Critique of the Document

"***Information Regarding Concerns about Farmed Salmon - Wild Salmon Interactions***"
Presented to the Provincial Government of British Columbia by Gary Marty, D.V.M., Ph.D., Diplomate, A.C.V.P. of the British Columbia Ministry of Agriculture, Animal Health Centre, Abbotsford.

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Background

The document, "Information Regarding Concerns about Farmed Salmon - Wild Salmon Interactions," dated March 16, 2015, was presented to Ministers Thompson and Letnik of the Government of British Columbia (BC) with the intention of providing scientific information upon which to base management and policy decisions regarding wild and farmed salmon in British Columbia.

Collectively, we are a group of scientists, mostly academic, whose research expertise includes salmon and infectious diseases (here we refer to infectious diseases in the broadest sense as those that may arise from parasitic, viral or bacterial pathogens). All of us have worked specifically on the influence of salmon aquaculture on diseases of wild salmon in BC, and the associated consequences for the sustainability of both wild and farmed salmon. We have published more than 40 peer-reviewed articles on the interactions between farmed salmon and wild salmon in the primary scientific literature. More generally, we have over 150 years of combined research experience and have published more than 400 peer-reviewed articles in the primary literature in the fields of marine biology, fisheries science, invasion biology, epidemiology, and population biology.

While we acknowledge Dr. Gary Marty's impressive credentials as a fish pathologist, we have deep concerns that the document he presented to the Provincial Government incorrectly represents the current science on the ecology of disease interactions between wild and farmed salmon. In particular there are several errors of interpretation and a selective use of the literature that we believe lead to a biased conclusion that farmed salmon pose minimal disease risks to wild salmon in BC. A more complete and balanced assessment of the scientific literature reveals abundant evidence that salmon aquaculture does pose a disease risk to wild salmon, although there is ongoing debate about the extent of that risk. Furthermore, the literature reveals that salmon aquaculture can depress wild salmon populations under some circumstances. For this reason, government agencies, academic institutions, non-profit organizations, and industry groups are currently engaged in active research into disease-mediated interactions between farmed and wild salmon in British Columbia.

In responding to Dr. Marty's opinion, we make the following six points:

1. The effects of disease on the survival of salmon in the wild can be more severe than in captivity, particularly for juveniles.
2. There is uncertainty about the cause of death of most salmon that die on salmon farms.
3. Dr. Marty misrepresents or misinterprets published work that he uses to support his claims.
4. Dr. Marty omitted evidence that contradicts a number of his claims.
5. Dr. Marty failed to mention genetic tests that suggest infectious salmon anemia virus (ISAv) is present in BC.
6. Dr. Marty failed to consider emerging and evolving diseases that have the potential to impact wild salmon populations.

1. The effects of disease on the survival of salmon in the wild can be more severe than in captivity, particularly for juveniles.

It is illogical to draw conclusions about the effects of disease in wild salmon based solely on the effects of disease in farmed salmon. Farmed salmon do not have to migrate, avoid predators, or compete for scarce food, unlike their wild counterparts. Published scientific evidence shows that wild salmon do suffer direct mortality from disease, but disease also compromises their ability to grow, to compete and to avoid predators. Farm-amplified sea lice alone cause an average of 39% loss of wild salmon returning to rivers every year in Europe (1). These losses occur in the context of modern fish-health practices aimed to control parasites such that mortalities of *farmed* fish due to parasites almost never occur. Mortality of wild salmon in British Columbia due to sea lice from farmed salmon is estimated to have been even higher in some years (2, 3). Other infectious pathogens are also linked with increased mortality, predation by seabirds, and migration failure for sockeye salmon (4).

Placement of salmon farms along salmon migration routes can expose wild salmon to pathogens precisely when they are most vulnerable. Migrating juvenile salmon are

particularly susceptible to the effects of pathogens due to their small size (5), high natural mortality due to predation (6), stress from smoltification (7), and underdeveloped scales (8). For example, experimental evidence indicates that sea lice make juvenile pink and chum salmon more prone to predation (9) by reducing swimming ability (10) and increasing risk-taking behaviour (9). Chinook salmon experimentally infected with *Renibacterium salmoninarum*, the causative agent of bacterial kidney disease (BKD), were almost twice as likely to be eaten by larger fish than their uninfected counterparts (11). Field studies also suggest that parasite-mediated predation is important; Miller et al. found a higher diversity and load of microparasites in juvenile sockeye salmon predated by Rhinoceros Auklets than those sampled by trawl, suggesting selective predation by these sea birds on infected sockeye (4).

Pathogens can also indirectly affect wild salmon by reducing juvenile growth rates (12), which in turn can be an important determinant of marine survival and subsequent returns of adults (13, 14). Juvenile salmon face a trade-off between allocating resources to body growth and allocating resources to immune function. Scientific studies indicate that juvenile sockeye salmon on the east coast of Vancouver Island that are infected with sea lice have reduced competitive foraging ability relative to uninfected conspecifics (15). This result aligns with a published scientific study that suggests the survival of Fraser sockeye is poor when farmed salmon are abundant along the juveniles' migration route and the potential for competition with pink salmon is high (16).

2. There is uncertainty about the cause of death of most salmon that die on salmon farms.

Only a small proportion of mortalities on salmon farms are ever examined for disease. Dr. Marty writes that: *"Less than 1% of BC farmed Atlantic salmon die of diseases that might be infectious to wild Pacific salmon. Among the other 99% of farmed salmon, 90% survive and 9% die of other causes."*

Indeed, data provided during the Cohen Commission show that the average mortality rate based on "fresh silvers", or recently deceased fish that can provide valuable information on disease or other causes of death, is 1-5% (17, p. 7). However, total mortality has averaged 9-13 % per annum and has been as high as 30% in 2003 (17, p. 7). Of this mortality, just 20-25% are fresh silvers that are examined for bacterial and viral pathogens. Many more fish die in the pens of "other" (unknown) causes and are never examined (17, Fig. 4). Even for the fresh silvers that are examined, no cause of death is established for about 60-70% of the fish in the BC Ministry of Agriculture and Lands audits (17, Fig. 5). Therefore, the actual incidence of potentially transferable pathogens among farmed salmon could be much higher than Dr. Marty's stated 1%.

Dr. Marty also makes the assumption, without proof, that asymptomatic farmed salmon cannot shed pathogens that are harmful to wild fish. We caution that some of the papers Dr. Marty cites to make his case (e.g., 18) seem to be misrepresented (see point 3, below).

Transfer of pathogens from asymptomatic hosts is common in terrestrial systems (19), and there is no reason to believe it would not occur in the marine environment.

3. Dr. Marty misrepresents or misinterprets published work that he uses to support his claims.

On page 5, Dr. Marty states: *"… a recent scientific publication … reported no relation between farm fish production in the Discovery Islands and Fraser River sockeye salmon returns (20)."* Dr. Marty's interpretation of this publication is completely opposite to the actual findings of the study. Based on analyzing data from over 35 sockeye populations, Ruggerone and Connors (20) corroborated the findings of previous research (16) showing that there is a negative correlation between sockeye salmon survival and the number of farmed salmon that wild Fraser sockeye migrate past early in marine life.

On page 4, Dr. Marty says*: "Data presented during [the Cohen Commission] did not show that salmon farms were having a significant negative impact on Fraser River sockeye."* However, Dr. Marty fails to include the line that immediately follows this quote from the Cohen Commission report, "… the statistical power of the database (containing fish health data from 2004 to 2010) was too low to rule out significant negative impact" (21, p. 24). By not including this additional context, Dr. Marty leaves the impression that we can confidently conclude that salmon aquaculture does not pose a potential risk to Fraser River sockeye. Justice Cohen, however, concludes that "…net-pen salmon farming in the Discovery Islands poses a risk of serious harm to Fraser River sockeye through the transfer of diseases and pathogens" (21, p. 25).

On page 6 where Dr. Marty discusses potential farmed salmon impacts on wild salmon in Norway, he quotes Husa et al. (22), "[t]he good ecological conditions of the parameters studied in the fjord show little evidence of a regional impact from the fish farming industry despite the intensive production level." The cited study did not monitor wild salmon, let alone pathogen occurrence in wild salmon, but was focused on algal communities in the fjord, and thus has no direct bearing on the question of risk posed to wild salmon by farmed salmon.

Dr. Marty goes on to state on page 6*, "…nominal catches of wild Atlantic salmon have declined in nearly all jurisdictions over the past few decades. However, these declines are not greater in Norway than in jurisdictions without abundant salmon farms (23)."* He fails to acknowledge that the very publication he cites does not support his assertion that pathogens from farmed fish pose at most a minimal risk to adjacent wild salmon populations. Rather, the authors state in the abstract, "Salmon lice originating from farms negatively impact wild stocks of salmonids, although the extent of the impact is a matter of debate" (23).

Lastly, on page 11 where Dr. Marty claims there is evidence that Atlantic salmon are not asymptomatic carriers of disease, he states, *"…six scientific studies have been conducted in which Atlantic salmon sourced from commercial farmers were cohabited with various Pacific*

salmon species under controlled laboratory conditions (18, 24–28). The Pacific salmon never developed unexpected disease from the Atlantic salmon: evidence that the Atlantic salmon were not carrying an unknown disease of concern to Pacific salmon." However, one of these studies actually looked for transfer of disease from Pacific to Atlantic salmon, and clearly found it. And none of these studies were designed to monitor or quantify the extent of "unexpected disease" or asymptomatic presence of a broad range of pathogens that may affect wild and farmed salmon (e.g., 4). Although mass unexplained mortality of Pacific salmon was not observed in the studies Dr. Marty cites (at least those that have been published) this is not evidence that Atlantic salmon are not potential asymptomatic carriers of disease under some conditions.

These misrepresentations or misinterpretations show that the conclusions Dr. Marty reached are based upon a selective use of the published literature, thereby casting doubt on his conclusion that there exists minimal risk of disease spread from farmed salmon to wild salmon.

4. Dr. Marty omitted evidence that contradicts a number of his claims.

By ignoring a large body of research that contradicts many of his claims, Dr. Marty presents a biased and overly certain view of the risk posed by salmon aquaculture to wild salmon. Many studies indicate that salmon aquaculture is associated with elevated mortality of wild salmon. These studies include analyses of multiple stocks (e.g., 1, 29) and species (e.g., 30). As previously mentioned, sea lice alone have been shown experimentally to cause up to 39% mortality of wild salmon in Europe (1), while observational studies suggest this number can be much higher for Pacific salmon (2, 3, 31). Further data indicate that other bacterial and viral pathogens can also increase mortality of juvenile wild salmon (32–34). Although these studies did not link wild salmon mortality directly to salmon farming, there is potential for farmed salmon to amplify a diversity of pathogens.

A key citation used by Dr. Marty to support the statement that salmon farms do not affect wild salmon is his own work (35). Dr. Marty fails to mention that his analysis was not conclusive because it had low power to detect an effect (2). Indeed, a reanalysis of the data, published in the same scientific journal, revealed that productivity of both pink and coho salmon was negatively related to abundance of sea lice on salmon farms in the Broughton Archipelago (2). We recognize that there is continuing debate over the magnitude of the effect of salmon farming on wild salmon (23), and that the impact may vary among species of salmon, locations, and years (e.g., 36). However, by failing to acknowledge the published works that found parasites from salmon farms may negatively affect wild salmon populations (3, 29, 37–40), Dr. Marty presents a biased perspective on the current state of knowledge regarding the potential effects of salmon farming on wild salmon .

5. Dr. Marty failed to mention genetic tests that suggest infectious salmon anemia virus (ISAv) is present in BC.

While we recognize that numerous samples of Pacific salmon have not tested positive for ISAv (e.g., 41), several laboratories have conducted tests that indicate genetic elements of ISAv are present in Pacific salmon (42). Although this is not conclusive evidence that ISAv is present in BC, it is misleading to ignore these test results. The following test results were reported to the Cohen Commission of Inquiry (43, ch. 9):

- Over 60 samples from the endangered (44) Cultus Lake sockeye salmon population produced positive readings in tests conducted in a DFO laboratory, and
- 40 samples from the depressed (45) Rivers Inlet sockeye salmon population produced at least one positive reading in tests conducted at four laboratories despite widespread recognition that the samples were considerably degraded.

Dr. Marty contends that such results are all false positives because the World Organisation for Animal Health (OIE) requirement that the virus be isolated was not met. However, failure to provide definitive proof of presence does not constitute definitive proof of absence. Circumstantial evidence of the presence of the ISA virus in the North Pacific should not be readily dismissed as false positives. Indeed, in the Cohen Commission report, Justice Cohen concluded that, "… the evidence does not allow me to conclude whether ISAv or an ISAv-like virus currently exists in Fraser River sockeye" (46, p. 60).

The risk that ISAv poses to wild salmon is a combination of the probability that ISAv is present and the consequences if it is. Conditions in crowded net pens can select for more virulent strains of ISAv (47, 48), and the virus has already caused significant mortality of farmed Atlantic salmon in Europe (49), Chile (50) and eastern Canada (51). Perhaps of greater concern is that the virus has been shown to cause mortality in rainbow trout (52, 53) and coho salmon (54). With such potentially significant consequences, we believe that the precautionary principle should be applied, and surveillance of farmed and wild salmon for ISAv be expanded.

6. Dr. Marty failed to consider emerging and evolving diseases that have the potential to impact wild salmon populations.

Evidence continues to emerge of viruses associated with salmon aquaculture that pose a potential threat to wild salmon, and the potential for cumulative and interactive effects of multiple infections by different viruses (e.g., 55).

Piscine reovirus (PRV) is widely acknowledged as present and widespread in British Columbia – in trout as well as salmon (56, 57). There is strong evidence of an association between PRV and the disease, heart and skeletal muscle inflammation (HMSI; 58, 59), if not a direct cause-and-effect relationship (60). Dr. Marty's commentary on PRV fails to mention these papers. His dismissal of the potential for PRV to cause HSMI in wild Pacific salmon is based on the fact that HSMI has not been observed in wild salmon; however, it is important

to bear in mind that infected wild fish may not survive for long once they develop disease (61).

The piscine myocarditis virus (PMCV), associated with cardiomyopathy syndrome (CMS), also warrants attention. CMS was described first in wild Atlantic salmon in 2003 (62), and it has been subsequently shown that the most likely causative agent is PMCV (63). In addition, it is possible that PMCV is present in British Columbia waters; potential symptoms of CMS in British Columbian farmed salmon was documented in 2002 (64). Similarly, the threat of salmon alphavirus (SAV) on Pacific salmon and trout should not be taken lightly given evidence of impacts of this virus on rainbow trout (65).

Finally, in concluding that pathogens from salmon farming pose at most a minimal risk to wild salmon, Dr. Marty did not consider the potential for established pathogens to evolve. Parasites such as sea lice can evolve resistance to current treatments, as has occurred in Europe, Chile and eastern Canada (23, 66). More virulent strains of introduced or native viruses can multiply in fish farms (47, 48, 67), and these could potentially spread to wild populations. These evolutionary changes in pathogens are favoured by the domesticated environment of farmed fish and can produce epidemics that are more severe and more difficult to control.

Conclusion

We are not opposed to salmon aquaculture in principle, nor do we believe that salmon aquaculture is responsible for all the challenges faced by Pacific salmon populations in British Columbia. However, a complete and balanced examination of the available evidence leads us to conclude that the risks posed by aquaculture to wild salmon are non-negligible. This is precisely why there is extensive ongoing research by government agencies, academic institutions, non-profit organizations and industry groups into disease-mediated interactions between farmed and wild salmon.

We believe that in order to develop evidence-based policies that minimize the risk of disease to farmed and wild salmon, policy-making must be informed by science. We have deep concerns that the science advice offered by Dr. Gary Marty to Ministers Thompson and Letnik of the Government of BC is an incomplete and biased summary of the current scientific understanding of disease interactions between wild and farmed salmon. As we have shown, a more complete assessment of the science on the interactions between farmed and wild salmon indicates a higher risk than Dr. Marty communicated, as well as a higher degree of scientific debate on the extent of this risk.

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