

Risk and precaution: Salmon farming

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A B S T R A C T

The salmon farming industry uses coastal, temperate marine waters to culture salmon in flow-through net pens. As marine currents pass through salmon farms, pathogens are carried in both directions between two highly contrasting environments. When wild fish are infected with pathogens spilling from the farm environment, the natural mechanisms that work to prevent epizootics become inoperative. The 18-year decline of Canada's largest salmon fishery, on Fraser River Sockeye Salmon, triggered a comprehensive federal commission to determine the cause. Two of the recommendations from this commission call for removal of the salmon farms from the Discovery Islands of British Columbia (BC), a bottleneck in the Sockeye Salmon migration route, if the evidence indicates that the industry generates greater than minimal risk of serious harm to the Fraser River Sockeye Salmon. Risk is interpreted as a probability and 'minimal risk', in the context of the Precautionary Principle, as a cut-off level on the strength of the scientific evidence needed to justify precautionary measures. Here the available evidence of the risk caused by sea lice and viruses from salmon farms on wild salmon is considered. From this perspective, the evidence is unambiguous. Salmon farms in the region of the Discovery Islands generate greater than minimal risk of serious harm to Fraser River Sockeye Salmon. Furthermore, there is no evidence that the risk factors identified are specific to Fraser River Sockeye Salmon, as many of them apply to other areas and salmon species in the north eastern Pacific and globally.

1. Introduction

The threat to ecological systems posed by agricultural activity is significant [3]. The risk of pathogen transmission from farmed to wild salmon has been demonstrated [17, 45], and open-net sea-pen salmon culture is recognized as a coastal ecosystem modifier across trophic levels [18], epidemiologically linking vastly separated wild salmonid populations [44]. There is also a long history of large-scale, unforeseen, negative consequences due to accidental import of exotic pathogens [46]. It is the primary cause for disease emergence in wild fish [92], with potentially irreversible effects [90]. [26] reported reduced survival and abundance of wild salmonids for all populations exposed to salmon farms in North America and Europe as compared to both (i) unexposed populations in Alaska and the western Pacific and (ii) less-exposed regions within salmon farming countries.

Marine waters are an exceptionally efficient pathogen dispersion medium [85]. Thus pathogens may pose particularly severe risk to ocean biodiversity [63]. When an infective agent enters a farmed environment, it is released from critical limits to growth. If allowed to spill back into the wild environment, it can generate unnaturally elevated local pathogen levels [71]. Indeed, salmon farms have been described as 'pathogen culturing facilities' [4].

In addition to elevating local pathogen levels, feedlot-type environments promote an increase in virulence [22, 46]. Increased virulence of pathogens in farm salmon has been observed with viral haemorrhagic septicaemia virus VHSV [23], infectious salmon anemia virus, ISAV [73] and *Flavobacterium columnare* [81].

When assessing the threat posed by salmon farm-origin pathogens to wild fish, one must look beyond direct mortality, as subclinical infections can have unforeseen ecological consequences, e.g. reduced feeding success or weakened predator avoidance [90].

The salmon farming industry has imported 30 million Atlantic salmon eggs into BC from Norway, Scotland, Ireland, eastern Canada and the USA since 1985 [22]. The majority of salmon reared in net pens in BC are Atlantic salmon of the Norwegian Mowi strain [99].

1.1. Fraser river sockeye salmon (*Oncorhynchus nerka*) population decline

The 18-year, more than three-fold decline in productivity of the Fraser River Sockeye Salmon (number of adult Sockeye Salmon divided by the number of spawning adults in the parent generation) triggered the \$37 million federal Cohen Commission Inquiry (Fig. 1). While reduction in fishing ensured that a viable numbers of spawners entered

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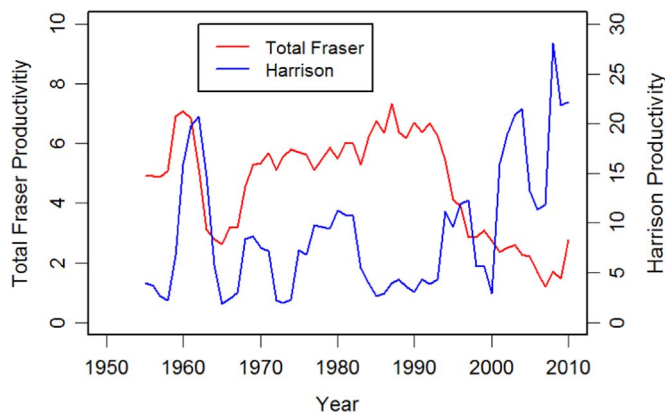


Fig. 1. Comparison of historic productivity between total Fraser River Sockeye Salmon and the Harrison River Sockeye Salmon component. This is a four-year moving average of total adult returns per spawner (not including the minor jacks component) divided by the total spawning adults in the parent generation 4 years before. Return year is the last year of the four used to produce the moving average. The horizontal dashed line indicates the productivity at which the population can replace itself without any fishing pressure, i.e. returns/spawner=1. (Data Courtesy of the Pacific Salmon Commission).

the river annually and freshwater survival from spawners to juveniles was high, the survival rate during the marine phase, from smolts to spawners, was very poor [78]. By contrast, the Harrison River component of this total demonstrates a surprising contrasting pattern of increasing productivity over roughly the same time period (Fig. 1).

There are two known marine migration routes for juvenile Fraser River Sockeye Salmon after they leave the river. The route used by most of these populations appears to be north along the eastern shore of Vancouver Island [95]. The DNA of the Harrison River Sockeye Salmon [95], however, has been identified only along the alternate route on the west side of Vancouver Island (Fig. 2). The two different migration routes represent contrasting exposure to farmed salmon. The group migrating along eastern Vancouver Island are exposed to a series of the heaviest concentrations of salmon farms in BC, while fish migrating along the southern route are largely unexposed.

1.2. Cohen commission of inquiry into the decline of the sockeye salmon in the fraser river

The Cohen Commission produced 75 recommendations [13] to reverse the decline of the Fraser River Sockeye Salmon. Two of these recommendations, 18 and 19, specify conditions for the removal of salmon farms from a specific region of the BC coast, called the Discovery Islands (Fig. 2). These recommendations are based on evaluation of the risk to Fraser River Sockeye Salmon posed by salmon farms sited in a bottleneck-type area on their migratory corridor.

Recommendation 18: If at any time between now and September 30, 2020, the minister of fisheries and oceans determines that net-pen salmon farms in the Discovery Islands [on a major juvenile migration route for Fraser River sockeye salmon] pose more than a minimal risk of serious harm to the health of migrating Fraser River sockeye salmon, he or she should promptly order that those salmon farms cease operations.

Recommendation 19: On September 30, 2020, the minister of fisheries and oceans should prohibit net-pen salmon farming in the Discovery Islands unless he or she is satisfied that such farms pose at most a minimal risk of serious harm to the health of migrating Fraser River sockeye salmon.

The risks associated with the salmon farming industry to be evaluated for the Discovery Islands region are threefold; (i) risk of introduction of exotic pathogens, (ii) risk of amplification of exotic or endemic pathogens and parasites, and (iii) risk of pathogen mutation to higher levels of virulence.

The Cohen Commission recommendations offer remedy to a global

societal issue – how to manage risk when common resources and private industry collide. Below is a framework for assessing risk portrayed by the scientific literature on the impact of salmon farms on Fraser River Sockeye Salmon in the region of concern, the Discovery Islands.

1.3. Minimal risk

The invocation of Recommendation 18 requires evidence of “more than minimal risk of serious harm”. Recommendation 19 reverses the burden of proof and recommends a date for a specific action unless evidence is produced that can demonstrate that the risk of serious harm is indeed minimal.

There are two questions related to these recommendations:

- Is there currently sufficient evidence to invoke Recommendation 18?
- What sort of evidence would be needed by September 30, 2020 to nullify Recommendation 19?

Step one is to assess formal statements of ‘risk’ to determine an appropriate interpretation of ‘minimal risk’ and then survey currently available evidence associated with this risk and assess its strength in light of this interpretation.

In order for a ‘risk’ to be judged as minimal, the only interpretation of several provided by either the Oxford English Dictionary (www.oed.com accessed July 9, 2016) or Merriam-Webster’s Dictionary (<http://www.merriam-webster.com> accessed July 9, 2016) is as a probability – in this context as the probability of serious harm to wild Pacific salmon. Probability is also the only technical definition of ‘risk’ reported by [9] to be in common, non-technical usage and so ‘risk’ as a probability.

The question then becomes: How large must a probability become before it is judged as greater than ‘minimal’? In this case, Justice [14] used, in his words, “the precautionary principle to guide [his] consideration of the appropriate response to the risks that salmon farms pose to the future sustainability of Fraser River sockeye.” This principle is used to guide the appraisal of whether the risk of serious harm to the Fraser River Sockeye Salmon is minimal.

By the strictest definition, ‘minimal’ means as small as possible. However, this interpretation must be dismissed because, if there is any uncertainty whatsoever, the only way to achieve minimal risk of serious harm would be to routinely ban any human activity that might conceivably cause harm. As critics of the Precautionary Principle have pointed out (e.g., [91,96], such a rigorous interpretation would rule out innovation of any sort, and would even stifle discovery [40]. Therefore it is more reasonable to use the alternative interpretation of ‘minimal’ as either very small or negligible.

The key question in assessing the evidence associated with the Commission’s Recommendations 18 and 19 then becomes: Is the probability of serious harm more than negligible? Experience informs us of two inherent difficulties in answering such a question. First, the nature of the uncertainties is typically so profound that the probability is incalculable [38], e.g., argue against using methods of formal risk analysis to estimate probability of serious harm, pointing to the common theme of unanticipated surprises, such as the role of CFC’s as catalysts in the destruction of stratospheric ozone. Such unidentified factors cannot be incorporated in any rigorous way into a calculation of the probability of serious harm. They promote the adoption of an attitude of humility and vigilance in the presence of such ‘ignorance’ of the often-complex nature of the underlying dynamics.

With a formal calculation of such probabilities off the table, one is left with a qualitative assessment of the viability of the evidence pointing to the potential for serious harm. The primary question then becomes: Does the viability of the available evidence exceed some appropriate minimal threshold above which a reasonable person might view the risk of serious or irreversible harm as greater than minimal? This is indeed the key question in many similar instances [31].



Fig. 2. Map of southern half of British Columbia showing the Fraser River, juvenile Sockeye Salmon migration routes and salmon farm locations. While the majority of Fraser River Sockeye Salmon migrate north out of the river between Vancouver Island and the mainland of British Columbia, the Harrison River Sockeye Salmon appear to migrate around the southern tip of Vancouver Island [95].

Formal statements of the Precautionary Principle and related Precautionary Approach focus on this general issue. Yet they provide limited guidance in how it should be handled. The Rio Declaration (1992, Principle 15) contains the following statement: “In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty [emphasis added] shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.” The phrase, “lack of full scientific certainty,” can be taken to imply, e.g., by the Canadian Chamber of Commerce (<http://www.chamber.ca/download.aspx?t=0&pid=45c1b24c-9bae-e211-8bd8-000c291b8abf>, accessed July 9, 2016), “that there is still a need for sufficient scientific data to establish that a plausible threat exists for the possibility of serious or irreversible harm.”

Further, limited guidance can be found in a [24] communication:

“It [the precautionary principle] covers cases where scientific evidence is insufficient, inconclusive or uncertain and **preliminary scientific evaluation indicates that there are reasonable grounds for concern** [emphasis added] that the potentially dangerous effects on the environment, human, animal or plant health may be inconsistent with the high level of protection chosen by the EU.”

The issue under review in this paper, while global in nature, takes place in Canada. Hence, a Canadian government policy paper [1] on implementing precautionary measures provides critical guidance, and suggests a somewhat more substantial evidentiary base:

“In determining what constitutes a sufficiently sound or credible scientific basis, the emphasis should be on providing a sound and credible case that a risk of serious or irreversible harm

exists. 'Sufficiently sound' or credible scientific basis should be interpreted as **a body of scientific information – whether empirical or theoretical – that can establish reasonable evidence of a theory's validity**, [emphasis added] including its uncertainties and that indicates the potential for such a risk."

Others, e.g., [91,96], also stress the importance of taking into account the relative cost of proposed precautionary measures vs. the associated losses due to the perceived threat – arguing that decision theory dictates that the lower this cost, the lower the minimum threshold for the supporting evidence need be. Justice Cohen's Recommendation 14 on licensing restrictions provides an example.

Recommendation 14: *Beginning immediately and continuing until at least September 30, 2020, the Department of Fisheries and Oceans should ensure that .*

- *the maximum duration of any licence issued under the Pacific Aquaculture Regulations for a net-pen salmon farm in the Discovery Islands (fish health subzone 3-2) does not exceed one year;*
- *DFO does not issue new licenses for net-pen salmon farms in the Discovery Islands (fish health sub-zone 3-2); and*
- *DFO does not permit increases in production at any existing net-pen salmon farm in the Discovery Islands (fish health sub-zone 3-2).*

Note that, in contrast to Recommendations 18 and 19, there is a lack of requirement for any further evidence. The evidence in hand at that time, though not deemed sufficient for calling for immediate removal of farms, apparently sufficed for restricting the duration of licenses to at most one year with no opportunities for expansion. The reduced requirements for evidence associated with these less costly precautionary measures is consistent with the above, decision-theoretic consideration.

What constitutes sufficient evidence of risk of serious harm to invoke the more severe provisions of Recommendations 18 and 19? The following ordered list of key types of evidence provides a more substantive context for this question. Items are arranged from least to increasingly definitive. Items 0 and 5 establish extreme upper and lower bounds.

1. Vague anxiety over the potential for serious or irreversible damage.
2. Evidence from other, similar instances of such damage having occurred.
3. Early warning signs of specific threats, including international experience similar to those observed in such instances.
4. Correlational or epidemiological evidence of potential substantial impacts on the entity of concern.
5. Evidence from controlled experiments demonstrating a potential causal mechanism for such impacts.
6. The occurrence of serious or irreversible damage with clear evidence of the causal mechanism.

2. Scientific evidence on farm salmon risk to fraser river sockeye salmon in the discovery islands

2.1. Sea lice

While sea lice are a naturally occurring salmon parasite, salmon farms provide anomalous, inshore, over-wintering host populations. As such, their presence degrades the allopatric barrier that otherwise has served to protect wild salmon from sea lice infection in the fish's most juvenile marine phase [15]. Sea louse infection reduces survival of juvenile Pacific salmon [67], and the smaller the fish, the greater its susceptibility to sea lice damage [27].

The impact of sea louse infection extends beyond direct lethal

impact. Infected juvenile salmon occupy high-risk, peripheral positions within the school, increasing the risk of mortality as a preferential target for predators [54].

Sea louse infection rates rise as juvenile Sockeye Salmon migrate past the salmon farms in the Discovery Islands [69] to an order of magnitude greater than juvenile Sockeye Salmon unexposed to salmon farms [79]. Furthermore, heavy sea lice infestations occur on Sockeye Salmon captured in the effluent plume, carrying live sea lice, from a farmed salmon processing plant in the Discovery Islands [80]. Early marine growth is an important determinant of lifetime survival for salmon [6], and hence it is a significant concern that sea-louse-infected juvenile Fraser River Sockeye Salmon were 20% less successful at consuming food, than lightly infected fish [37]. As well, at least one aquaculture-related virus, ISAV, is transmitted between salmon hosts by the salmon louse (*Lepeophtheirus salmonis*) [72].

Removal of farmed salmon from wild salmon migratory waters reduces sea louse infection of juvenile wild Pacific salmon [68] with a corresponding increase in survival [7]. While chemical treatments aimed at reducing sea lice abundance on the farms can achieve similar effects [77], there is growing evidence that this a temporary solution as drug resistance evolves in sea lice [5,57,98]. There is reporting that drug resistance may exist among sea lice in BC [84].

Release of larval sea lice (*Lepeophtheirus salmonis*) from salmon farms is a known population-scale risk of serious harm to wild salmon including Pink Salmon (*O. gorbuscha*) and the much larger juvenile Atlantic Salmon and Sea Trout, (*Salmo trutta*) [55,90].

3. Viruses

3.1. Infectious haematopoietic necrosis virus, IHNV

IHNV is a rhabdovirus. Endemic to the Pacific Northwest, it has spread to Asia and Europe through movement of infected salmon eggs (OIE, 2009). IHNV is an internationally notifiable disease as per the World Organisation for Animal Health [75].

The IHN virus was first reported in BC Sockeye Salmon over 50 years ago, but was considered a freshwater disease [83]. Detection of IHNV in seawater-phase Sockeye Salmon is uncommon [94]. The virus is known to have a devastating impact on Sockeye Salmon [70], with greatest susceptibility occurring in juvenile salmon [100], OIE, 2009) where 50 – 95% mortality can occur [56,93].

Atlantic Salmon are acutely vulnerable to IHN and once infected, will carry higher IHN virus titres than Sockeye Salmon [59], which suggests Atlantic Salmon shed higher concentrations of IHNV than wild salmon. There have been three IHN outbreaks reported in the BC salmon farming industry [84,88] in 1992-6, 2001-3, and 2012.

The 1992–1996 epizootic occurred in the Discovery Islands where it spread to 14 farms within an 11 km radius. The genetic homogeneity of the virus was specific to the farmed, but not the adjacent wild fish, and the outbreak ended abruptly with the onset of an area management plan suggesting that farm-to-farm transmission, not repeat infection from wild fish, sustained this outbreak [98].

The 2001–2003 epizootic also began with a single farm in the Discovery Islands. Genetic sequencing was used to trace the virus's spread to 32 salmon farms across the southern half of the BC coastline, including 81% of the farms in the Discovery Islands [84]. In addition to farm-to-farm transmission, juvenile Atlantic Salmon became infected as they were transported through the Discovery Islands aboard a vessel pumping raw seawater through the fish holding tanks. Farms along the route of this vessel, off Port Hardy (Fig. 1), became infected, as did the receiving farms on the Central Coast and in the Broughton Archipelago, where the virus resumed its farm-to-farm spread. Once the vessel adopted a 'water-off' protocol as it passed infected farms and a farm salmon processing plant in the Discovery Islands, no further outbreaks occurred in newly introduced smolts [84].

[30] reported that vaccinating farm salmon for IHN reduces

downstream farm infection by only 31%, and that a vaccinated site will contaminate the entire width of Discovery Island waterways for over 30 km.

It seems apparent that IHNV is exceptionally contagious, that Fraser River Sockeye Salmon are at times migrating through narrow passages infused with the virus as it is shed from Atlantic Salmon farms and processing plants, and that this exposure occurs during the Sockeye Salmon's most susceptible marine lifestage. The evidence suggests that farm-origin IHNV presents a greater than minimal risk of serious harm to Fraser River Sockeye Salmon.

3.2. Salmon leukemia virus (SLV)

In the early 1990 s, SLV was identified as the causative agent for the disease, plasmacytoid leukemia (PL), discovered in farmed Chinook Salmon (*O. tshawytscha*) in the Discovery Islands [21]. PL was diagnosed in 96% of the salmon farms sited in the Discovery Islands and the Broughton Archipelago where it caused 80–100% mortality in these Chinook Salmon freshly recruited from wild stocks to populate the salmon farms [89]. One hundred percent of Sockeye Salmon exposed to SLV-infected farmed Chinook Salmon became infected [47].

[48] report that the apparent northward spread of the virus from Discovery Islands salmon farms to Broughton Archipelago farms was due to farm stocking with infected fish. The salmon farming industry responded to this devastating disease by switching largely to Atlantic Salmon; however, some Chinook Salmon farms persisted in the Discovery Islands through 2007 (Cohen Commission Exhibit #CC1001187). The following year (2008) was the first year that outmigrating Fraser River Sockeye Salmon were not exposed to Chinook Salmon farms since the early 1990s. Perhaps coincidentally, the cohort of Fraser River Sockeye Salmon that migrated to sea in 2008, returned in 2010 in record historic numbers [11], reversing the 18-year trend that began at the onset of Chinook Salmon farming in the Discovery Islands. Chinook Salmon farming continues in Clayoquot Sound.

[89] provide a diagnostic definition for PL, which includes “*hyperplasia of the interstitial cells of the caudal kidney*.” This lesion is reported by the Province of BC Animal Health Center, “*Interstitial (hematopoietic) cell hyperplasia (kidney)*” with reference to “marine anemia [plasmacytoid leukemia]” in farmed Atlantic and Chinook Salmon in every quarterly farm salmon health audit from 2006 to mid-2009 (the entirety of the dataset) (Cohen Exhibit #1549). No genetic sequencing of SLV exists; therefore, its impact on Fraser River Sockeye Salmon today, along with its current status in BC farmed salmon, is unknown.

A contagious, oncogenic virus [48], SLV causes high mortality in at least one Pacific salmon species, it is highly transmissible to Sockeye Salmon, and an epizootic caused by this virus is correlated with the onset of the decline of the Fraser River Sockeye Salmon. It therefore poses greater than minimal risk of serious harm to Fraser River Sockeye Salmon. The evidence below further elucidates the potential scope of the impact of this virus.

3.3. Mortality related signature

A significant contribution to the Fraser River Sockeye Salmon collapse is the 40–90% in-river prespawm mortality [43,65] that was first detected in 1995 [39]. After intense effort to discover the cause of this mortality, a distinctive genomic profile, named ‘the mortality related signature’, was identified exclusively in the Fraser River Sockeye Salmon that die in the river before spawning [65]. Sockeye Salmon captured in the marine approaches to the Fraser River that carry this genomic signature have a 13.5-fold greater chance of dying before spawning than other salmon; once the fish are in the river, this mortality increases to 50% [65].

Among the genes up-regulated in this genomic profile, are genes

linked to viral activity and leukemia [65]. This raises the question: Are Fraser River Sockeye Salmon dying of the virus named the Salmon Leukemia Virus? The lack of genetic sequencing of SLV and the discontinuance of research on the mortality related signature [82] leaves this weighty question unanswered.

3.4. Piscine reovirus (PRV)

Piscine reovirus belongs to the *Reoviridae* family, and evidence continues to build that it is the causative agent of heart and skeletal muscle inflammation (HSMI) in both Norway [29,32,76] and Chile [36]. HSMI has never been detected in the absence of PRV [29], and PRV titre is directly correlated with the severity of HSMI lesions [28]; however, PRV does occur in fish without HSMI [76]. PRV-infected farmed salmon do not exhibit HSMI until 5–9 months after marine transfer [51], and have been shown to recover [58].

[34] reported on a strain of PRV in BC that diverged from a Norwegian strain in ~2007. [87] provided evidence on a different strain of PRV in BC, and suggest that it is endemic to BC. Eighty percent of BC farmed salmon are infected with PRV [61].

HSMI is a highly infectious disease that has swept through the Norwegian salmon farming industry over long distances [53], causing significant economic impact [28]. HSMI has been identified in PRV-infected farmed Atlantic Salmon and in one Pacific salmon species, Coho Salmon (*O. kisutch*) in Chile [37]. HSMI is reported as the 3rd main cause of reduced farmed Atlantic salmon survival by Marine Harvest globally [60]. While [62] reported no evidence of HSMI in BC, the BC Animal Health Center reported the pattern of heart inflammation consistent with HSMI in BC Atlantic Salmon in 2008 (Final Report AHC Case: 08-3362). Further evidence of HSMI in PRV-infected farmed Atlantic Salmon in BC is emerging [10].

HSMI lesions are a form of severe heart damage reducing cardiovascular capacity [25], causing near 100% morbidity in farm salmon populations [52]. HSMI has not been identified in wild salmon, perhaps in part because the opportunity to sample moribund wild salmon is greatly reduced by salmon predators [8,64]. Indeed, [66] report that the survival rate for Fraser River Sockeye Salmon returning to the Chilko River was significantly reduced in PRV-infected fish ($p=0.014$), with the estimated 10–20 day survival rate for PRV-infected fish 2–3 times lower than in uninfected co-migrants.

[33] reported that PRV transmits from Atlantic Salmon to cohabiting Sockeye Salmon.

HSMI is only one outcome recorded for PRV infection. PRV also causes erythrocyte inclusions in red blood cells with potential fish health implications [29]). To date, there has been no reporting on erythrocyte inclusions in PRV-positive Pacific or Atlantic salmon in BC.

There exists uncontested evidence that PRV is present and widespread in farmed and wild salmonids in BC, that it can transmit to Sockeye Salmon, and that it is the leading contender as the causal agent of HSMI, a disease causing severe impairment to the heart and skeletal muscles of salmon. There is also published evidence indicating (i) that at least one strain of the virus was introduced to BC from Norway in the last decade, (ii) that PRV can decrease the success rate for Sockeye Salmon attempting to return to the Upper Fraser River watershed, (iii) that HSMI occurs in BC, and (iv) that at least one species of Pacific salmon (Coho Salmon) can develop symptoms of HSMI. Therefore the abundant presence of the highly contagious PRV-infected Atlantic farmed salmon in net pens in the Discovery Islands presents greater than minimal risk of serious harm to Fraser River Sockeye Salmon.

3.5. Infectious salmon anemia virus (ISAV)

Infectious salmon anemia (ISA) is a salmon disease caused by the ISA virus (ISAV) of the *Orthomyxoviridae* family. This family also includes the human influenza virus [86].

As with IHNV, ISAV is an internationally notifiable fish virus. Two

major genotypes exist, North American (Western Atlantic Ocean) and European (Eastern Atlantic Ocean). The European ISAV genotype has now been detected in all major salmon farming regions worldwide. ISAV responds to the culture environment with elevated levels of virulence [12] through mutations that delete regions of the virus RNA [35]. When ISAV caused the most economically damaging epizootic in the history of the salmon farming industry, in 2007, in Chile, it was a highly virulent strain (HPR7b) that was traced to Norway [50,97]. ISAV remains an emerging fish pathogen because asymptomatic infections go undetected allowing opportunity for emergence of virulent epizootic strains [35].

Partial European ISAV genetic sequence has been reported in BC farmed and wild salmon [36]. Four other laboratories have provided supporting evidence of ISAV-positive PCR results in BC farmed and wild salmon [36]. This does not ‘confirm’ ISAV in BC as the regulatory-required tests cannot be satisfied without access to the moribund fish. When ISAV was reported in 2001 in Chilean farmed Coho Salmon [49], the region was not declared ISAV positive until the outbreak in 2007.

The discovery of segments of European ISAV strains in BC farmed Atlantic Salmon and Sockeye Salmon in the Fraser River warrants the conclusion that ISAV poses a greater than minimal risk of serious harm to Fraser River Sockeye Salmon.

4. Discussion

The multiple lines of evidence described above reasonably suggest that culturing large populations of salmon, both endemic and exotic, among wild salmon is significantly altering the naturally occurring virome and parasite communities. Farm-origin pathogens are a significant risk to wild salmon because, even as ocean currents link the farmed and wild ecosystems, each continues to operate on intrinsically contrasting rules. This prevents opportunity for host/pathogen equilibrium, which is critical to thriving populations. Predators contribute an essential service in the suppression of epizootics by removing disease-weakened wild fish. However, they have no access to the fish inside the pens. Hence, if the source of the epizootic is coming from the farm salmon, this disease suppression mechanism is disabled. Epidemiological linking of cultured and wild fish populations destabilizes natural host-pathogen equilibria creating a farmed/wild hybrid ecosystem rich with uncertainty and risk. Irreversible harm is anticipated when such ecosystem limits are exceeded [38].

Rearing exotic species in an open system invites introduction of exotic microbes. If not thoroughly avoided, these can cause serious, long-term damage. The evidence that ISAV and PRV have spread between widely separated regions of the world elevates the risk to Pacific salmon. Importation of Atlantic Salmon eggs into BC began in 1985, 25 years before PRV was identified [20,76]. In addition, Canada's *Fish Health Certificate* accompanying all live egg imports into the country does not request ISAV testing [19].

Furthermore, farm-amplified sea lice abundances are resulting in infestation levels found to reduce feeding ability in young Fraser River Sockeye Salmon. Reduced feeding threatens growth rates and thus juvenile salmon viability. Over a 20-year period, amplified IHNV levels have challenged generations of Fraser River Sockeye Salmon at the most susceptible marine phase of their life history. The risk of an ISAV epizootic in BC farmed salmon is supported by the lack of scientific evidence that this virus will exist indefinitely in a pre-epizootic state without generating virulent mutations. The evaluation of the risk posed by an ISAV outbreak in Fraser Sockeye Salmon includes evidence of a genomic influenza-type response to ISAV in Fraser River Sockeye Salmon tissue that was PCR-positive for ISAV (Cohen Commission Exhibit 2051).

And finally, evidence that the highly contagious piscine reovirus found in Fraser River Sockeye Salmon may reduce fitness suggests a substantive risk in consideration of the evidence that 80% of the Atlantic Salmon in pens are shedding this virus. Preliminary epide-

miological evidence that PRV infection reduces the spawning success rate for at least one Upper Fraser River Sockeye Salmon population, coupled with symptoms of HSMI in at least one species of Pacific salmon (Coho Salmon) elevates the risk associated with PRV alone to the second-highest level (four) in the ordered list in the introduction.

There is indeed a substantial body of scientific evidence providing early indicators of the risk from exotic Atlantic salmon pathogens to Fraser River sockeye salmon.

There are concerns in an opposing direction: Can the sum of the above lines of evidence even be classified as an early warning sign? Given what is known, can the situation be described as an early phase of assessing the risk of impact of salmon farms on the Fraser River Sockeye Salmon? Here are two examples: First, evidence pointing to the presence of ISAV in 100% of Cultus Lake Sockeye Salmon tested in 2004, a component of the Fraser River Sockeye population (Cohen Commission exhibit #2045). Cultus Lake Sockeye Salmon were listed as endangered in 2003 [16]. There has been no evidence to support or challenge this finding, until the [36] report of preliminary detection of European ISAV in Fraser River sockeye salmon.

Similarly, the scientific record on Fraser River Sockeye Salmon exposure to farm-amplification of the salmon leukemia virus did not generate a response by fishery managers to reduce this threat. Nor did the discovery of a genomic profile specific to dying Fraser River Sockeye Salmon of an immune system response to a virus with linkages to leukemia. Discovering the identity of the virus that may have caused this profile was not a priority for Fisheries and Oceans Canada, even as Canada invested \$37 million into the Cohen Commission of Inquiry into potential causes of the collapse of Canada's largest salmon fishery [74]. Nonetheless, with this genomic information, evidence of risk of serious harm from the salmon leukemia virus moved beyond theoretical. These are disturbing parallels to failures to respond to similar signs of risk in classic examples described in [38].

Continuation of the status quo could be justified only thorough refutation of most, if not all, of the multiple lines of evidence generated from multiple research groups. To ignore the weight of scientific evidence that salmon farms pose a greater than minimal risk of serious harm to Fraser River Sockeye Salmon risks repeating the events leading to the collapse of Northwest Atlantic cod populations in the late twentieth century [41,42].

Justice Bruce Cohen of the BC Supreme Court (now retired) recommended that salmon farms “cease operations” in the Discovery Islands as a precautionary measure to reduce risk of serious harm to Fraser River Sockeye Salmon. A recommendation requiring that all expansion of the salmon farming industry be in closed containment facilities has also been made to protect wild salmon in eastern Canada (Anon 2016). Adapting the salmon farming industry away from direct contact with wild fish, from open-net to closed facilities, is considered a solution that will be beneficial to the industry [2]. Adopting the precautionary approach “actively fosters innovation pathways that are more sustainable over the longer term” [38].

5. Conclusion

Through the lens of the Rio Declaration on Environment and Development, and subsequent, more prescriptive documents, it is clear that Canada should (i) invoke the Cohen Commission Recommendation 18, (ii) facilitate a transparent process to assess the evidence that the greater BC coast should be exempt from this precautionary measure and the companion Recommendation 19 to protect other populations of wild salmonids, and (iii) foster innovation in aquaculture systems that are epidemiologically isolated from wild fish.

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