well have adequate evidence to draw conclusions. The public needs to understand that was not true on aquaculture, which the Commission treated as just one of a great many issues.

On aquaculture, the choice to limit the evidence to nine days at the end of the hearing, and then to divide those nine days into six panels, and call multiple witnesses for each panel, many of them supportive of DFO, left little chance for reliable fact-finding on a complex matter. Given the number of participants, the time for cross-examination for each witness on a panel became so reduced to be ineffective and meaningless to the task – if the purpose of cross-examination is to test the evidence. Instead, cross-examination became an attempt to get a few exhibits in, and to test one or two points.

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[241] See correspondence of July 13, July 18, and Aquaculture submissions on admissibility of BCSFA expert reports.

In those circumstances, normal rules of assessing witness evidence become valueless. Because the witness evidence is untested on a particular point, does not mean it is accepted, as might arise in a trial where the right of cross-examination is not artificially limited. Because a particular document or statement has not been put to a witness, does not mean it should be given lesser weight. Because a particular proposition has not been proven (e.g. for or against fish farms) cannot be taken as evidence weakening that proposition, when the participant has been given no chance to prove it – other than by a minimal chance at cross-examination.

The choice of Commission Counsel to take an impartial or neutral stance, while appropriate in some inquiries, was unfortunate in this time-limited environment. Commission Counsel generally took up the largest segment of time on each day with their questioning, followed by government (Canada and BC). In taking a neutral stance, Commission Counsel could be understood to leave it to the other parties to raise controversial matters or to challenge the weaker parts of the evidence. However, by giving inadequate time to the other participants to do so, the process failed. The net result is that on most days, 90% of the evidence consisted of relatively non-controversial or self-serving evidence, given in most cases from the government's side.

We continue to have strong confidence in the judgment of the Commissioner, and the capacity of the Commission to make helpful recommendations.

What we ask of the Commissioner, under these circumstances, is to be careful to explain to the public the limitations of this Inquiry. We believe there is compelling evidence of the potential risks of fish farms to wild sockeye salmon, and compelling evidence that the government has failed to appropriately address that risk. However, the Commission must be careful not to draw conclusions or to comment on the weakness or lack of evidence on a particular point, where no opportunity to provide or test that evidence has been given.

Documents

We should also make an observation about documents.

It will be said that there were 600,000+ documents in this case, produced mostly by government, and entered into the ringtail database. That was a phenomenal and commendable effort by the Commission, and by Canada, its civil servants and legal counsel. That database should be a treasure trove to assist in the Commission's work.

However, the decision of the Commission to make all such data secret *ab initio*, and to allow only those documents produced as exhibits (in the end, under 2,000) is very regrettable. The result is that unless a document was presented during the hearings, it cannot be referred to in argument *or used by the Commissioner*. Every counsel at this hearing had the experience of presenting a long list of documents which they intended to have entered as exhibits, only to find that the limited time for cross-examination precluded more than a few. On many days, the number of exhibits entered amounted to less than 20% of the documents listed by participants' counsel. In the end, the other 80% -- deemed to be important enough to list -- have been lost to the Commission and the public.

It would have been better to allow participants to address in argument all relevant documents, and not just those they were fortunate enough to have entered in the limited time available.

It will be tempting for the Commission, or the media, to mention the total number of documents produced (ie. 600,000 plus) as providing support for the 'comprehensive' nature of the inquiry. But, because of the rules adopted, we suggest that would be misleading. We suggest that the Commissioner has not, and will not, read all those documents -- nor likely has any participant or its counsel. Those documents, including many relevant ones, are now lost to the Commission and to the public, unless the Commissioner releases participants and their legal counsel from the undertaking, and declares these documents public.

We ask the Commissioner to ensure that these documents are not lost.

APPENDIX B – DISEASE DATABASES

These databases disclose a great number of histopathological findings consistent with disease. They also disclose the extent to which Dr. Marty, Dr. Sheppard and other veterinarians have, through careful application of definitions, failed to disclose information of vital importance to the public interest and of regulatory concern. Reliance on 'summary' or aggregated data does not suffice.

2850 (Exhibit 1549-206)

TAB: 1 Farm- Column Q contains a great deal of data in relation to fish farm audits.

IHN mentioned six times, lines. 3, 4, 5, 11, 17, 167.

BKD mentioned in 70 visits out of 503.

Marine Anemia:

l. 214 "high number of mortalities may be due to marine anemia".
l. 262, "mostly marine anemia seen in mortalities." (had been treated for BKD)
l. 287, "silvers are mainly made up of marine anemia (70-80%)".
l. 331 "three marine anemia"
l. 332 (Culloden) "197,261 chinook on site. Fish 6 was a moribund with pale gills… all samples anaemic"
l. 355 "three marine anemia"
l. 385 "1029 mortalities were categorized by farm as silvers, but they were actually BKD."
l. 397 "three marine anemia".
l. 398 "marine anemia".

TAB 3 – Histology, Column I

- "no significant findings" in only 82 of 496 examinations. In other words, there were 'significant findings' in 496 (84%).
- "Infectious etiology unknown" in 52 listings; "no infectious etiology" in two more
- IHN/VHS identified seven samples.[242]
- ISH supportive of marine anemia (2006 Farm – P.2-21)
- "Findings most consistent with marine anemia (ISH) but no direct evidence. Farm 2006.4 P. 3-22 216.[243]

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[242] All of these in 2005, contrary to Dr. Noakes.
[243] Note the reference to "ISH" here and farm P.2-21 above. Dr. Marty attempted to disavow that as a reliable evidence of marine anemia in his testimony. However, in his own documents, that is what he used .

- "23 of 24 had hematopoietic hyperplasia compatible with marine anemia, but no direct evidence (clinical signs include anemia) – Farm 137, 2006.4 Farm P.3-24 (Conville Bay).
-

TAB 5 – ViroVirus – Column E – an indication of suspected viruses, based on histology, for which PCR tests were done. IHNV shown 933 times; IPNV 933 times; ISA 934 times; VHS 933 times; note that there were 357 tests for ISA in 2006-7 (182 in 2007).

TAB 12 – Cause Other l. 100 (2007) "marine anemia"; l. 59 (2006) (Farm P.2-21) "marine anemia".[244]

TAB 15 – HistoMorph – 256 entries for "rain" under column G

"Hyperplasia of lymphoid tissue in the kidney" or "hyperplasia of interstitial tissue" (in kidney diagnosed 60 times)

Interstitial hemorrhage or interstitial hemorrhage/congestion an additional **85 times**.[245]

TAB 16 – Histo Summary: "Marine Anemia" appears 4 times (Farms P.2-1, P.3-19, P.3-11, P.3-18)

TAB 17 TentDx: "Marine Anemia" appears 5 times – note line 216 (2006Q1), and line 231(2006Q4) – Farm P.3-24 (Conville Bay in the Discovery Islands)

Sort by Column – "Open" appears 107 times, "No Significant Findings" – twice

"BKD" 57 times

TAB 18- FARM Dx "Open" appears 152 out of 583 times (72 in 2007)

"Marine Anemia" appears 3 times, BKD 64 times

2864 (Exhibit 1549-217)

"Classic Symptoms of ISA"
HEM Column AY (Atlantic): 908/2259 positive
SSC Column AF (Atlantic): 745 of 2259 positive

"Classic Symptoms of Marine Anemia"
ISH (Atlantic Tab), Column AT: 175/2259
ISH (Pacific Tab), Column AT: 291/447

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[244] These entries conflict with Dr. Marty's testimony that marine anemia is not found.
[245] Interstitial cellular hyperplasia is described by Dr. Kent and Dr. Stephen as the key indicator of marine anemia.

BCP001645

TAB FH Audit notes and Diag: Column S Shows the "disease at population level" (13 times) even though, in Columns P,Q, and R there are many other mentions of disease. Note Column T – "Diagnosis" shows "Open" for numerous lines.

Salmon Alphavirus:

There are 8 reports to the salmon farming companies of the lesions associated with Salmon Alphavirus, the number of these case increased in 2009. This virus causes several diseases in Norway including Pancreatic disease which is now epidemic in Norway.

> "Rena eosinophilic granules have also been described in Atlantic samon naturally infected with chrinc pancreas disease in Norway (Salmonid alphavirus subtype 3, SAV3;McLoughlin and Graham 2007), but SAV3 has not been identified in BC salmon)"

Exhibit 1549-304 (BCP002957) Case# 07 4140

Exhibit 1549-309 (BCP002962) Case# 08 697, Case#08 3362

Exhibit 1549-318 (BCP002971) Case# 09 1914, Case# 09 3819, Case# 09 2849, Case# 09 2969, Case# 09 3542

HSMI

There is one case of the diagnostic lesions of Heart and Skeletal Muscle Inflammation, a frequently lethal salmon disease (http://www.plosone.org/article/info:doi/10.1371/journal.pone.0011487) reported to Mainstream in 2008 by Dr. Marty. This is a newly discovered virus and was diagnosed this month for the first time in Chilean salmon farms this month. This is thought to be caused by Salmon Alphavirus and indeed Dr. Marty reports lesions of both in an Atlantic salmon in 2008

> "This pattern of inflammation has also been described with Heart and Muscle Inflammation in Atlantic salmon readed in Europe, but this disease has not been identified in BC salmon."

Exhibit 1549-309 (BCP002962) Case# 08 3362

Chilean Coho Virus

There have been 2 recent reports by Dr. Marty of a virus that has been reported, but not fully identified in Chilean Coho. There is concern that this virus may be a strain of Infections Salmon Anemia. Both of his reports were in Chinook salmon and made to Dr. Sonja Saksida

> "The clinical signs in these fish are similar to what is thought to be a viral infection in coho salmon cultured in Chile (Smith et al. 2006)."

Exhibit 1549-324 (BCP002977) Case# 10 1347, Case# 09 113

IPNV

There is one report to Marine Harvest in 2010 of symptoms of Infectious Pancreatic disease.

> "The lesion is considered characteristic of IPNV infections (pp. 190, "Systemic Pathology of Fish" Second edition, 2006, edited, by H. Ferguson), but IPNV has never been identified in farmed salmon in BC. "

Exhibit 1549-322 (BCP00297 Case# 10 2700

ISA Reports to CFIA
Case# 07-1353, Case# 07-1859, Case# 07-2120, Case# 07-2123

Case #08-4567, Case# 08-4813, Case# 08-533, Case# 08-2143,

Case# 09-2492 Case# 09-26, Case# 09-109, Case# 09-111, Case# 09-711, Case# 09-805, Case# 09-805Case# 09-1617 Case# 09-1714 Case# 09-1766 Case# 09-1932 Case# 09-1999 Case# 09-2477 Case# 09-2594 Case# 09-2849 Case# 09-2936 Case# 09-2969 Case# 09-3042 Case# 09-3272 Case# 09-4967

Case# 10-1442 Case# 10-314 Case# 10-329 Case# 10-799 Case# 10-1034 Case# 10-1368

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(FOR REFERENCE ONLY)

Histopathology of Pacific salmon sampled as part of the BC Auditing and Surveillance Program

#	Most significant Lesion	Cause of Death	KIDNEY - ISH	HEM
1	SRS	SRS	3	0
2	ISH	none	3	1
3	HRS	HRS	3	0
4	EPL	none	2	0
5	KRS	KRS	2	0
6	LRS	LRS	2	0
7	LRS	LRS	2	0
8	LRS	LRS	2	0
9	LFN	none	2	0
10	LKR	none	2	0
11	ISH	none	2	0
12	KRS	KRS	2	0
13	KRS	none	2	0
14	KPS	KPS	2	0
15	cholecystitis	none	2	0
16	LPS	LPS	2	0
17	SRS	SRS	2	1
18	CPL	none	2	1
19	LRS	LRS	2	1
20	LPS	LPS	2	1
21	hepatic vasculitis and nec	hepatic vasc	2	0
22	HRS	HRS	2	0
23	HTH	none	2	0
24	SLS	none	2	0
25	CPL	CPL	2	0
26	SLS	none	2	1
27	GLS	GLS	2	0
28	SLS	none	2	0
29	SLS	none	2	1
30	SLS	none	2	0
31	KLS	none	2	0
32	LPS	LPS	2	1
33	LPS	LPS	2	0
34	ISH	none	2	0
35	ISH	none	2	0
36	HRS	HRS	2	0
37	KRS	KRS	2	0
38	KRS	KRS	2	0
39	KRS	KRS	2	0
40	KRS	KRS	2	1
41	nephritis, neutrophilic,	nephritis,	2	0
42	GLS	GLS	2	1
43	KRS	KRS	2	0
44	LRS	LRS	2	0
45	HRS	HRS	2	0
46	KRS	KRS	2	0
47	SRS	SRS	2	0
48	KRS	KRS	2	0
49	LRS	LRS	2	0
50	KRS	KRS	2	0
51	HRS	HRS	2	0
52	GLS	none	2	1
53	LPS	LPS	2	0
54	KPS	KPS	2	0
55	KRS	KRS	2	0
56	HRS	HRS	2	0

#	significant Lesion	of Death	ISH	HEM
57	LRS	LRS	2	0
58	HRS	HRS	2	0
59	GLS	GLS	2	2
60	LGR, CPL	LGR, CPL	2	2
61	KRS	KRS	2	1
62	KGR	KGR	2	0
63	BRS	BRS	2	0
64	KRS	KRS	2	1
65	ENH, ECH	ENH, ECH	2	0
66	KRS	KRS	2	1
67	HTH,ECH ('Pacific Salmo	none	2	0
68	BGR	BGR	2	1
69	SSF, renal hematoma	SSF, renal h	2	0
70	LPS	LPS	2	0
71	KRS	KRS	2	0
72	BRS	BRS	2	0
73	GLS	GLS	2	0
74	GLS	GLS	2	0
75	KRS	KRS	2	0
76	SCN	none	2	2
77	EPL	EPL	2	0
78	BRS	BRS	2	1
79	BRS	BRS	2	0
80	LRS	LRS	2	0
81	HRS	HRS	2	0
82	HRS	HRS	2	0
83	HRS	HRS	2	0
84	KRS	KRS	2	0
85	KRS	KRS	2	1
86	LGR,ECH	LGR,ECH	2	0
87	IFB	IFB	2	0
88	HRS	HRS	2	0
89	KRS	KRS	2	0
90	LRS	LRS	2	0
91	SCN,PFB	SCN,PFB	2	0
92	ISH	none	2	0
93	MEN	none	2	1
94	GLT	none	2	0
95	PFB	none	1	0
96	HGR	none	1	0
97	ENL	none	1	0
98	SRS	none	1	0
99	ENL	none	1	0
100	GRS	none	1	0
101	SSF	none	1	0
102	SCN	none	1	0
103	LIP	none	1	0
104	KRS	KRS	1	0
105	ENH	none	1	0
106	SCN	none	1	0
107	HRS	HRS	1	0
108	HRS	HRS	1	0
109	ISH	none	1	0
110	SSC	none	1	0
111	KRS	KRS	1	0
112	VAC	none	1	0
113	KRS	KRS	1	0
114	HRS	HRS	1	0
115	MGN	none	1	0

#	significant Lesion	of Death	ISH	HEM
116	EPH	none	1	0
117	MGN	none	1	0
118	VAC	none	1	0
119	GLS	GLS	1	0
120	GLS	GLS	1	0
121	GLS	GLS	1	0
122	GLS	GLS	1	0
123	LPS	LPS	1	0
124	SSF	SSF	1	0
125	BPH	none	1	1
126	GLS	GLS	1	0
127	splenitis, neutrophilic	none	1	0
128	ISH	none	1	0
129	LPS	LPS	1	0
130	GLS	GLS	1	0
131	MGN	none	1	0
132	KPS	KPS	1	0
133	KPS	KPS	1	0
134	SSF	none	1	0
135	SLS	none	1	0
136	LFN	none	1	0
137	GLS	GLS	1	0
138	GLS	GLS	1	0
139	LFN	none	1	0
140	SLS	none	1	0
141	KLS	none	1	0
142	SLS	none	1	0
143	ENH	none	1	0
144	SLS	none	1	0
145	HLS	none	1	0
146	ENL	none	1	0
147	EPL	none	1	1
148	HLS	none	1	1
149	HLS	none	1	1
150	HLS	none	1	0
151	MGN	none	1	1
152	KRS	KRS	1	0
153	PGP	none	1	0
154	SCN	none	1	0
155	HGR	HGR	1	0
156	HLS	none	1	0
157	HGR	none	1	1
158	SSF	none	1	1
159	SSF	SSF	1	0
160	KRS	KRS	1	0
161	KRS	KRS	1	1
162	KRS	KRS	1	0
163	KRS	KRS	1	1
164	SCN	none	1	0
165	GLS	GLS	1	0
166	BGR	none	1	0
167	KRS	KRS	1	0
168	GLS	GLS	1	0
169	PVL	none	1	0
170	GLS	GLS	1	0
171	GLS	GLS	1	0
172	MGN	none	1	0
173	KGR	none	1	0
174	SCN	SCN	1	0

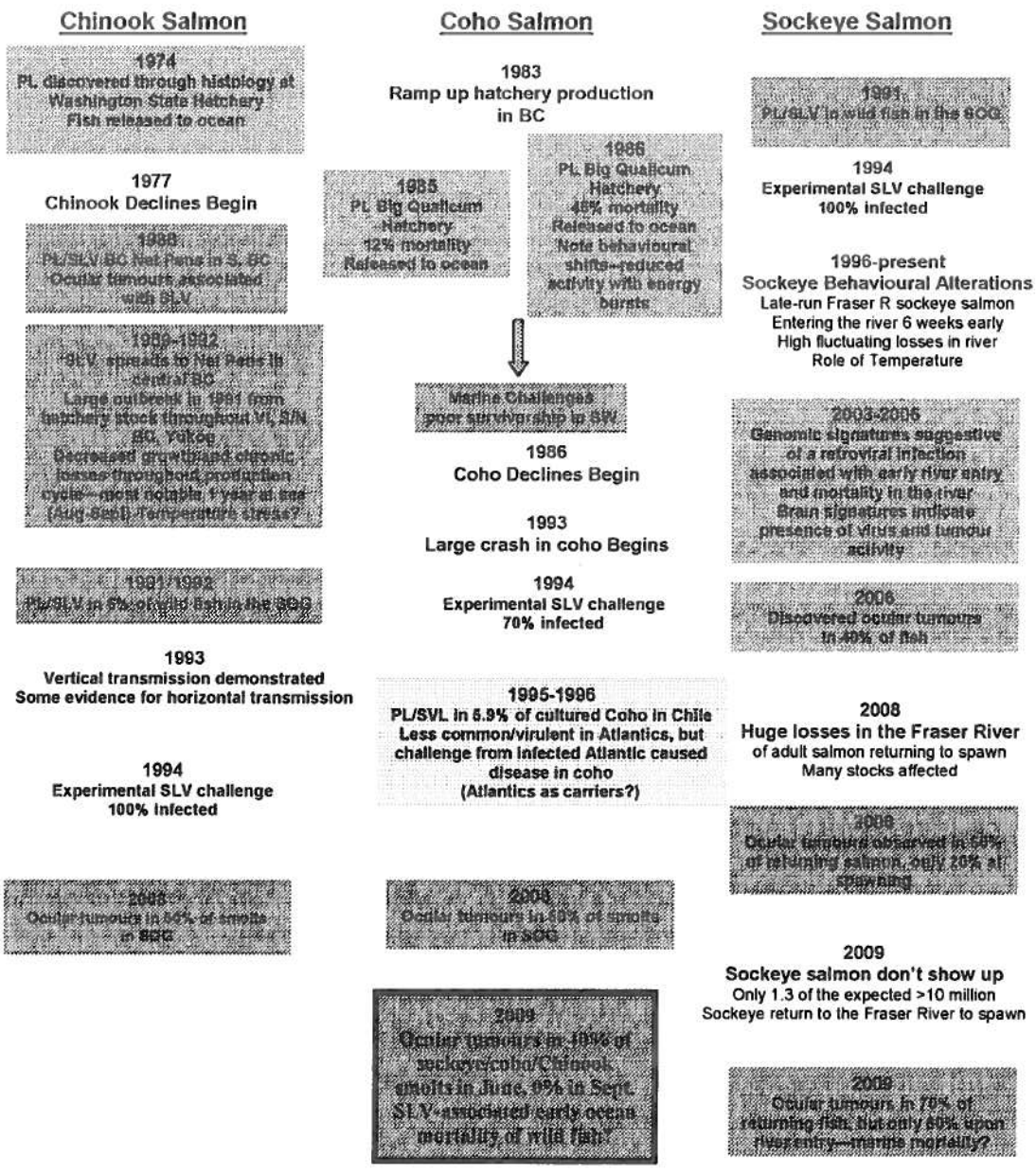
#	significant Lesion	of Death	ISH	HEM
175	EPH	EPH	1	0
176	KRS	KRS	1	0
177	HRS	HRS	1	0
178	HRS	HRS	1	0
179	HRS	HRS	1	0
180	KRS	KRS	1	0
181	KRS	KRS	1	1
182	KRS	KRS	1	0
183	KRS	KRS	1	0
184	KRS	KRS	1	0
185	LKR	none	1	0
186	KRS	KRS	1	0
187	KRS	KRS	1	0
188	KGR, IFB	KGR, IFB	1	0
189	KRS	KRS	1	0
190	MEN	none	1	0
191	KRS,BHM	KRS,BHM	1	0
192	LRS	LRS	1	0
193	GLS	GLS	1	0
194	GLS	none	1	2
195	KRS	KRS	1	0
196	HRS	HRS	1	0
197	KRS	KRS	1	0
198	SSF, VHSV	SSF, VHSV	1	0
199	GLS	GLS	1	0
200	KRS	KRS	1	0
201	LRS	LRS	1	0
202	KRS	KRS	1	0
203	HEM	none	1	0
204	HEM,SCC	HEM,SCC	1	0
205	HEM,SCC	HEM,SCC	1	0
206	BRS	BRS	1	0
207	BRS	BRS	1	0
208	MEN	none	1	1
209	BLS	BLS	1	2
210	BPC	none	1	0
211	CPL	none	1	0
212	SGR	none	1	0
213	GRP	none	1	1
214	VAC	none	1	0
215	GLS	GLS	1	1
216	BPS	BPS	1	1
217	BCC,MCC	BCC,MCC	1	1
218	CPL	none	1	2
219	RTN	none	1	2
220	GLS	GLS	1	2
221	KRS	KRS	1	2
222	HRS	HRS	1	1
223	BRS	BRS	1	0
224	KRS	KRS	1	1
225	KRS	KRS	1	0
226	EPL, CPL	EPL, CPL	1	0
227	KRS	KRS	1	1
228	BRS	BRS	1	0
229	BRS	BRS	1	0
230	HTH,ECH ('Pacific Salmo	HTH,ECH ('	1	1
231	HTH,ECH ('Pacific Salmo	HTH,ECH ('	1	0
232	PFB	PFB	1	0
233	SSF,HTH ('Pacific Salmor	SSF,HTH ('I	1	0

#	significant Lesion	of Death	ISH	HEM
234	BRS	BRS	1	0
235	CPL	none	1	0
236	BRS	BRS	1	0
237	GLS	GLS	1	0
238	LRS	LRS	1	1
239	KRS	KRS	1	1
240	GLS	GLS	1	1
241	KRS	KRS	1	1
242	BRS	BRS	1	0
243	AIB	none	1	0
244	HRS	HRS	1	0
245	BRS	BRS	1	0
246	LKR	none	1	0
247	MEN	none	1	0
248	KRS	KRS	1	0
249	KRS	KRS	1	0
250	SSC,HRS	SSC,HRS	1	0
251	KRS	KRS	1	0
252	LRS	LRS	1	1
253	MEN	none	1	0
254	MEN	none	1	0
255	CPL	none	1	1
256	KRS	none	1	0
257	MEN	none	1	0
258	BCC	none	1	0
259	PFB	none	1	0
260	RTN	none	1	0
261	BPC	none	1	0
262	PGP	none	1	1
263	IPR	none	1	1
264	MEN	none	1	1
265	LRS	LRS	1	1
266	BRS	BRS	1	1
267	BRS	BRS	1	0
268	KRS	KRS	1	1
269	BHM	none	1	0
270	IFB,SCN	IFB,SCN	1	0
271	LRS	LRS	1	0
272	HRS	HRS	1	0
273	SSF,SCN	SSF,SCN	1	0
274	IFB,SCN	IFB,SCN	1	0
275	BPH	none	1	0
276	CPL	none	1	0
277	MEN	none	1	0
278	KRS	KRS	1	0
279	HRS	HRS	1	0
280	GLS	none	1	0
281	MEN	none	1	0
282	KRS	KRS	1	0
283	SSF,LFN	SSF,LFN	1	1
284	mbosis, posterior cardinal , posterior ca		1	0
285	dermatitis, ulcerative, with	dermatitis, u	1	0
286	dermatitis, ulcerative, with	dermatitis, u	1	0
287	dermatitis, ulcerative, with	dermatitis, u	1	2
288	IPR	none	1	0
289	PFB	none	1	0
290	SSF	none	1	0
291	HTH	none	1	0
292	PGP	none	0	0

#	significant Lesion	of Death	ISH	HEM
293	PGP	none	0	1
294	MIN	none	0	0
295	PFB	PFB	0	1
296	GGR	none	0	0
297	renal thrombosis	none	0	1
298	PGP	none	0	0
299	RTN	none	0	0
300	MGN	none	0	0
301	MGN	none	0	0
302	SCN	SCN	0	0
303	KRS	KRS	0	0
304	LIP	none	0	1
305	SCN	none	0	0
306	CPL	none	0	1
307	EPH	none	0	0
308	LIP	none	0	0
309	VAC	none	0	0
310	TEP	none	0	0
311	RTN	RTN	0	0
312	TEP	none	0	0
313	PER	none	0	0
314	none	none	0	0
315	HEM	none	0	0
316	none	none	0	0
317	MIN	none	0	0
318	VAC	none	0	0
319	RTN	none	0	0
320	BPH	none	0	0
321	VAC	none	0	0
322	MGN	none	0	0
323	PGP	none	0	0
324	HLS	none	0	1
325	none	none	0	1
326	VAC	none	0	0
327	HRS	none	0	1
328	ECH	none	0	0
329	MGN	none	0	0
330	HLS	none	0	0
331	renal thrombosis	none	0	1
332	MEN	MEN	0	1
333	KRS	KRS	0	0
334	SRS	SRS	0	0
335	VAC	none	0	0
336	IRS	IRS	0	0
337	KRS	KRS	0	0
338	RTN	none	0	0
339	KRS	KRS	0	0
340	IRS	IRS	0	1
341	BRS	BRS	0	0
342	TDI	none	0	0
343	SSF	none	0	0
344	HEM,BCC	none	0	0
345	LRS	LRS	0	0
346	IPR, BCC	IPR, BCC	0	0
347	GLS	none	0	0
348	GLS	GLS	0	0
349	GLS	GLS	0	0
350	VAC	none	0	0
351	BCC	BCC	0	0

#	significant Lesion	of Death	ISH	HEM
352	BCC	BCC	0	0
353	BHM	BHM	0	0
354	BRS	BRS	0	0
355	ENL	none	0	0
356	IPC	none	0	0
357	hepatic vasculitis	none	0	0
358	MEN	none	0	0
359	BRS	BRS	0	0
360	LRS	LRS	0	0
361	BRS	BRS	0	0
362	myonecrosis, with filamen	myonecrosis	0	1
363	SRS	SRS	0	1
364	BRS	BRS	0	0
365	MEN	MEN	0	0
366	KRS	KRS	0	1
367	GLS	GLS	0	0
368	BCC	BCC	0	0
369	KRS	KRS	0	0
370	MEN	none	0	2
371	BLS	BLS	0	0
372	HLS	none	0	1
373	BGR	none	0	1
374	MEN	none	0	0
375	BRS	BRS	0	0
376	KRS	KRS	0	1
377	KRS	KRS	0	0
378	KRS	KRS	0	0
379	KRS	KRS	0	0
380	KRS	KRS	0	1
381	KRS	KRS	0	0
382	MEN	none	0	1
383	BCC,HEM,SSC	BCC,HEM,S	0	0
384	PGP	none	0	1
385	LFN, VHSV	LFN, VHSV	0	0
386	BCC,EGC	none	0	1
387	BHM	none	0	1
388	MEN	none	0	0
389	MEN	none	0	1
390	BCC	BCC	0	0
391	BCC	none	0	0
392	IPC	none	0	0
393	BCC	none	0	0
394	BCC	none	0	0
395	RTN	RTN	0	0
396	KRS	KRS	0	0
397	LRS	LRS	0	0
398	BRS	BRS	0	0
399	BPC	none	0	0
400	CPL	none	0	0
401	MEG	none	0	0
402	BCC	none	0	0
403	BRS	BRS	0	2
404	MEG	none	0	0
405	SSC, *Listonella anguillaru*	SSC, *Liston*	0	0
406	SSC, *Listonella anguillaru*	SSC, *Liston*	0	1
407	SRS	SRS	0	1
408	*Listonella anguillarum* Ty	*Listonella a*	0	0
409	KRS	KRS	0	0
410	KRS, BHM	KRS, BHM	0	0

#	significant Lesion	of Death	ISH	HEM
411	BPC	none	0	0
412	BCC	none	0	0
413	BCC	none	0	0
414	BCC	none	0	0
415	IPR	none	0	0
416	MEN	none	0	0
417	VAC	none	0	0
418	MEN	none	0	0
419	BRS	BRS	0	0
420	BHM,BRS	BHM,BRS	0	0
421	BRS	BRS	0	1
422	BHM,BRS	BHM,BRS	0	0
423	BRS	BRS	0	0
424	PER	none	0	0
425	TDI	none	0	0
426	CPL	none	0	0
427	PGP	none	0	0
428	MEN	none	0	1
429	BRS,BHM	BRS,BHM	0	0
430	MEN	none	0	0
431	BCC	none	0	0
432	PFB, *Listonella anguillaru*	PFB, *Liston*	0	1
433	renal mycosis	renal mycos	0	0
434	BRS	BRS	0	1
435	KRS	KRS	0	1
436	SSC	none	0	0
437	BHM	none	0	1
438	stomatitis, ulcerative, with	stomatitis, u	0	0
439	stomatitis, ulcerative, with	stomatitis, u	0	1
440	HRS	HRS	0	0
441	stomatitis, ulcerative, with	stomatitis, u	0	1
442	dermatitis, ulcerative, with	dermatitis, u	0	0
443	Skin: panniculitis, lymphol	none	0	2
444	hepatic sinusoidal neutrop	none	0	0
445	ECH	none	0	0
446	EPL	none	0	0
447	hepatic vasculitis	hepatic vasc	0	1


Chinook Salmon
1974
PL discovered through histology at Washington State Hatchery
Fish released to ocean
1977
Chinook Declines Begin
1988
PL/SLV BC Net Pens in S. BC
Ocular tumours associated with SLV
1989-1992
SLV spreads to Net Pens in central BC
Large outbreak in 1991 from hatchery stock throughout VI, S/N BC, Yukon
Decreased growth and chronic losses throughout production cycle—most notable 1 year at sea (Aug-Sept) temperature stress?
1991/1992
PL/SLV in 5% of wild fish in the SOG
1993
Vertical transmission demonstrated
Some evidence for horizontal transmission
1994
Experimental SLV challenge
100% infected
2008
Ocular tumours in 54% of smolts in SOG
Coho Salmon
1983
Ramp up hatchery production in BC
1985
PL Big Qualicum Hatchery
12% mortality
Released to ocean
1986
PL Big Qualicum Hatchery
48% mortality
Released to ocean
Note behavioural shifts--reduced activity with energy bursts
Marine Challenges
poor survivorship in SW
1986
Coho Declines Begin
1993
Large crash in coho Begins
1994
Experimental SLV challenge
70% infected
1995-1996
PL/SVL in 6.9% of cultured Coho in Chile
Less common/virulent in Atlantics, but challenge from infected Atlantic caused disease in coho
(Atlantics as carriers?)
2008
Ocular tumours in 69% of smolts in SOG
2009
Ocular tumours in 10% of sockeye/coho/Chinook smolts in June, 0% in Sept.
SLV-associated early ocean mortality of wild fish?
Sockeye Salmon
1991
PL/SLV in wild fish in the SOG
1994
Experimental SLV challenge
100% infected
1996-present
Sockeye Behavioural Alterations
Late-run Fraser R sockeye salmon
Entering the river 6 weeks early
High fluctuating losses in river
Role of Temperature
2003-2005
Genomic signatures suggestive of a retroviral infection associated with early river entry and mortality in the river
Brain signatures indicate presence of virus and tumour activity
2006
Discovered ocular tumours in 40% of fish
2008
Huge losses in the Fraser River
of adult salmon returning to spawn
Many stocks affected
2008
Ocular tumours observed in 50% of returning salmon, only 20% at spawning
2009
Sockeye salmon don't show up
Only 1.3 of the expected >10 million
Sockeye return to the Fraser River to spawn
2009
Ocular tumours in 70% of returning fish, but only 40% upon river entry—marine mortality?

AQUACULTURE COALITION
PROPOSED RECOMMENDATIONS FOR COHEN COMMISSION

Siting:

1. Siting: Fish farms should be removed from the major Fraser sockeye migration routes as soon as practicable. Fish farms in the confined channels within the Discovery Islands and Johnston Strait should be removed immediately.
2. DFO should identify proximity to a Fraser sockeye migration route as a priority siting criterion; and, siting criteria should prohibit farms in close proximity to migrating salmon until DFO has conducted sound, supportable, peer-reviewed research into disease interactions between wild salmon and aquaculture operations.
3. DFO recognize at a regulatory level that neither the conditions of licence, nor the FHMP, can prevent the transfer of disease from fish farms to wild salmon; and, that the disease risk is significant and unacceptable. DFO should adopt other measures to protect wild salmon from this disease risk, by siting farms away from Fraser sockeye migration routes.
4. The siting of all existing fish farms, which were transferred in December 2010 without detailed site review, should be reviewed by DFO, and with a full public, science-based process.
5. Future siting decisions should be based upon an appropriate zoning plan to pre-determine suitable locations, reached through a formal public process, with local community and First Nations involvement, and based upon a precautionary approach to pathogen risk to minimize contact with wild salmon populations.

Disease Reporting and Monitoring:

6. DFO needs to establish regulations, and an appropriate regulatory arm, to require strict, open and independent disease and sea lice monitoring and response. Full disease testing and auditing data, including raw data, should be open and available to other scientists and to the public.
7. Canada must establish regulations allowing DFO scientists to test salmon farms for the presence of potential new diseases, including live fish, without requiring consent of the farm operators, and should fund current studies to determine the presence or absence of parvovirus and salmon leukemia virus on all current farms.
8. DFO should institute a program to audit helath and disease in live fish on a regular basis.

Research:

9. Research: The science and research function of DFO should be given structural independence and freedom from political interference. Canada should separate the science and research function from the political and aquaculture promotion functions of DFO. Aquaculture research should be funded by the aquaculture industry and not by government.

10. DFO should prioritize scientific research that impartially investigates the impacts of aquaculture on wild salmon stocks and the ocean ecosystems and aimed at ensuring the conservation of wild salmon and salmon habitat.
11. Research into disease in wild salmon and aquaculture; and disease interactions between the two, be given highest priority.
12. Research should include comprehensive research into the potential parvovirus and/or salmon leukemia virus, its origins and a strategy to mitigate or eliminate it and conduct genomic profiling on all Atlantic salmon farms.
13. Sufficient funding should be provided to ensure the above research is scientifically-sound and supportable.
14. DFO stop orienting its science toward improving aquaculture's image and specifically from rebutting independent science that raises concerns about the impact of aquaculture.
15. DFO stop funding research which has as a clear objective assisting industry in increasing output and financial return (by technological advance or otherwise). This research is in the corporate interest of aquaculture companies and the cost ought to be borne by them alone.
16. DFO re-visit its decision to grandfather existing farms on the migration route;
17. Those farms be removed until further, supportable research is undertaken and no new farms be sited on the migration route until that research is completed; and,
18. DFO regulate aquaculture truly using a precautionary approach that puts wild salmon first.

Regulation

19. Remove the aquaculture industry promotion, liaison and public affairs functions from DFO, and give DFO a clear mandate to protect the wild salmon and other species from aquaculture and aquaculture pathogens.
20. DFO: Section 35 and 36 of the Fisheries Act should continue to apply and be enforced in respect of impacts to fish and fish habitat (defined in keeping with the Fisheries Act and to include more than benthic impacts) and the deleterious deposit of substances from fish farms. DFO should not exempt fish farms from pollution rules. Field staff should have support from Ottawa to enforce compliance. The habitat regulations should be vigorously and diligently applied to aquaculture operations such that fish farms will be held to standards for impact to fish and fish habitat set out in statute, supported by science, and expected by Canadians.
21. Aquaculture licences should include clear prohibitions on the spread of pathogens to wild salmon, with penalties for transfer of disease to wild salmon. DFO should prepare contingency plans for immediate action in the event of disease outbreaks, including a requirement to cull or withdraw fish from the ocean environment forthwith.
22. Egg Imports: Canada should not allow any further egg imports from foreign jurisdictions, or transfer between coasts.