WILD SALMON
FOREVER

The Risks of Open Net Pen Salmon Farms to Wild Pacific Salmon: Summary of Scientific Findings

A report prepared for Wild Salmon Forever by Lawrence M. Dill, PhD FRSC, Professor Emeritus, Simon Fraser University

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About the Author

Lawrence Dill obtained his PhD from UBC in 1972, returning to university after having spent 2 years as a DFO salmon biologist. He joined the Department of Biological Sciences at SFU in 1974 and rose through the ranks to become a University Research Professor. He was elected as a Fellow of The Royal Society of Canada in 1997 and was awarded the 2004 IgNoble Prize in Zoology for his landmark study of herring flatulence. Currently he is a Professor Emeritus at SFU and continues to supervise graduate students. During his career he has published nearly 170 scientific papers in refereed journals. In recent years much of his research and that of his students has been on the interactions between sea lice and wild salmon. He produced a research report for the Cohen Commission on the impacts of salmon farms on Fraser River sockeye salmon, and co-authored the WWF Salmon Aquaculture Dialogue report on sea lice. Never happy far from the water, he lives in a floating home in Victoria, BC with his wife Elizabeth and Digby, his Nova Scotia duck toller.

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Executive Summary

Concern about the potentially harmful interactions between wild Pacific salmon and farmed salmon contained in open net pens has been a longstanding issue in British Columbia and elsewhere. Here I review recent scientific findings relevant to this debate.

My principal findings are as follows:

1. Because of the large numbers of farmed Atlantic salmon in close proximity in open net pens (ONPs), lice, viral and other pathogen populations can grow to very large sizes, shedding millions of infective stages (lice) or copies into the local environment outside the farm, where they can infect wild fish. In addition, conditions inside the farms are exactly those which evolutionary theory predicts will lead to selection for increased pathogen virulence, i.e., an increased negative effect on its host, and there is evidence that this has happened in aquaculture facilities. Therefore, what comes out of ONPs can be much more dangerous to wild salmon than the pathogens that the wild salmon may have passed to the farmed Atlantic salmon in the first place.
2. The risk to wild salmon from sea lice produced in Open Net Pens (ONPs) is unambiguous and substantial. Lice have been shown to reduce productivity of both wild pink and coho salmon populations in the Broughton Archipelago, and there is no reason to think they are not having similar effects elsewhere on the BC coast. The mechanisms by which lice impact individual survival are well understood, and these individual and population level effects have been found consistently throughout the world and are supported by large-scale experiments.
3. Piscine orthoreovirus (PRV) and the disease it causes (Heart and Skeletal Muscle Inflammation or HSMI) have recently been confirmed on a BC salmon farm. The virus has been implicated in the heavy pre-spawning mortality of Fraser River sockeye salmon. Additionally, it has been shown that productivity of these stocks depends in part on the number of Atlantic salmon in the ONPs that the smolts pass on their northward migration to the open ocean. While we do not know what it is about the farms that underlies this latter relationship, pathogen transmission remains the most likely explanation. It is tempting to speculate that PRV may be involved but we don't yet know the source of the PRV with certainty.
4. A number of other viruses and disease-causing organisms (bacteria, myxozoans and microsporideans) are known to be present in ONPs The risk they present to wild Pacific salmon is currently unknown, but could be substantial. There is evidence that some can be passed to wild salmon with harmful effect, but we cannot say with certainty that any wild salmon population has declined because of them.
5. Lice (and to an extent, viruses) have been shown to affect the vulnerability of wild salmon to other mortality agents, including starvation and predation. Even if these pathogens do not kill the fish directly, infected fish are likely to be rapidly removed from the population by a predator, making the business of proving that

a given agent causes widespread wild salmon mortality and population decline a very difficult task.

6. As a result of these indirect effects, the impact of parasites and viruses on wild salmon depends on environmental factors such as water temperature and competition with other species. The less benign the environment, the greater the impact to be expected.
7. Apparently healthy fish in the ONPs may still be fighting infection and releasing viral particles into the waters surrounding the farm, where they can infect wild fish. Therefore the fact that only a small percentage of farmed salmon die of a given disease greatly underestimates the risk they present to wild salmon.
8. Lice impacts on wild salmon can be mitigated by appropriate control strategies on the farms, particularly the timing of parasiticide treatment. Although there is concern that lice may evolve resistance to SLICE and other chemicals used to control them, a large wild fish population may help to maintain the efficacy of SLICE and delay the evolution of resistance, meaning that the preservation of healthy wild salmon populations is in the salmon farmers' self interest.
9. The evidence of risk to wild salmon is sufficient that the precautionary principle should be invoked, and Governments should mandate and support the aquaculture industry's move from ONPs to land-based closed containment production systems.

Introduction

Most farmed salmon in BC are grown to market size in open net pens. At any one time there are approximately 80 active farms in BC (out of 119 tenures), each consisting of a number of separate net pens, containing up to ¾ of a million fish in total. Roughly 95% percent of the fish raised in BC are non-native Atlantic salmon (*Salmo salar*); a small minority of farms, all in Clayoquot Sound, contain endemic chinook salmon. The farms are distributed widely along the coast, mostly south of Port Hardy in the Inside Passage as well as along the west coast of Vancouver Island. This places them along the migratory routes of wild juvenile salmon heading to the open ocean as well as of the adult fish returning to their natal streams to spawn.

Since the Cohen Commission of Enquiry (2011) and its associated scientific reports, there has been a considerable amount of new research published on the risks that open net pen salmon farms (hereafter ONPs) may pose to wild Pacific salmon, especially the juveniles. The present report is an attempt to update and summarize our scientific understanding of these risks. Because I believe that research findings do not become accepted knowledge until published in the peer reviewed scientific literature, I will base this report only on such sources, and not on grey literature, unpublished research, anecdotes or opinion. Although the focus will be on research conducted on the interactions between farmed Atlantic salmon and wild Pacific salmon (*Oncorhynchus* species) here in BC, research conducted in Europe will be referred to where appropriate. To avoid long lists of citations I will refer to synthesis or review articles wherever possible. I will also indicate some areas where more research is warranted.

The risk of ONP's comes entirely from the fact that they are "open" and form a single interacting system with the surrounding waters and their wild salmon inhabitants. As a result, anything infecting the salmon outside the pens can be transmitted to the fish inside, and vice versa. Both parts of this two way street are important, but we are concerned here with risks to wild salmon coming from inside the farms. Parasites and diseases, albeit sometimes introduced by wild fish, change in abundance and perhaps virulence in the ONP environment and can then be transferred back to the wild populations, sometimes at earlier and more vulnerable life stages. The likelihood of this occurring is likely to be increased by wild juveniles being attracted to the ONPs by excess food and
nighttime lighting. Unlike Las Vegas, what happens in net pens doesn't stay in net pens.

As implied above, parasites (lice) and diseases are the main potential threats to wild fish from salmon farms and will be the main focus of this report. I will deal with each separately before more briefly considering some other possible risks to wild salmon posed by ONPs.

Lice

There are two species of ectoparasitic lice commonly found in relatively large numbers on the Atlantic salmon in the farms: *Lepeophtheirus salmonis*, the salmon louse (hereafter Leps), and *Caligus clemensi*, the sea louse. Both are generally referred to as sea lice. A main difference between these two, apart from the fact that Leps is much larger, has to do with their host specificity: Leps is only able to complete its life cycle and produce eggs on salmonids, while *Caligus* is a host generalist and is commonly seen on herring (e.g. 60) and other species of fish. This has some important implications discussed below. Most of the research on lice has been conducted on Leps; little is known about the impacts of *Caligus* on their hosts. Despite the fact that *Caligus* is sometimes more abundant on farmed Atlantic salmon than is Leps, Government regulations mandating treatments at certain threshold louse infection levels deal only with the latter species.

Genetic analysis (85) has suggested that Leps from Europe and BC are not identical, but there is no indication that this is anything other than random variation, perhaps due to genetic drift, or that the two types are functionally distinct. In other words there is no reason to believe that the results from host-impact studies in Europe, where lice have been a huge problem for wild salmon and trout, are not just as applicable here in BC.

Despite earlier arguments in the scientific literature it is now undisputed that ONPs are the primary source of heavy Leps infestations on wild juvenile salmon, including on pink and chum salmon in the Broughton Archipelago where most of the BC field work has been conducted (45, 60, 33, 51) as well as on sea trout and salmon in Europe (76, 70). In addition, there is evidence that pink and chum salmon and Fraser River sockeye smolts pick up both Leps and *Caligus* as they pass ONPs on their way north through the Discovery Islands (68, 69). The only remaining contentious issue is what impact this has on wild salmon populations; the evidence, to be discussed below, suggests it may be considerable.

Viruses

Sea lice are relatively large and obvious, easily observed and counted, and can even be cultured in the laboratory. This is one reason that they have been extensively studied. Most other salmon pathogens are invisible to the naked eye, and so have largely flown under the radar. However, recently, and aided by new molecular methods, much more attention has been paid to understanding the potential risk that viruses and microparasites pose to wild fish in BC and elsewhere.

Piscine orthoreovirus: Among the viruses, much of the current concern has focused on Piscine orthoreovirus (PRV). It has long been suspected (e.g., 65, 17) to cause a disease known as Heart and Skeletal Muscle Inflammation (HSMI), and this has very recently been confirmed experimentally (83). One reason it has taken so long to identify PRV as the causative agent of HSMI is that the virus can be present without causing any obvious signs of disease (84, 23). Another has to do with disagreement over whether clinical behavioural indicators of disease must be present before HSMI can be diagnosed. The virus first appears in the fish's red blood cells where it replicates before spreading to other organs and causing the lesions associated with HSMI (18).

PRV is ubiquitous in farmed Atlantic salmon in Norway and has been shown to transfer to wild Atlantics there (22) and PRV or PRV-like viruses (there may be a diversity of "species") have been found in coho salmon in both Chile (24) and Japan (74) and in hatchery rainbow trout in Norway (64). PRV has also been isolated from wild cutthroat trout, and from steelhead, coho, chinook and chum salmon in BC (38, 71). It can be passed from fish to fish by cohabitation (41).

HSMI has been found on at least one open net salmon farm in BC (14; see also 38, 71). The presence of some sort of virus in ocean caught adult Fraser sockeye is a predictor of very low survival to spawning (they have a 13.5-fold greater chance of dying en route; 55), and PRV is one of the pathogens that seem to correlate with pre-spawn mortality (56). The latter study (56) was the first record of PRV in sockeye salmon, and it was subsequently reported in sockeye smolts (25). While it is tempting to suggest these fish picked up the virus when passing salmon farms, either as smolts or adults, there is no evidence to confirm or refute this hypothesis at this time. However, the PRV found in BC is genetically very similar to Norwegian strains, and may have diverged from it as recently as 2007 (38), suggestive of farm origin, at least initially.

There are good reasons why PRV may compromise a fish's ability to complete the arduous migration to the spawning grounds. The high proportion of red blood cells infected in the early stages of HSMI is likely to reduce their oxygen carrying capacity and result in anemia and poor swimming performance; the subsequent lesions in heart and muscle tissue undoubtedly also make the salmon less likely to complete their migration successfully. Finally, Atlantic salmon infected with PRV have reduced tolerance for high temperatures (48). Should this be true for Fraser sockeye salmon, it could also help to explain why PRV seems to be associated with low survival, given the unusually high temperatures in the river in recent years.

The findings that many (perhaps even a majority) of apparently healthy farmed fish may be infected with PRV and in a disease state, i.e., actively mounting a cellular defense to the virus (14), have exceedingly important implications. If this is generally true then these fish are most likely shedding millions of viral particles in their faeces, or through their gills and skin, into the ONPs and the water surrounding them, potentially putting wild fish at risk. Therefore the fact that only a small percentage of farmed salmon die of a given disease greatly underestimates the risk they present to wild salmon.

***Other viruses*:** Concerns have been raised that three other viruses may pose a risk to wild salmon: infectious salmon anemia virus (ISAV), infectious haematopoietic necrosis virus (IHNV), and salmon leukemia virus (SLV). A recent paper on risks of ONPs (58) summarizes the available information on each of these viruses and concludes that all of them (as well as PRV) pose " a greater than minimal risk of serious harm" to wild salmon in BC. SLV and IHN have certainly been responsible for disease outbreaks in BC farms, and (57) determined that a small percentage of wild migrating sockeye had IHNV, using a powerful new molecular technique able to detect an active viral disease state in fish that otherwise appear healthy. The evidence for ISA in BC is controversial but there is published evidence of a variant form of ISA being in both farmed Atlantic and wild Pacific salmon (39).

Another virus beginning to raise concern is ENV – erythrocytic necrosis virus, which is known to cause severe physiological disruption in chum salmon fry (49.). Herring is a major host for this virus (16, 28), which suggests the possibility that it could be introduced to ONPs by herring attracted there by feeding opportunities. Indeed, ENV has been found in farmed Atlantic salmon (57). The involvement of herring in the host-parasite dynamics, as is also the case with *Caligus* (see above), leads to the possibility of some deleterious food chain effects for wild salmon, i.e., reduced food availability.

Other Pathogens

In addition to sea lice and viruses, a number of other pathogens found in farmed fish may pose a risk to wild salmon.

Bacteria: Two bacterial diseases have the potential to impact wild salmon. The first, bacterial kidney disease (BKD) is caused by *Renibacterium salmoninarum*. It is relatively uncommon in Atlantic salmon in net pens (37) but very pathogenic to sockeye. The second, *Piscirickettsia salmonis* is a significant pathogen of fish in net pens, including Atlantics, chinook and coho, but has not been found in wild salmon to date (37).

Myxozoans: These tiny parasites, distantly related to jellyfish, have a two-host lifecycle involving an invertebrate. One species, *Parvicapsula minibicornis*, is found in both smolts and adults of sockeye salmon and heavy infection impedes the fish's ability to recover from exercise (81) and can cause mortality (37). It is considered to be of "high risk" to Fraser River sockeye (37).

Microsporideans: This is another group of microparasites, related to fungi. One species, *Loma salmonae*, a well-known aquaculture pathogen (37), reduces the probability of sockeye surviving to spawning (56).

While all of these other pathogens can on occasion be found in ONPs, and can pose a threat to wild salmon, there are no documented cases of disease transfer. It is unclear how one would demonstrate this, other than with large-scale manipulative experiments combined with genetic markers.

Interactions between Pathogens

Because they cause skin damage and impair the immune system, being infected with lice may increase the fish's susceptibility to other pathogens, including *Loma* (62) and ISAV (3). Infections by lice (or being in any disease state) may also be expected to increase susceptibility to adverse environmental conditions, such as the higher water temperatures associated with climate change. It is also noteworthy that co-infection (i.e., simultaneous infection by more than one pathogen) is one of the factors selecting for increased virulence (see the following section).

Finally there is some evidence that sea lice can act as a vector for bacteria (2) and viruses (30), transmitting these pathogens from fish to fish as the lice switch hosts, a not uncommon behaviour (10).

The Red Herring of Endemism

It is sometimes claimed that because a particular disease is already found in wild salmon (i.e., is endemic), its presence on farmed salmon is not a threat to the wild fish. This is not necessarily the case. Because of the large numbers of hosts in close proximity in ONPs, lice, viral and other pathogen populations can grow to very large sizes - a process called bioamplification - shedding millions of infective stages (lice) or copies into the local environment outside the farm, where they can infect wild fish. Additionally, conditions inside the farm are exactly those which evolutionary theory predicts will lead to selection for increased pathogen virulence, i.e., an increased negative effect on its host (36). Although evolutionary processes like this will take several generations, the generation time of these pathogens is short. In fact there is considerable evidence that evolutionary change has happened in aquaculture facilities: ISAV apparently mutated to a more virulent form in Norwegian net pens (53), as did the bacterium *Flavobacterium columnarae* (73). Of particular relevance here, Leps sampled from farms cause more skin damage to their hosts, and cause greater growth reduction, than do lice sampled from wild fish (78). Although evolutionary processes like this will take several generations, the generation time of these pathogens is short. The result is that what comes out of ONPs can be much more dangerous to wild salmon than the pathogens that the wild salmon passed to the farmed Atlantic salmon in the first place.

This is further exacerbated by the farms disrupting what has been called "migratory allopatry" (44), meaning that returning adult wild salmon that may be infected with sea lice or other pathogens do not interact directly with juveniles on their way to sea, because they are not in the same place at the same time. This prevents pathogens on the former from infecting the latter. However, placing ONPs on the migration route allows for the pathogens to find a readily available host population in the fall, and to retain and grow the pathogen population over the winter, providing a source of infection for juvenile fish

passing by the farms in the spring. The fact that these fish are small, and in the case of very young pink and chum salmon, without scales, means they're less able to cope with infection, making the problem worse.

Consequences of Infection for Individuals

Sea lice and diseases may in some cases kill their salmon hosts directly, through stress and physiological dysfunction (12, 76, 6). For example, skin damage caused by lice may lead to osmoregulatory failure. However, it is widely believed that they more frequently make their hosts more susceptible to other mortality agents, particularly starvation and predation.

Recent research suggests that heavy infections with *Caligus* can reduce the ability of juvenile sockeye salmon to compete for food and thus reduce their growth (25, 26). This is important because salmon biologists have known for a long time that smaller fish in a cohort have a much lower probability of survival to adult return (e.g. 5), perhaps due in part to being more likely to be eaten by predators (77).

Predators may have an even more direct effect on salmon infected with sea lice because the lice *per se* may make them more susceptible to predators, as has been shown for pink and chum salmon fry (47). The mechanism for this is not entirely clear but may involve compromised swimming ability (50, 63), less attentiveness to predators while concentrating on feeding (47), and/or altered schooling behaviour (47) or surface activity (82).

Very little research of this sort has been done on fish infected with other disease agents but having BKD makes chinook salmon more vulnerable to predators (54), and Rhinoceros auklets (a seabird) have more sockeye infected with the myxozoan *Parvicapsula* in their diets than would be expected based on the proportion such fish make up of the population (56). Also, Chilko sockeye smolts showing signs of viral infection (including IHNv) have a much lower chance of surviving downstream migration to the mouth of the Fraser River than do their uninfected counterparts (31), perhaps due to in-stream predation. The source of these infections in not known with certainty, though ONPs are certainly one possibility.

The implications of these findings are extremely important. If generally true it means that juvenile fish heavily infected with lice, or fighting off viral infection, may be quickly removed from the population, ending up either in the guts of predators or sinking to the sea floor. As a result, it will be most unlikely that sampling of wild fish populations will find many of them to be infected, as only the survivors will still be present, thereby greatly underestimating the impact of ONPs. It also means that laboratory studies in benign environments devoid of predators (e.g., 35) will greatly overestimate the threshold level of infection likely to cause death. Thus (35) found that 7.5 lice per gram in small juvenile pink salmon were necessary to cause death in the lab, yet found few of such fish in the field, implying that lice were not a major cause of mortality (34). The fallacy of this argument should be apparent. It was clearly articulated 20 years ago (52):

"In contrast to cage or tank situations, sick fish in the natural environment that show any abnormal behaviour are likely to be rapidly removed from the area by predators and any random samples of fish taken will almost inevitably show only healthy animals, those with non-pathogenic infection levels or those with benign types of disease". (McVicar 1997)

Population Consequences

Worldwide Picture

A global assessment (20) suggests that local native salmonids are impacted negatively wherever there are fish farms (see also 13). A particularly well-documented case study of the effect of sea lice has recently been provided for sea trout in Europe, based on many years of research in Ireland, Scotland and Norway (76). As well, Atlantic salmon returns to the Erriff River in Ireland are 50% lower in years following high lice levels on farms (70).

It should be noted that since it is possible (and perhaps even highly likely) that fish infected by lice may be co-infected with other pathogens, some of the negative effects attributed to lice may be due to bacteria or viruses, which are harder to detect and may not have even been assayed. This caveat applies equally to the Broughton Archipelago lice studies to be described next.

BC

Broughton Archipelago pink salmon: An argument raged in the literature for several years over whether Broughton pink salmon populations were being severely impacted by sea lice. Early predictions (45) that lice would cause local pink salmon extinction if downward populations trends continued proved untrue, but this was likely due in part to changes in louse management practices (timing of anti-louse treatment prior to the wild salmon migration window; 66). In and of itself, this would suggest an impact of lice on wild fish. One study by the Provincial pathologist (51) was unable to find an effect of farmed salmon louse levels on pink salmon survival, but more thorough and powerful analyses (46, 43) revealed a significant effect on recruitment. Worryingly, lice levels on wild salmon in the Broughton have recently increased; this may be due to a combination of warmer water and less well-timed treatment on the farms (4).

Coho salmon: There is evidence that Broughton Archipelago coho salmon populations are also negatively impacted by salmon farms (9). Like the pinks, coho probably pick up lice directly from the farms, but they also pick up lice indirectly when consuming parasitized pink salmon (8).

Chum salmon: Curiously, although chum salmon fry are often just as heavily parasitized by lice as are pink fry, their survival does not seem to be negatively affected to the same extent (67). It is believed that this may be due to predators concentrating their attention on the more preferred, and now vulnerable, pinks, thereby reducing predation pressure on the chum.

Fraser sockeye: An analysis conducted for the Cohen Commission, and subsequently published (11), suggested that the number of fish in the ONPs passed by migrating

sockeye smolts was a predictor of subsequent adult returns, i.e., more fish in the pens led to lower sockeye returns. But this was true only when competition with pink salmon in the open ocean was likely to be intense. Interestingly, this result is consistent with the above-mentioned finding that lice compromise sockeye competitive abilities (25).

Correlation, Causation and Experimentation

Studies such as that showing that heavily liced fish are less able competitors (25) can justly be criticized for assuming that the correlation implies causation. It may be that lower food intake compromises the fish's ability to avoid infection, or that inherently low quality fish are both competitively inferior and more vulnerable to lice. However, if a causal hypothesis based on a correlation leads to a prediction that can be confirmed by further observation, or if several correlations triangulate at the same cause from different angles, one can begin to have some confidence that the proposed causal mechanism is correct. This is especially true if the proposed cause aligns with known biological principles. Thus correlations provide important data in several fields, including epidemiology – and salmon epidemiology is essentially what we are dealing with here.

However, while it would be unwise to discount correlational evidence, a better way forward is through controlled experiments. This is not always possible, particularly at the individual level of analysis; it would require placing predetermined numbers of lice on randomly selected clean fish, and no one has devised a way to do this yet. But there are two kinds of experiments that have been conducted at the population level. The first is fallowing. Fallowing of farms during late winter and spring has been shown to reduce lice infection of sea trout in Ireland and increase their survival (21). A similar experiment was conducted in BC in 2003, when the ONPs along an entire migration corridor in the Broughton Archipelago were left fallow during the spring migration of wild fry. This resulted in an increase in adult returns the following year (59; see also 61) A problem with a study of this sort is the lack of replication, meaning that the improved survival in that year could have been due to some other factor favouring the fish, such as increased food availability in the ocean or reduced salinity lowering survival of the lice (33).

A far stronger experimental result has recently been reported. SLICE (emamectin benzoate) is used to rid farm salmon of sea lice. It has also been applied to batches of hatchery Atlantic salmon as a chemotherapeutic in the hope of reducing their likelihood of picking up lice when passing fish farms. A meta-analysis of 118 separate experimental releases of this sort leaves no doubt that it is effective in increasing survival (79; see also 72) and implicates ONP-origin lice as the cause of reduced survival in the absence of treatment. An interesting result of the analysis was that the impact of the anti-parasiticide, and by inference of lice, was stronger when the survival of the untreated control group was poorest. The treated salmon were 1.7 times more likely to survive as the untreated ones under such conditions. Like the analysis conducted on sockeye for the Cohen Commission (11) this suggests that the impact of ONPs may be greatest when other biotic and abiotic conditions are less favourable for wild salmon survival.

No similar experiments have been conducted with lice chemotherapeutics in BC and no such experiments have been conducted on bacteria and viruses. This would be a very worthwhile research project.

Experiments are difficult to conduct in large field systems with numerous uncontrollable variables, so researchers are sometimes forced to "experiment in silico" with mathematical models. Models describe the workings of a system to the extent it is currently understood, and allow manipulation of variables to see the consequences. They can focus attention on gaps in knowledge and the simulation results should be viewed as hypotheses for further testing; they can also suggest improved management practices. The extensive literature on salmon-sea louse epidemiological models has recently been reviewed (27). One of the outcomes was a greater realization of the importance of incorporating spatial structure, i.e., spacing and interactions between farms along a migration route.

Other Potentially Negative Impacts of ONPs

Escapes

The recent escape of something like 150,000 farmed Atlantic salmon from an aging ONP in Washington State, and their subsequent dispersal and capture far from the site, has cast the spotlight on another potential risk to wild salmon. It is known that farmed Atlantics can survive in the wild and may have established permanent populations in BC streams (80, 19). Because they are not closely related to Pacific salmon, there is very little likelihood of interbreeding and loss of genetic identity (with the possible exception of the Clayoquot Sound area where the farms raise chinook salmon, wild populations of which are found in local streams; 40). Rather, the risks come from their potential for competing with wild juvenile Pacific salmon and steelhead in streams, and possibly from disease transfer. Studies on the former suggest that while competition is possible it is unlikely to have severe consequences (summarized in 75). There has been no scientific study of disease transfer from escapees in BC, though it is known that some diseases can transfer from Atlantic to Pacific salmon sharing the same water, as could occur in streams (23, 41), and escaped Atlantic salmon are suspected of transmitting furunculosis (a bacterial disease) to wild salmon and trout in Norway (32).

ONPs have other negative consequences for the ecosystems that house them, including:

- attraction of wild forage fish (such as herring) and salmon and incidental harvesting of them ;
- pollution of the seafloor immediately below the pens with faeces and excess food;
- pollution from plastic debris (29), chemical agents (e.g. those used to clean nets; 7), diesel (spilled at a farm in the Broughton Archipelago in early 2017) and antibiotics;
- reduction of local crustacean populations as a result of SLICE spill-over;
- reduction of predator populations, including seals and sea lions, due to shooting.

Several of these are discussed in my report to the Cohen Commission (15), but are not treated in detail here because they are unlikely to have significant effects on wild salmon stocks comparable to the potential impacts of parasites and diseases.

Concluding Remarks

In my opinion the risk to wild salmon from sea lice produced in ONPs is unambiguous. Lice have been shown to reduce productivity of both wild pink and coho salmon populations in the Broughton Archipelago, and there is no reason to think they are not having similar effects elsewhere on the BC coast. The mechanisms by which lice impact individual survival are well understood, and these individual and population level effects have been found consistently throughout the world and are supported by large-scale experiments.

Experience in the Broughton Archipelago suggests that lice impacts on wild salmon can be mitigated by appropriate control strategies on the farms, particularly the timing of parasiticide treatment. However, there is concern that lice may evolve resistance to SLICE and other chemicals used to control them, as is happening elsewhere (1). Ironically, it seems that a large wild fish population may help to maintain the efficacy of SLICE and delay the evolution of resistance (42), meaning that the preservation of healthy wild salmon populations is in the salmon farmers' self interest!

PRV (and HSMI, the disease it causes) has been implicated in the heavy pre-spawning mortality of Fraser River sockeye salmon. Additionally, it has been shown that productivity of these stocks depends in part on the number of Atlantic salmon in the ONPs that the smolts pass on their northward migration to the open ocean. While we do not know what it is about the farms that underlies this latter relationship, pathogen transmission remains the most likely explanation. It is tempting to speculate that PRV may be involved but we don't yet know the source of the PRV with certainty.

The case is not so clear for other pathogens. While harmful pathogens – including viruses, bacteria, myxozoans and microsporideans - are certainly present in the ONPs, and there is evidence that some can be passed to wild salmon with harmful effect, we cannot say with certainty that any wild salmon population has declined because of them.

Research on these topics is badly needed, and indeed is ongoing, but in the meantime it seems that the evidence of risk to wild salmon is sufficient that the precautionary principle should be invoked, and Governments should mandate and support the aquaculture industry's move from ONPs to land-based closed containment production systems.

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