Review

Pathogens from Salmon Aquaculture in Relation to Conservation of Wild Pacific Salmon in Canada: An Alternative Perspective

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Pathogens from Salmon Aquaculture in Relation to Conservation of Wild Pacific Salmon in Canada: An Alternative Perspective

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Abstract: Several articles over the past two decades have provided data, analyses, and interpretations that suggest significant impacts of farm salmon pathogens on wild Pacific salmon populations in British Columbia (BC), the westernmost province of Canada. Because disease is a normal part of all animal populations, there is always a potential for pathogen transfer between animal populations that interact. However, the evidence is weak for significant impacts of farm salmon pathogens on wild fish populations. We provide additional data and alternative interpretations of the available evidence to show that (i) many studies overestimate the risk of farm salmon pathogens and their diseases to wild salmon, (ii) these risks have not manifested as having significant impacts on wild salmon populations and, therefore, (iii) the evidence better supports the conclusion that farm salmon pathogens are having no more than minimal impact on wild salmon populations. Based on this information, we hypothesize that removing open net pen salmon farms will have no detectable effect on wild salmon population productivity in relation to reference salmon populations.

Keywords: salmon aquaculture; pathogens; risk to wild salmon

Introduction

The focus of our analysis is a recent review paper (Krkošek et al. 2024). We provide an alternate perspective for 49 of the 203 citations in this review, and we add data and 69 additional citations to support our perspective. Here we refer to Krkošek et al. (2024) as "*The Review*", and quotations from *The Review* are in "purple text." The following linked headers may be used to navigate our manuscript:

1. *The Review* omits data that support minimal cumulative impact of farm salmon diseases on wild salmon populations.
2. *The Review* omits independent assessments that conclude farm salmon are either (i) low risk to wild salmon or (ii) have no significant negative impact on wild salmon.
3. qPCR test results alone do not provide a reliable estimate of the risk or impact of disease on a population.
4. Basic principles of disease transmission support minimal impact of salmon farm diseases on wild salmon.
5. Analyses cited in *The Review* that link salmon farm diseases to wild salmon population impacts (i) do not reflect reality, (ii) are unreliable because many are based on test results that might be

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false positives, iii) are based on imaginary data that are contrary to the evidence, (iv) avoid contrary information, (v) did not include medical examinations to rule out other causes of mortality, (vi) result in a conclusion that is weak and has not withstood the test of time, or (vii) are correlative with weak evidence of causation. Also, *The Review* does not consistently follow best practices for reporting new molecular findings.

6. Many statements in *The Review* are contrary to information in the cited references, overstate information in the cited references, omit important information, omit contrary information, deceptively report cited references, or are not ecologically relevant.
7. Did competing interests minimize scrutiny on publications cited by *The Review*?
8. *The Review* is not an independent assessment of the impacts of BC's farmed salmon industry.

How we cite References and Notes: To avoid repetition, we refer to References and Notes already listed in *The Review* by using *The Review*'s number (e.g., "Reference 37"). Our Reference and Notes section is limited to information not already cited in *The Review*, with only one exception (Reference 39 = Meyers and Hickey 2022).

The Review summarizes ways in which current net pen aquaculture practices in British Columbia (BC), Canada, pose risks to populations of wild salmon. However, disease is a normal part of all populations, and a risk of pathogen transfer leading to disease occurs whenever populations interact.

A risk of disease transfer is almost meaningless unless it results in a consequence such as decreased population size in relation to reference populations. We base this assessment on our experience over the past four decades diagnosing and researching diseases of cultured and wild fish on the Pacific coast of North America (e.g., References 134, 169, and Bartholomew et al. 2017; Batts et al. 2017; Ferguson et al. 2011; Kent et al. 1998; Marty et al. 2010; Meyers 2005; Noakes et al. 2000; Soto et al. 2020).

BC has hosted marine net pen salmon farms for over 40 years. If salmon farm diseases were impacting wild salmon populations enough to justify the Canadian government's decision to ban salmon farms by 2029 (DFO 2024b), then impacts on wild salmon populations would be readily apparent. But impacts are not apparent.

[Main point #1] *The Review* omits data that support minimal cumulative impact of farm salmon diseases on wild salmon populations.

When wild salmon populations remain stable or increase in the presence of salmon farms, this is a strong indicator that regardless of the risk of farm-source pathogens, the cumulative impact for wild salmon is minimal. *The Review*'s Figure 1B shows declining commercial salmon harvests in BC since about 1990, stating that the decline is partly due to "low returns for commercially important stocks, including Fraser River sockeye (Figure 2)." However, the following information is omitted from *The Review*:

1. The estimated number of Fraser River **sockeye salmon** (*Oncorhynchus nerka*) spawning adults more than doubled during the 30 years that salmon farms were along the main juvenile outmigration route in BC (Table 1). Also, the estimated total run size was the greatest during the salmon farming years.

Table 1. Estimates of the summed total (millions) of Fraser River sockeye salmon spawners, catch, and total run size (includes Run Size Adjustment) for 30-year periods from 1900 – 2019 (PSC 2024). Salmon farms were active along the main juvenile migration route (Discovery Islands area) from 1988 – 2021 (shaded row); farms have since been removed from this area.

Years	Spawners	Catch	Total run size
1900 – 1929	42.9	175.9	218.8
1930 – 1959	31.9	109.0	141.0
1960 – 1989	45.1	152.2	200.0
1990 – 2019	97.5	134.9	257.4

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2. In **Clayoquot Sound** on the west coast of Vancouver Island, estimates of the numbers of wild **Chinook salmon** (*Oncorhynchus tshawytscha*) spawners in three key rivers generally decreased during the 1960s and 1970s, with variable recovery since several salmon farms were established in Clayoquot Sound in the 1980s and 1990s (Table 2). Estimates for the annual average number of spawners in all three rivers combined did not decrease from the first 30 years of records (before salmon farms, 1953 – 1982, 757 fish per year) to the most recent 30 years of records (with salmon farms, 1993 – 2022, 964 fish per year). Salmon farms remain active in Clayoquot Sound.

Table 2. Estimates of the average number of Chinook salmon spawners for key rivers in Clayoquot Sound (Canada 2024c). Decadal periods with active salmon farms are shaded.

	River			
Years	**Bedwell**	**Cypre**	**Megin**	**Sum**
1953 – 1962	450	410	1125	1985
1963 – 1972	30	50	79	159
1973 – 1982	18	34	74	126
1983 – 1992	187	22	39	248
1993 – 2002	317	88	313	718
2003 – 2012	92	639	52	783
2013 – 2022	347	1005	40	1392

3. In the **Broughton Archipelago** of BC, estimates of the numbers of wild **pink salmon** (*Oncorhynchus gorbuscha*) adult returns (spawners plus catch) before salmon farms (1960 – 1989) and with salmon farms (1990 – 2014) were within the range of estimated returns to a nearby reference area during the same time (Table 3). Average annual return estimates to the nine Broughton Archipelago rivers increased 14% from the 30 years before salmon farms to the first 25 years with salmon farms; this estimate is between similar estimates based on all 55 reference-area rivers (40% decline) and a 5-river subset of reference-area rivers (43% increase, Table 3).

   Presenting data from all 55 rivers and the 5-river subset identifies potential bias in the dataset – Available data from the 55 rivers changes from nearly complete during 1960 – 1989 (average = counts from 47 rivers per year) to less complete during 1990 – 2014 (average = counts from 35 rivers per year), with a decreasing trend within 1990 – 2014 (Table 3). The 24% decrease in the number of rivers with counts from 1960 – 1989 to 1990 – 2014 is nominally less than the 40% decrease in average annual returns over the two periods (Table 3). Therefore, at least some of the decrease might represent a true decrease in returns rather than just a decrease in efforts to count the returns. Data from the 5 selected rivers are nearly complete, but basing conclusions on these selected rivers risks biasing results away from decreasing returns.

Table 3. Estimates of the average number of annual pink salmon returns (spawners plus catch) for two regions of British Columbia. Salmon farming began in the Broughton Archipelago in 1987, but annual harvests did not approach 1,000 tonnes until 1990 (Reference 134). Shaded are counts and return estimates with active salmon farms during all years. The "# counted" refers to the average number of water bodies with adult salmon counts each year.

	Reference Region				**Broughton Archipelago**	
	55 rivers[a]		**5 rivers[b]**		**9 rivers[c]**	
Years	# counted	# returns	# counted	# returns	# counted	# returns
1960 – 1989	46.7	1,279,709	4.9	138,290	8.4	927,594
1990 – 2014	35.4	758,421	4.8	198,234	8.5	1,055,581
Change	-24%	-40%	-1.9%	43%	1.4%	14%
1990 – 2001	43.2	901,103	4.7	173,833	8.3	1,700,257
2002 – 2003	34.0	1,505,702	5.0	527,751	9.0	150,538
2004 – 2014	27.1	466,898	5.0	164,941	8.6	516,850

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[a]These are the same reference rivers used by Reference 34, with spawner data updated through 2014 from Canada (2024b) adjusted by exploitation [catch] estimates from English et al. (2018). [b]The five rivers in this column are a subset of the 55 rivers used by Reference 34; they are the only rivers among the 55 that have counts for all years 1999 – 2014. [c]These rivers were used by Reference 34 and 134, with data updated through 2014 from Canada (2024a) adjusted by exploitation [catch] estimates from Reference 134. Broughton Archipelago catch data are not publicly available for years 2010 – 2014, so values that include these years might be less than best estimates.

Assigning the date when sea lice infestation began is another source of potential bias – Regarding sea lice on wild pink salmon, References 21 and 72 classify 1990 – 2001 as pre-infestation, but Reference 134 and Morton and Volpe (2002) provide data that farm sea lice were abundant in 2000 when farm-source lice might have impacted the 2001 returns. This difference is relevant because pink salmon exposed to these lice in 2000 returned to the Broughton Archipelago as adults in 2001 in numbers that **were the second highest among records that date back to the early 1950s** (Reference 134). All other references that mention sea lice on BC farm salmon report that lice were common on farm salmon in the late 1980s (Hicks 1989) and early 1990s (Reference 169 and Brackett et al. 1991). Whether sea lice were common in the 1990s is relevant because compared to the pre-farm years 1960 – 1989, from 1990 – 2001 pink salmon returns were greater in the Broughton Archipelago compared with either reference area dataset (i.e., the 55-river dataset or the 5-river subset; Table 3).

Proposal to increase our understanding – To better understand the impact of assigning different years as the start of sea lice infestations, we recommend that the analysis published in References 21 and 72 be redone, with the 1990s Broughton Archipelago returns reclassified as "exposed" to farm-source sea lice. Multiple analyses could be done incrementally (e.g., analyze with infestation beginning in 1998, 1996, etc.). We hypothesize that lice-exposed strong returns in the 1990s would balance out the weak Broughton Archipelago returns in 2002 and 2003, when all references agree that (i) these pink salmon were exposed to abundant farm-source sea lice and (ii) pink salmon returns were less in the Broughton Archipelago than in either reference area dataset (i.e., the 55-river dataset or the 5-river subset; Table 3).

[Main point #2] *The Review* omits independent assessments that conclude farm salmon are either (i) low risk to wild salmon or (ii) have no significant negative impact on wild salmon.

1. **Cohen Commission** (Cohen 2012): In 2009, the Canadian government established the Cohen Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River. The 2012 final report (Volume 3, page 24) concluded (emphasis added), "Data presented during this Inquiry did not show that salmon farms were having a significant negative ***impact*** on Fraser River sockeye."
    a. Final Report Volume 2, page 164 – This conclusion was based, in part, on "a statistically significant declining trend in the number of high-risk diseases reported by salmon farms between 2003 and 2010."
    b. This is a good example of the difference between risk and impact, because Justice Cohen's assessment of the ***risk*** (Final Report Volume 3, page 22) was "I therefore conclude that the potential harm posed to Fraser River sockeye salmon from salmon farms is serious or irreversible." Here, the evidence did not support a conclusion that a ***potential*** risk had manifested as an ***actual*** impact.
    c. Qualifier – Justice Cohen also stated (Final Report Volume 3, page 113), "Although the data available to this Inquiry do not suggest that salmon farms are having a significant negative impact on Fraser River sockeye, I am not prepared to conclude, based on that data, that there is a low risk to sockeye from salmon farms. It is simply too early to reach that conclusion." Today, more than a decade of additional information since the Cohen Commission—including information in Table 1 and points 2, 3, and 4 below—supports a conclusion of low risk to sockeye salmon from salmon farms.

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2. **Pacific Northwest Fish Health Protection Committee** (Meyers 2017): "The ubiquitous nature of piscine orthoreovirus (PRV), its apparent historic presence in wild Pacific salmonid stocks in the Pacific Northwest and the lack of clear association with disease in Pacific salmonids suggest the virus poses a low risk to wild species of Pacific salmonids." This committee is an American organization of technical, scientific, and policy representatives from conservation agencies, Indigenous tribes, and commercial fish producers from the Pacific Northwest.
3. **Independent scientists in the USA states of Alaska and Washington**: From part of Reference 39 omitted by *The Review* (emphasis added), "The scientific information continues to support the conclusion that endemic PRV-1a in the PNW should be considered as posing a **low risk (not zero)** to Pacific salmon, thus requiring no significant changes to agency fish health policies."
4. **Independent scientists**: A 2012 paper reported that "recent decreases in abundance and productivity of Fraser River sockeye salmon occurred across a large geographic area" ranging from Washington to southeast Alaska (Peterman and Dorner 2012). Restated, this academic study found no differences between sockeye salmon populations regardless of whether they were exposed to salmon farms.

[Main point #3] qPCR test results alone do not provide a reliable estimate of the risk or impact of disease on a population (Meyers and Hickey 2022)**.** The progression of naturally occurring infectious diseases involves several steps, each of which is a subset of the previous step, from exposure → infection → lesions → disease → death of individual → population-level impacts

1. With rare exceptions, the progression does not skip any stage. The progression can end at any stage; for example, fish can be infected but never develop lesions, or fish with disease can recover and eliminate the pathogen.
2. qPCR tests can detect pathogen nucleic acid at any stage from exposure to death. However, qPCR tests also detect segments of nucleic acid that are not part of viable organisms or an active infection.
3. qPCR tests can detect transcriptional changes (e.g., gene expression converting DNA to mRNA) at any stage from pathogen infection to death. However, qPCR tests also detect changes that occur as part of normal life in the absence of infection or disease.
4. To demonstrate population-level impacts, the predictive value of information for making reliable estimates generally increases from early in this progression (e.g., exposure, as evidenced by PCR tests of gills with no other evidence of infection) to later in the progression (e.g., lesions as evidenced by ulcers on the skin or gills).

Also, information about one stage does not alone provide reliable information about prevalence at the next stage. For example, the worldwide estimate of 677 million human COVID-19 cases does not provide a reliable estimate of the number of deaths due to COVID-19, estimated at 6.9 million (1.0%) (Johns Hopkins University 2023).

Here is a specific example where progression from exposure to population impacts is relevant. *The Review* includes, "Recent studies on wild Pacific salmon have indicated that the spread of *T. maritimum* from salmon farms is a risk to infection and survival of wild fish (9, 19)."

Problem: For Reference 9, this estimate of risk to survival of wild salmon **is unreliable** because it is based solely on a less reliable indicator (i.e., qPCR+ test results) that is contrary to other available indicators like "no reported lesions" and "lack of population-level impacts" that are more reliable indicators of wild salmon survival.

Comment: Reference 9 includes qPCR test results for the bacterium *Tenacibaculum maritimum* in samples from juvenile Fraser River sockeye salmon that migrate past salmon farms in the Discovery Island region of BC. One mathematical model in Reference 9 estimates that "between 6.5% and 56.1% of sockeye smolts appear to die as a result of farm-origin *T. maritimum* exposure." This analysis has three limitations:

1. Reference 9 does not provide any evidence that the qPCR+ fish were infected with *T. maritimum*. *Tenacibaculum* was so named because it is sticky (Latin root *tenax*) and adheres to surfaces

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(Avendano-Herrera et al. 2006); therefore, testing of gills—as done by Reference 9—might detect environmental or commensal bacteria on the gill surface (i.e., **exposure**) that have **not infected** the gill tissues.

2. Reference 9 does not provide any evidence that their qPCR+ fish had lesions or disease associated with *T. maritimum*. *The Review* cites several studies that document external lesions on skin, fin, or gills that are characteristic of *T. maritimum* infection in Pacific salmonids (References 104 – 108), but Reference 9 **reports none of these lesions in their 2,270 sampled fish**.
3. Reference 9 does not provide any statistical evidence that their qPCR+ findings are associated with population-level impacts. Contrary to the Reference 9 estimate of increased mortality due to farm-source *T. maritimum*, total adult returns of Fraser River sockeye salmon during the salmon farming years 1990 – 2019 were greater than every other successive 30-year period back to 1900 (Table 1).

[Main point #4] Basic principles of disease transmission support minimal impact of salmon farm diseases on wild salmon.

The spread of infectious disease has two basic components, both of which are enhanced within salmon farms but reduced for wild salmon:

1. How long is the fish infectious? Sick wild salmon are eaten by predators, so they are not infectious for as long as farm fish, which are protected from predators.
2. How many contacts does the infectious fish have before it dies? Wild salmon are more dispersed than farm salmon, so wild salmon have fewer contacts than farm salmon.

Public government records about disease on BC salmon farms provide good evidence that only about 3% of farm salmon die each year from infectious disease (page 11-9 of BCSFA 2024). With an estimate of how long a given wild salmon population is in "contact" with farm salmon, we can estimate the increase in mortality from exposure to salmon farm diseases.

Fraser River sockeye salmon – We estimate that farm-source infectious diseases increase annual Fraser River sockeye salmon mortality by 0.00068%. **It would take nearly 1,500 years for this mortality to add up to 1%.**

Evidence – Farm salmon are in contact with each other 24/7 throughout the year = 8,760 hours/year. Telemetry studies inform us that juvenile Fraser River sockeye salmon spend only about 2 hours of their lives near salmon farms (Rechisky et al. 2021), which is 0.02% of the time that farm salmon are in contact with other farm salmon (i.e., 2/8,760 = 0.02%). To estimate annual Fraser River sockeye salmon mortality from farm-source diseases, multiply 3% by 2/8,760 = 0.00068%. Information in Table 1 is consistent with this estimate.

Chinook salmon – We estimate that farm-source infectious diseases increase annual mortality of Chinook salmon near salmon farms by 0.15%. It would take nearly 7 years for this mortality to add up to 1%.

Evidence – Some juvenile Chinook salmon stay in nearshore environments and potentially near salmon farms during their first year in the ocean (Trudel et al. 2009). To estimate the potential for transfer of infectious pathogens from salmon farms to wild salmon, we need a marker of pathogen transfer that does not kill the fish. Because PRV prevalence approaches 100% in BC farmed salmon (Reference 8), PRV does not kill Chinook salmon under controlled laboratory conditions (References 95 and 96), and PRV infection persists for months (Reference 95), we can use PRV prevalence to estimate the number of infectious contacts during the year that wild Chinook salmon are near salmon farms.

Reference 8 provides evidence that PRV infects 10% of wild Chinook salmon nearest to salmon farms. Reference 8 did not publish individual fish data, but the corresponding author confirmed that Reference 8 used the dataset published with Reference 37 (G. Mordecai, personal communication). The dataset for Reference 37 includes PRV+ test results from 141 of 2,823 (5.0%) Chinook salmon sampled in BC from 2008 – 2018 during the Fall-Winter period, when Reference 8 reports an association with PRV and distance to salmon farms. [We did not receive information about which of the 2,823 Chinook salmon were considered to have been sampled near salmon farms.] References 8

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and 37 did not follow their cited protocol (Miller et al. 2016) for test interpretation: "it is imperative that the assays run on the platform be carefully evaluated for sensitivity (limit of detection) … so that the limitations of what can and cannot be concluded are well established and measures are implemented to minimize the potential for false positive or negative results." Based on the published limit of detection = 1.0 (page 20 of supplemental file, Bass et al. 2024), among the fall-winter samples, only 49% of the 141 PRV+ test results have a copy number above the limit of detection. Therefore, an estimate of PRV prevalence of Chinook salmon *near salmon farms* that accounts for the limit of detection is 49% of 10% = 4.9%.

If we assume that other salmon farm pathogens transfer at the same rate as PRV, we then multiply the annual mortality due to infectious disease among farmed Chinook salmon (3%) times the estimate of pathogen transfer (4.9%) to estimate that farm-source pathogens cause a 0.15% increase in annual mortality among Chinook salmon near salmon farms.

In support of estimates of minimal effects of farm-source PRV on population productivity, as measured by the smolt-to-adult return ratio, prevalence of PRV does not seem to correlate with poor survival. For example, Reference 8 (Figure S4) shows that Chinook salmon PRV sample prevalence is about 10-fold greater in Columbia River stocks—which migrate far from salmon farms—than in Fraser River stocks—which migrate close to salmon farms. Yet, since 1990, Columbia River Chinook salmon population productivity has not trended less than Fraser River stocks (Reference 59).

Differences in farm and wild salmon disease processes

The Review: "Reasons for increased risk in wild salmon include size and life-stage differences; differences in physiological infection susceptibility; an inability to benefit from vaccines, antibiotics, and other treatments; and susceptibility to mortality from multiple ecological processes that pathogens can modulate, such as predation, competition, migration, and environmental stress."

1. Comment on "size and life-stage differences": Among the three infectious agents highlighted in *The Review*, only sea lice has been shown to differentially impact smaller wild salmon compared to farmed salmon. However, as we have outlined in Main Point #1 above, these differences are not manifest at the population level after 40 years of salmon farming in BC.
2. Comment on "differences in physiological infection susceptibility": Twenty years ago, the understanding that Pacific salmon were more resistant to endemic diseases than Atlantic salmon farmed in BC was considered to be standard knowledge that did not require citation; for example, from the Introduction section of (Kim et al. 2004): "In recent years, production of Atlantic salmon has dominated the industry but the resistance of Pacific salmonids to endemic disease organisms is leading to increased interest in their culture."
3. We are not aware of any pathogens other than sea lice for which wild salmon are more susceptible to infection than farmed salmon. However, wild Pacific salmon are probably less susceptible to *Tenacibaculum maritimum* than farmed Atlantic salmon (*Salmon salar*). This hypothesis is based on greater frequency of disease due to *T. maritimum* in farmed Atlantic salmon vs. farmed Pacific salmon species (see Main Point #6, item 3 below).
4. Comment on "inability to benefit from vaccines, antibiotics, and other treatments": Most farmed Chinook salmon in BC are grown organically without antibiotics or sea lice treatments. No BC farmed Atlantic salmon or Pacific salmon species are vaccinated against any of the three infectious agents highlighted in *The Review* (i.e., PRV, sea lice, or *T. maritimum*). And yet, **only about 3% die each year from infectious diseases** (page 11-9 of BCSFA 2024). Where lack of treatments for wild salmon increased mortality of infected fish, the time that those fish were infectious to cohorts would also decrease.
5. Comment on "susceptibility to mortality from multiple ecological processes that pathogens can modulate, such as predation, competition, migration, and environmental stress": Where these variables increase mortality of infected wild salmon, the time that those fish were infectious to cohorts would also decrease.
6. Important information omitted by *The Review*: Study of 652 samples from BC sockeye salmon (Thakur et al. 2019) collected "from before and during aquaculture expansion in BC (1985–94)"

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yielded a conclusion that, "In general, our data suggest that agent distributions may not have substantially changed because of the salmon aquaculture industry." This supports the hypothesis that wild salmon populations have been living with the potential pathogens that affect farm salmon for as long as there have been wild salmon. A possible exception is PRV (Reference 8), but BC PRV has not been shown to cause significant disease to Pacific salmon species under controlled laboratory conditions (References 94 – 97).

[Main point #5] Analyses cited in *The Review* that link salmon farm diseases to wild salmon population impacts (i) do not reflect reality, (ii) are unreliable because many are based on test results that might be false positives, iii) are based on imaginary data that are contrary to the evidence, (iv) avoid contrary information, (v) did not include medical examinations to rule out other causes of mortality, (vi) result in a conclusion that is weak and has not withstood the test of time, or (vii) are correlative with weak evidence of causation. Also, *The Review* does not consistently follow best practices for reporting new molecular findings.

The Review:

1. "In this review, we focus on pathogen-related risks of salmon farming to wild salmon in BC. We begin with three prominent pathogens—Piscine orthoreovirus (PRV), *Tenacibaculum* spp., and sea lice (*Lepeophtheirus salmonis*)—that previous studies have shown are prevalent in farmed salmon and linked to negative impacts on wild Pacific salmon populations in BC (19, 34, 35)."
2. "Many studies have now established a correlative link between marine survival of Pacific salmon in BC and exposure to salmon farms or pathogens of farmed salmon (19, 34, 69, 72–74)."

Reference 19 includes a disclaimer regarding its analysis (emphasis added), "In this study, we primarily tested for pathogen associations with survival one pathogen at a time, but **clearly this does not reflect reality ....**"

References 19 and 35 report results from qPCR-testing of wild BC Pacific salmon for 59 infectious agents. For both papers, assessments of PRV and *Tenacibaculum maritimum* are unreliable because they are based on a high proportion of test results that might be false positives. Therefore, the interpretation of qPCR+ test results as evidence of infection also has high uncertainty:

1. Neither reference follows their cited protocol for test interpretation (Miller et al. 2016): "it is imperative that the assays run on the platform be carefully evaluated for sensitivity (limit of detection) … so that the limitations of what can and cannot be concluded are well established and measures are implemented to minimize the potential for false positive or negative results." Neither Reference 19 nor 35 used a limit of detection, instead counting every duplicated result as a detection if the infectious agent was detected in at least one fish in the study at a copy number greater than the limit of detection.
2. For *T. maritimum*, Neither Reference 19 nor 35 adjusted the results based on the false positive rate established by the testing laboratory in Reference 36. Because all members of the *Tenacibaculum* genus, including *T. maritimum*, are obligate marine bacteria (Nowlan et al. 2020; Wakabayashi et al. 1986) and do not occur in freshwater, **all positive test results from freshwater samples are false positives** unless proven otherwise using methods other than qPCR alone (e.g., immunohistochemistry or *in situ* hybridization). Reference 36 reports that samples from 18 of 427 (4.2%) juvenile Atlantic salmon sampled from fresh water were qPCR+ for *T. maritimum*, with copy number of positive samples ranging from 0.3 – 200. These results were not reported as **false positives**; instead, they were reported as a new finding, with the hypothesis that "It is possible that these hatcheries introduced saltwater in the weeks before ocean transfer, to prepare smolts for release." However, the hatcheries participating in the Reference 36 study did not introduce salt water in the weeks before ocean transfer (veterinarians Terra MacDonald, Mowi Canada West, and Peter McKenzie, Cermaq Canada; personal communication).
3. In the dataset published by Reference 37—the source of data for Reference 35—the proportion of qPCR+ test results that are potentially false positives for PRV ranges from 24% for coho

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salmon to 77% for sockeye salmon (Table 4); for *T. maritimum*, the range is from 90% for Chinook salmon to 95% for coho salmon (Table 5).

4. Based on the Reference 37 dataset, the proportion of juvenile migrating Fraser River sockeye salmon with *T. maritimum* copy number greater than the limit of detection is more than 10-fold greater for fish not migrating near salmon farms on the west coast of Vancouver Island (20%, 6 of 30 fish) than for fish migrating near salmon farms on the east coast of Vancouver Island (1.5%, 32 of 2188 fish). Also, the sockeye salmon with the highest *T. maritimum* copy number (17,900) in the Reference 37 dataset was sampled from the west coast of Vancouver Island (away from salmon farms).
5. Reporting false-positive results, or those that have not been properly confirmed, might negatively impact regulatory decisions with large implications, such as the unjustified loss of an industry.

Table 4. qPCR test results for piscine orthoreovirus (PRV) in three wild Pacific salmon species, as reported by Reference 37. Prevalence greater than the limit of detection (LOD = 1.0 copy, from Bass et al. 2024) minimizes the potential for false positives (shaded row, Miller et al. 2016).

	Chinook	Coho	Sockeye
# sampled	5696	2025	4019
copy # >0	294 (5.2%)	119 (5.9%)	61 (1.53%)
copy # > LOD	157 (2.8%)	90 (4.4%)	14 (0.35%)
qPCR+ < LOD[a]	47%	24%	77%
# reported with jaundice	0 (0%)	0 (0%)	0 (0%)

[a]The proportion of qPCR+ test results that are potentially false positives.

Table 5. qPCR test results for *Tenacibaculum maritimum* in three wild Pacific salmon species, as reported by Reference 37. Prevalence greater than the limit of detection (LOD = 1.9 copies, from Bass et al. 2024) minimizes the potential for false positives (Miller et al. 2016). A precautionary approach also adjusts for the false positive rate established by the testing laboratory with known negative samples from freshwater Atlantic salmon, as reported in Reference 36 (i.e., copy number > 200; shaded row).

	Chinook	Coho	Sockeye	Negative samples
# sampled	3547	1995	3500	426
copy # > 0	254 (7.2%)	153 (7.7%)	106 (3.0%)	18 (4.2%)
copy # > LOD	207 (5.8%)	117 (5.9%)	71 (2.0%)	9 (2.1%)
qPCR+ < LOD	19%	24%	33%	50%
copy # > 200	26 (0.7%)	7 (0.4%)	6 (0.2%)	0 (0%)
qPCR+ < 200[a]	90%	95%	94%	100%
# reported with lesions[b]	0 (0%)	0 (0%)	0 (0%)	0 (0%)

[a]The proportion of qPCR+ test results that are potentially false positives. [b]Lesions characteristic of disease due to *T. maritimum* infection in Pacific salmonids are described in References 104 – 108.

Reference 19 reports that both Chinook and coho salmon have a high probability that fish condition—as compared using "length-weight residuals"—is negatively associated with *T. maritimum* copy number (i.e., fish with a higher *T. maritimum* copy number are more likely to be in poor body condition). However, this relationship is limited to only the nine fish (out of 5,542 = 0.16%) that had the greatest copy number (> 2,500) for *T. maritimum*; all nine of these fish had negative length-

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weight residuals. The Reference 19 dataset includes 24 other fish (0.43%) with probable true positive *T. maritimum* test results (i.e., copy number between 200 and 2,500), but these fish are equally distributed among positive and negative length-weight residuals. Also, the 16 fish in the dataset with the most negative length-weight residual all have a *T. maritimum* copy number = 0. In our experience, fish in poor condition tend to be more susceptible to bacterial infections. Reference #19 was not designed to determine whether poor body condition was a cause or result of infection with *T. maritimum*.

While the information in Reference 19 and 35 might be used to generate hypotheses for further study, neither regulatory nor judicial decisions should be based on studies (i) that do not reflect reality or (ii) where 24-95% of the detections are unreliable because they might be false positives.

References 21, 34, 72, 73, and 74 used imaginary data (e.g., low or no sea lice from 1990 – 2000) that are contrary to the evidence, and they did not conduct a complete medical examination to rule out other causes of mortality.

These five modeling studies reported a significant effect of sea lice on population productivity of wild **pink salmon** (References 21, 34, 72, and 74) and **coho salmon** (References 34 and 73) in the **Broughton Archipelago** region of BC.

Reference 72 (published in December 2007) includes, "If outbreaks continue, then local extinction is certain, and a 99% collapse in pink salmon population abundance is expected in four salmon generations." This prediction was unreliable. In the study area after 2007, sea lice on wild juvenile pink salmon continued at levels similar to 2007 for the next 7 years (Figure 1 of Reference 25). Four salmon generations after 2005 and 2006 (the last years of data used in the analysis by reference 72), the numbers of adult spawners in four rivers in the study area were **the greatest ever recorded** (records date back to the early 1950s; salmon farms first arrived in 1987) (Canada 2024a):

1. Lull River 2005 = 2,755 fish; 2013 = 36,779 fish
2. Viner River 2005 = 15 fish; 2013 = 2,288 fish
3. Ahta River 2006 = 1,218 fish; 2014 = 68,871 fish
4. Embley River 2006 = 49,459 fish; 2014 = 146,603 fish

In the reference area described by Reference 72, the even year pink salmon run decreased nearly 98% over three salmon generations from an estimate of ~1.5 million spawners in 2002 to an estimate of ~35,000 recruits (spawners plus catch) in 2008 (Dataset S1). This demonstrates that pink salmon population productivity—as measured by the recruit to spawner ration—is highly variable, and declines approaching the predicted 99% collapse occur independent of sea lice from salmon farms.

The other four papers have conclusions similar to Reference 72:

1. Reference 21 (from 2010) – "Populations exposed to salmon farms (those from the Broughton Archipelago) show a sharp decline in productivity during sea lice infestations relative to pre-infestation years."
2. Reference 73 (from 2010) – "During a period of recurring salmon louse infestations in a region of open netpen salmon farms, coho salmon productivity (recruits per spawner at low spawner abundance) was depressed approximately sevenfold relative to unexposed populations."
3. Reference 34 (from 2011) – "Our results show that sea lice abundance on farms is negatively associated with productivity of both pink and coho salmon in the Broughton Archipelago."
4. Reference 74 (from 2013) – "there was a strong negative relationship between pink salmon survival and sea lice infection of juveniles, implicating that efforts by the salmon farming industry to reduce sea lice levels during the wild salmon out-migration have positive implications for wild salmon survival and productivity."

We lack confidence in these conclusions because the available evidence about sea lice infestation on salmon farms is contrary to the assumptions used in the analyses. Here is how the Broughton Archipelago wild salmon populations were designated in these five papers during the years before 2001:

1. Reference 72 (2007) – "exposed preinfestation populations"

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2. Reference 73 (2010) – "before … salmon louse infestations"
3. Reference 21 (from 2010) – "prior to sea lice infestations (recruitment 1972–2001)"
4. Reference 34 (2011) – "there are no data on sea lice numbers on wild juvenile salmon before 2001. In our analysis we have dealt with the absence of louse records from farms in the 1990s by treating them as missing data …." [For the year 2000, the best-fitting model for pink salmon estimated that they were exposed to 50,000 farm lice; the best fitting model for coho salmon estimated that they were exposed to 10.42 million farm lice.]
5. Reference 74 (2013) – "Data describing louse abundances on farm and wild salmon from the onset of salmon farming in the Broughton Archipelago to the first reported infestation in 2001 (Morton and Williams 2003) were not available, but it is reasonable to assume that sea lice abundances were not epizootic during this period as outbreaks were not reported on salmon farms ([Reference 134]) or noticed on wild juvenile salmon (Morton and Williams 2003). However, to address this uncertainty, we treated $W_{a,t-1}$ as missing data for return years 1991 to 2001." [This reporting is contrary to Reference 134, which said, "[because] sea lice were sometimes common on farmed Atlantic salmon during the 1990s, farm-source sea lice probably infested juvenile pink salmon many years before they were first examined for sea lice in 2001 (1). Before 2000, farm fish sea lice were usually not quantified, but infestations were common enough that sea lice treatment options were investigated in the early 1990s (28), and publicly available records confirm that those treatments were used as early as 1996 …."]

Omitted from *The Review* and these five papers is the fact that we have no record that juvenile pink salmon or coho salmon were examined for sea lice before 2001. Each of these four papers used the resultant lack of evidence to assume no or few lice during the 1990s. As pointed out in Reference 161 by *The Review*'s coauthors AWB, SG, and GM (emphasis added), "regular reliance on the long-discredited technique of **alluding to an absence of evidence can become particularly worrying**." Contrary to the assumptions in these five papers, we have abundant evidence that sea lice were common on the farms before 2001 (none of the five papers cite any of this information):

1. References 34 and 72 coauthor AM reported more than 25 lice per infested farm salmon in the Broughton Archipelago in 2000 (Morton and Volpe 2002).
2. All early records of sea lice infestations on farmed BC Atlantic salmon better fit the hypothesis that lice have been common on these fish since Atlantic salmon farming began in BC during the 1980s:
    A. (Hicks 1989) – "Lice are commonly present on both wild and farmed stock."
    B. (Brackett et al. 1991) – "Indeed, in this survey, only sea lice were nearly comparable to BKD as a cause of morbidity in Atlantic salmon."
    C. Reference 169 (1992) – "In the Pacific Northwest, two species of sea lice commonly infect and cause disease in sea-farmed salmonids: *Lepeophtheirus salmonis* (the salmon louse) and *Caligus clemensi* (Figure 31)."
    D. (Stephen and Iwama 1997) – "Sea lice management in B.C. is accomplished largely through non-medicinal approaches such as fallowing and single-year class production and selection of species farmed, as well as by the presentation of ivermectin in feed."

If farm salmon had no sea lice in the 1990s, there would be no need for protocols to manage those infestations.

Population productivity of pink salmon during the 1990s and through 2001 does not fit the conclusions of these four papers. Omitted from *The Review*, but Reference 34 acknowledges the significance of treating the 1990s as missing data, "However, if lice were in fact abundant and infestations of wild juvenile salmon occurred in the 1990s, the estimated effect of lice on wild salmon survival would likely be diminished due to high salmon returns in those years." Indeed, the juvenile pink salmon that migrated past the lice-infested salmon farms in 2000 (Morton and Volpe 2002) returned as adults in 2001 with a **record high number of spawners** for the odd-year run (see Reference 134, Figure S2A). Also, for the entire period 1990 – 2001, average annual adult pink salmon returns to the Broughton Archipelago were (i) more numerous compared to the pre-farming years

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1960 – 1989 in the Broughton Archipelago and (ii) relatively more numerous than the nearby reference area with no farms from 1990 – 2001 (Table 3).

The conclusions of References 21, 34, 72, 73, and 74 are further weakened by the **lack of medical examination for other causes of mortality**, something that we consider to be routine in any diagnostic evaluation of a population with increased mortality (Marty et al. 1998; Middleton et al. 2021). Mathematical models, regardless of their complexity, are no better than their underlying data. These five studies provide no information about whether other pathogens or toxins might have influenced differences in productivity. This becomes problematic when trying to explain the following:

1. In the reference area, the number of recruits in 2004 and 2006 was 74 – 81% less than the number of spawners two years before (Table 6). In the Broughton Archipelago, the number of recruits in 2006 was 67% less than the number of spawners in 2004 (Table 6). References 21, 34, 72, 73, and 74 provide no evidence to rule out whether whatever decreased the number of recruits 74 – 81% in the reference area didn't also cause the 67% decrease in the Broughton Archipelago from 2004 - 2006.
2. In the Broughton Archipelago, progeny of the 2002 broods might have been exposed to more sea lice than progeny of the 2004 brood (Table 6), but the spawner/recruit ratio for the 2002 brood year returning to spawn in 2004 was the greatest in 27 even return years from 1962 – 2014, while the ratio for the 2004 brood year returning to spawn in 2006 was less than 24 other years over the same period (Table 6). References 34, 72, 73, and 74 provide no evidence to explain the cause of these differences.

Without medical examination for other causes of mortality, these analyses are no better than comparing head lice prevalence among two human populations with different mortality, noting a higher prevalence of head lice among the population with increased mortality, and then concluding that head lice are the reason for increased mortality without considering any other health variables.

Table 6. Estimates of pink salmon adult spawners and recruits (spawners plus catch) in the Broughton Archipelago and nearby reference area for brood years 2002 and 2004 (Dataset S1). The "farm lice #" is the estimated total number adult female *Lepeophtheirus salmonis* on all area farms in April of the year between the brood and return years, when susceptible juveniles would be exposed to farm-source sea lice (data from Reference 134). Sea lice mortality estimates are from Reference 74. Shaded are data associated with active salmon farms.

Area	Brood Year	# Spawners	farm lice # ($\bullet 10^6$)	Return Year	# Recruits	Spawner/recruit Ratio	Spawner/recruit Rank[a]	Sea lice Mortality
Reference	2002	1,545,980	0.0	2004	396,296	0.26	25/27	0%
Broughton	2002	110,319	4.8	2004	1,130,941	10.25	1/27	13%
Reference	2004	340,230	0.0	2006	61,175	0.19	27/27	0%
Broughton	2004	1,074,962	4.0	2006	355,995	0.33	25/27	40%

[a]Ranking is based on within-area spawner-recruit ratios for the even-year run for 1962 – 2014; catch data are not available for the Broughton Archipelago from 2010 – 2014, so ratios for those years are spawners per spawner.

When other medical reasons for poor health were studied in wild pink salmon in the Broughton Archipelago (Saksida et al. 2012), "Logistic regression analysis found skin lesions and hepatocellular hydropic degeneration significantly associated with sea lice." We expect sea lice infestations to be associated with skin lesions, but If liver lesions independent of farm-source sea lice are driving changes in population productivity, then efforts to decrease sea lice exposure (e.g., by closing salmon farms) will not improve wild salmon survival.

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Neither regulatory nor judicial decisions should be based on studies that **use imaginary data** (e.g., low or no sea lice from 1990 – 2000), especially when those data **are contrary to coauthor evidence** (Morton and Volpe 2002), that evidence is not disclosed, and other causes of morbidity are not investigated.

In **Reference 69**, what *The Review* describes as "a correlative link between marine survival of Pacific salmon in BC and exposure to salmon farms" is weak and has not stood the test of time – This 2012 reference examined the relation of Fraser River **sockeye salmon** productivity and different variables, including annual farm salmon production along the main sockeye salmon migration route. Study findings (emphasis added):

1. "Pink-salmon abundance was estimated to have the strongest negative influence on sockeye productivity…."
2. "SST [sea surface temperature] was also negatively associated with sockeye productivity and appeared in all models in the top model set (Tables 1 and 2)."
3. "Farmed-salmon production appeared in 5 of 6 models in the top model set (Table 1; RVI of 0.89 in Table 2), but **the estimated effect of farmed-salmon production** at average SST and pink-salmon abundance **by itself was essentially zero** (i.e., two orders of magnitude smaller than the other main effects; Table 2). As a result, accounting for the influence of farmed-salmon production on its own did not improve our ability to predict the observed decline in Fraser sockeye productivity since 1990 (Table S3)."

When Reference 69 was updated with similar analysis (Reference 41, published in 2020), salmon farms were not included in the analysis. Reference 41 cites Reference 69 only once, and then it does not mention salmon farms: "This approach is consistent with research that has suggested sockeye from Southeast Alaska through British Columbia primarily exhibit responses to pink salmon during their second and third growing seasons ([Reference 69])."

Neither regulatory nor judicial decisions should be based on a 12-year-old study that reports an effect that is "essentially zero," and those results are not confirmed by the same lead author's research on an updated dataset.

Correlation alone is weak evidence of causation

Most conclusions in *The Review* regarding threats of farm salmon pathogens to wild fish populations are based on spatial and temporal concurrence, supported by mathematical models. However, additional evidence is needed to equate spatial and temporal concurrence as cause and effect (Evans 1976; Hill 1965). For example, Evans (1976) provides a list of criteria needed to support cause and effect, including "The whole thing should make biologic and epidemiologic sense." Many of the conclusions in *The Review* and many of the papers that it cites fail to meet this criterion (e.g. the sea lice extinction hypothesis).

Hill (1965) points out the importance of temporality, or the "relationship of the association" for cause and effect. Consider Hill's example, "Does a particular diet lead to disease or do the early stages of the disease lead to those peculiar dietetic habits?" This example can be modified to consider the relation between *Tenacibaculum maritimum* and poor body condition: does exposure to *T. maritimum* lead to poor body condition, or does poor body condition lead to *T. maritimum* infection? Because Reference 19 reports that among the 100 fish with the poorest body condition—as measured by negative length-weight residuals—90% were qPCR-negative for *T. maritimum*, and only 2% had a copy number greater than 200; we interpret this as evidence that *T. maritimum* was at most a minor cause of the negative length-weight residuals. This is a situation where Hill's recommendation for experimental evidence could help clarify the relative susceptibility and potential consequences of Pacific salmon species exposed to *T. maritimum*.

Best practices for reporting new molecular findings

When investigators publish molecular data about potential new infectious agents, we recommend the following ethical guidelines for reporting these results in proper context (Fredricks and Relman 1996; Meyers and Hickey 2022; Middleton et al. 2021):

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1. Any implication that these detections are pathogenic must include supporting information (e.g., associated lesions or other evidence of an inflammatory or degenerative response). Without this information, limit discussion to general statements like, "Additional study is needed to determine whether this microbe is pathogenic."
2. Any implication that these detections impact populations must include supporting information (e.g., evidence of pathogenicity under controlled laboratory conditions; correlation with actual population data rather than modelled population data). Without this information, limit discussion to general statements like, "Additional study is needed to determine whether this microbe impacts population health."
3. Present any new molecular discovery information in the proper context regarding its significance for fish health in a way that can be understood by the media and the public for further constructive discussions.

The Review is inconsistent in meeting these recommendations:

Well done: "The infectious agents of wild Pacific salmon that we have identified as likely to be amplified by farmed salmon are based on patterns of molecular detections in wild salmon, farmed salmon, and environmental samples. However, such data do not necessarily represent viable infectious agents, as opposed to genetic material from agents that were nonviable at the time of sampling. Interpretation of such data needs care, and further work is needed to establish infectivity, pathogenicity, and ecological effects on wild fish (39)."

Needs improvement: Based in part on a study that does not reflect reality (Reference 19), a modelling study (Reference 9) with only three fish that we can be confident are true positives, and no reports of associated lesion in any qPCR+ fish, *The Review* concludes, "Thus, the combined body of evidence indicates that salmon farms may elevate exposure levels of *T. maritimum* for many species, that farms are a primary source of exposure for Fraser River sockeye salmon, and that *T. maritimum* may cause population-level impacts for some species."

[Main point #6] Many statements in *The Review* are contrary to information in the cited references (see below, #s 1, 4, and 5), **overstate information in the cited references (# 2), omit important information** (#s 3, 4, 6, 7, 8, 9, 12)**, omit contrary information** (#s 9, 13), **deceptively report cited references** (#s 9, 10, 11), **or are not ecologically relevant** (# 8).

1. *The Review* regarding the disease heart and skeletal muscle inflammation (HSMI; emphasis added): "Controlled laboratory challenge trials conducted in Norway have established that different isolates of PRV-1 vary in virulence (i.e., disease severity) and that **a PRV-1a isolate from BC can cause lesions diagnostic of HSMI**, but in a lower proportion of infected individuals than the most virulent PRV-1b isolate dominant in Norway (86)."

   Problem: Incorrect reporting of a cited reference.

   Comment: The statement that "a PRV-1a isolate from BC can cause lesions diagnostic of HSMI" is contrary to what Reference 86 concludes (emphasis added): "Following peak replication in blood, the two Norwegian 2018 isolates induced histopathological lesions in the heart consistent with HSMI, whereas all three historical Norwegian and **the Canadian isolates induced only mild cardiac lesions**." Mild cardiac lesions are not diagnostic for HSMI. Neither regulatory nor judicial decisions should be based on incorrect summaries of the evidence.
2. *The Review*: "Recent studies on wild Pacific salmon have indicated that the spread of *T. maritimum* from salmon farms is a risk to infection and survival of wild fish (*9, 19*)." Also, *The Review*'s critique of Reference 110 includes, "low impact was predicated on a low detection rate, an interpretation that subsequent work has shown to be invalid (*9*)." *The Review* cites Reference 9 four other times in similar context. The "low detection rate" is based on Reference 158, which reported 5 of 2,006 (0.25%) juvenile Fraser River sockeye salmon sampled in the spring and summer of 2012 and 2013 that were qPCR+ for *T. maritimum* with copy number greater than the limit of detection.

   Problem: Overstating the significance of a reference.

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Comment: Our efforts to obtain the datasets for References 9 and 158 from the authors were not successful. However, the data used for Reference 9 seem to be included in the Reference 37 dataset. Of the 2,280 sockeye samples in the Reference 37 dataset that seem to match the description of the data in Reference 9, only 1.4% have a copy number greater than the limit of detection (LOD = 1.9 copies, from Bass et al. 2024), which is not substantially different from the 0.25% reported by Reference 158 and used by Reference 110. Further, we believe that the analysis reported in Reference 9 is invalid because all but three of the detections are less than 200 copies and, therefore, might be false positives. The data in Reference 9 are from 2008 – 2018, but the three probable true positives are all from 2015, which was an unusually warm year (Reference 9). We agree with Reference 9 that this is evidence that *T. maritimum* is more likely to grow in BC during abnormally warm years. However, **neither regulatory nor judicial decisions should be based on a dataset that includes only three fish**.

3. *The Review*: "*T. maritimum* is known to have caused substantial health issues for Pacific salmonids in California (*104*), Chile (*105, 106*), New Zealand (*107*), and Alaska (*108*)."
   Problem: Important information omitted.
   Comment: Omitted is (i) disease due to *T. maritimum* has never been reported in wild sockeye salmon, and (ii) disease due to *T. maritimum* is rare among farmed Chinook salmon and coho salmon in BC. From 2002 – 2018, government veterinarians in charge of health audits of BC salmon farms diagnosed **farm-level** mouthrot (which is caused by *T. maritimum*) for 106 of 1,446 Atlantic salmon farm audits but for 0 of 273 Pacific salmon farm audits (Wade and Weber 2020). Since its inception by the BC provincial government, this audit program samples only moribund and freshly dead fish because it "enhances the likelihood of detection of disease" (BC-MAL 2006). Among these 273 Pacific salmon farm audits, two audits included **individual fish** (i.e., not farm level) diagnosed with mouthrot: three Chinook salmon sampled in 2009 (Wade and Weber 2020) and one coho salmon sampled on 2014-03-20 (DFO 2024c). The DFO audit program has not diagnosed mouthrot among the 655 farmed Pacific salmon sampled as part of the program since 2014-03-20 and through 2022 (DFO 2024c).
4. *The Review*: "While *T. dicentrarchi* is highly virulent in some hosts (Atlantic salmon, rainbow trout), including wild and farmed Chinook in BC (*127*), it may have little effect in others, such as coho (*119*)."
   Problem: Incorrect reporting of a cited reference. Important information omitted.
   Comment: Reference 127 does not report sampling or examining any farmed fish. Instead, Reference 127 is a study of Chinook salmon that developed lesions associated with *T. dicentrarchi* infection 4 days after they were captured from the wild and held in tanks. Also, Reference 127 reports no lesions at the time of capture; therefore, Reference 127 provides no information about the virulence of *T. dicentrarchi* among Chinook salmon in the wild. Finally, Reference 127 is a non-peer reviewed manuscript that was posted on BioRxiv on 2023-02-21, 16 months before *The Review* was accepted for publication; *The Review* omits reasons that Reference 127 has not passed peer review.
5. *The Review*: "Wild BC Chinook salmon infected with PRV-1a in their first year of ocean life have … lesions that indicate the early signs of jaundice/anemia (*16*)." Also, *The Review* says that recent studies "indicate that PRV-1a is associated with jaundice/anemia in farmed (*15*) and wild Chinook salmon in BC (*16*) …."
   Problem: Incorrect reporting of a cited reference. Contrary to the statement in *The Review*, Reference 16 does not report that any sampled wild salmon had jaundice or anemia.
   Comment: Reference 16 reported qPCR test results for 46 infectious agents, including PRV, in samples from 319 juvenile Chinook salmon. Histopathology was done on selected samples from 44 fish that tested strongly qPCR-positive for at least one of six infectious agents, including samples from eight fish with the greatest PRV copy numbers. The analysis included no fish that

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had no or low copy numbers for all six infectious agents (i.e., no reference fish). This analysis and interpretation have several limitations:

A. Lesions that "indicate" the early signs of jaundice/anemia are hypothesized but unknown because BC PRV has never been shown to cause jaundice under controlled laboratory conditions (References 94 – 96).

B. Lesions in the eight PRV+ fish are nonspecific. As evidence, all lesions that occurred among the eight selected PRV+ fish also occurred in at least one of the other 36 fish that were selected for copy load for one of six other infectious agents (Reference 16, Table S3).
   - A nephrosis score = 1 is reported for two of eight fish selected for PRV copy number, one of four fish selected for *P. theridion* copy number, and one of six fish selected for *C. shasta* copy number.
   - A renal interstitial hyperplasia score = 1 is reported for six of eight fish selected for PRV copy number and for 17 of the other 36 fish, including at least two fish in each of the six groups of infectious agents.

C. Some information in the Reference 16 text does not match the results in its Table S3.
   - Reference 16 describes "one [fish] with renal tubular vacuolar degeneration leading to necrosis of kidney tubules," but its Table S3 does not include "renal tubular vacuolar degeneration," and the score = 0 for all eight PRV+ fish in the kidney "necrosis" column.
   - Reference 16 describes "some cases of mild necrosis in liver, spleen ellipsoids …," but Table S3 lists score = 0 for all PRV-fish in the liver "necrosis" and spleen "ellipsoids necrosis" columns.

6. *The Review*: "PRV infection of wild Pacific salmon has been correlated with exposure to salmon farms and impaired migration success (18, 57)."
   Problem: Important information omitted.
   Comment: Reference 57 hypothesized, "the decline in PRV infection between the low and high migration challenge groups suggests that PRV infection may reduce a host's capacity to complete a challenging upriver migration." For the Fraser River, the pre-migration challenge and post-migration challenge samples were mostly different species. The 38 pre-challenge samples included **no sockeye salmon (0%), but the 77 post-challenge samples included 74 sockeye salmon (96%)**. Samples were tested for three viruses (see the Pivot table in the S1 file of Morton et al. 2021), but only PRV was included in the analysis, despite similar trends in the decrease in prevalence pre- and post-migration challenge for all three viruses (Table 7).

   A. Reference 57 coauthor RR is also a coauthor on Reference 149, which is a critique of Reference 139. Reference 149 includes, "Selective reporting of analysis runs counter to basic statistical practice and scientific integrity …."

   B. The two viruses not included in the analysis—infectious salmon anemia virus (ISAV) and salmonid alphavirus (SAV)—are reportable to the World Organization for Animal Health (WOAH). The WOAH's list of susceptible species for ISAV (WOAH 2024) does not include any of the six species reported as positive for ISAV in the Reference 57 correction (Morton et al. 2021). Therefore, the WOAH does not recognize these ISAV test results as true positive results. Among the seven species listed as SAV+ by the Reference 57 correction (Morton et al. 2021), only steelhead (*Oncorhynchus mykiss*) are considered susceptible to SAV infection; these steelhead account for 6 of the 49 (12%) SAV+ results reported in the study. The WOAH-recognized competent authority for Canada is the Canadian Food Inspection Agency (CFIA); CFIA reports that Canada has "Pathogen freedom at the level of the country" for SAV (CFIA 2022), so CFIA does not recognize these SAV test results as true positive results.

   C. *The Review* does not explain the reason(s) that one coauthor (AM) is reporting sample prevalences of ISAV (21%) and SAV (24%) while another coauthor (KMM) reports sample prevalence of 0% for these viruses in wild salmon samples (e.g., Thakur et al. 2019).

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D. Of the 115 PRV Ct values reported by Reference 57, 110 (96%) have a Ct value >34.0. The five fish with a PRV Ct value < 34.0 include two fish classified as exposed to salmon farms and three fish classified as not exposed to salmon farms. These results do not support *The Review*'s citation of Reference 57 for "PRV infection of wild Pacific salmon has been correlated with exposure to salmon farms."

Table 7. Virus detection by qPCR in salmonids sampled from the Fraser River watershed. Sample prevalence of qPCR+ test results for piscine orthoreovirus (PRV), infectious salmon anemia virus (ISAV), and salmonid alphavirus (SAV), as reported by Reference 57 and its correction (Pivot Table in the S1 file of Morton et al. 2021). According to the second correction of Reference 57 (Morton et al. 2023), the shaded information reported in (Morton et al. 2021) was not part of the underlying data analyzed for Reference 57.

	Lower Watershed	Upper Watershed
# tested	38 or 39	76 or 77
PRV	40%	23%
ISAV	21%	11%
SAV	24%	4%

7. *The Review*: "PRV infection of wild Chinook likely also affects predation, competition, and migration outcomes, which could remove infected individuals earlier in the disease progression than the advanced stages of jaundice/anemia observed in farmed Chinook."
   Problem: Important information omitted.
   Comment: *The Review*'s coauthor AM in 2019 reported several jaundiced wild salmon and wrote (Morton 2019), "I took samples, which I will have tested [for PRV]." This is evidence that jaundiced wild salmon do survive to be observed. Omitted from *The Review* are the results from those PRV tests. Also, if the Miller laboratory tested jaundiced wild salmon for PRV, those results are also omitted from *The Review*.
8. *The Review*: Regarding the sea lice species *Lepeophtheirus salmonis*, "Controlled laboratory trials have shown that sockeye salmon infected with *L. salmonis* experience mortality, skin erosion, scale loss, and high levels of stress (145)."
   Problem: Statement is not ecologically relevant.
   Comment: Reference 145 reports mortality with a laboratory-induced lice load—50 preadult *L. salmonis* per 135 g fish—that is not ecologically relevant. Surveys of BC sockeye salmon report that 95% of juvenile BC sockeye salmon have no *L. salmonis*, and only 0.2% have more than five *L. salmonis* per fish (page 10-29 of BCSFA 2024). Reference 145 reports that for fish at average mass of 40 g or 80 g when exposed to *L. salmonis*, none died during the experiment that lasted 34 – 36 days postinfection; when skin abrasions and scale loss were first noted at 21 days postinfection, these fish were infested with about 14 preadult lice per fish. **Reference 145 did not test the effect of ecologically relevant sea lice loads.**
9. A relatively new sea lice treatment, hydrolicing, involves transferring farm salmon from the net pen to a well-boat, using jets of water to remove the lice, and then returning the fish to the net pen; unlike some treatment alternatives, hydrolicing involves no chemicals. Regarding hydrolicing in BC, *The Review* reports the following:
   A. "… the implementation of hydrolicing is coincident with an increase in incidental bycatch mortality of herring reported from salmon aquaculture sites in BC from less than 100,000 fish annually in 2011 to 2020 to over 800,000 in 2023 (*156*)."
      Problems: Incorrect and deceptive reporting of a cited reference. Important information omitted.
      Comment: Reference 156 is incorrectly listed in *The Review*'s References and Notes section with a date of 2020, and the year with over 800,000 herring is 2022, not 2023. Reference 156 shows that in 2023, incidental catch of herring was 52,000 fish, which is about the same as

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in 2016. To put the 800,000 fish in perspective (page 1-16 of BCSFA 2024), "The total Pacific herring incidental catch mortality at all salmon farms combined for all areas was equivalent to 0.086% of the herring spawning biomass for West Coast Vancouver Island that year". At that level of mortality, it would take more than 11 years to add up to 1%. Hydrolicing alone cannot explain the high incidental catch in 2022. Hydrolicing is used throughout BC to control sea lice, but in 2022, 99.6% of the Pacific herring (*Clupea pallasii*) incidental catch was in a single area (Clayoquot Sound); 88% of that was during the first 6 months of the year (Reference 156).

Whatever the cause in 2022, incidental catch is now closer to historical lows. For the first nine months of 2024, incidental catch of Pacific herring in Clayoquot Sound was 17,779, which is on track to be among of the lowest annual totals since 2011 (updated dataset cited as Reference 156; accessed 2025-01-24).

Finally, to further put this number in perspective, humpback whales (*Megaptera novaeangliae*) in the eastern Pacific are estimated to eat about 1,000 tonnes of prey in a 100-day feeding season, or 10 tonnes per day (Savoca et al. 2021). In British Columbia, about 10% of this prey is estimated to be juvenile Pacific herring, or 1,000 Kg per day (Moreaux 2025). If the consumed juvenile Pacific herring are 20g each (= 50 per Kg), then a single humpback whale consumes about 50,000 Pacific herring per day. Therefore, the total incidental catch of Pacific herring by the entire BC salmon aquaculture sector in 2023 is what one humpback whale eats in a single day.

B. "… these alternative treatment methods … can increase stress on treated salmon (153, 154) through loss of mucus and scales that normally help protect fish against infectious microbial agents. These host effects likely increase vulnerability to disease (154, 155) …."
Problem: Omitting evidence communicates risk that is contrary to the evidence.
Comment: In BC, alternative treatments like hydrolicing have not been associated with a significant change in infectious disease Fish Health Events reported to regulators as a condition of licence (Figure 3 in Jyoti et al. 2024). Hydrolicing was first used in BC to control sea lice in 2018, followed by annual increases in the number of treatments through 2022 (DFO 2023).

C. "Although hydrolicers and bath treatments may remove lice from farms, viable larval lice are released back into the nearby marine environment (A. Morton, personal observation)."
Problem: Omitting regulatory information communicates risk that is contrary to the evidence.
Comment: This quote could be interpreted to say that all viable larval lice removed from farm salmon are released back into the nearby environment, but this interpretation is not correct. Marine finfish B.C. aquaculture licence and conditions of licence (DFO 2024a) include (emphasis added), "6.13 The Licence Holder must ensure that all mechanical and bath treatments carried out on vessels and barges as Sea Lice Mitigation **capture and retain removed sea lice, which must be collected and disposed of on land**."

10. *The Review*: "CSAS concluded that there is a high likelihood of transmission of PRV-1 from farmed salmon to wild sockeye salmon, but with uncertainty ranging from low to high (162)."
Also, regarding the Canadian Science Advisory Secretariat (CSAS) assessment of the risk of PRV to BC wild salmon, "The 2019 risk assessment concluded that PRV-1 has been associated with severe heart inflammation in farmed Atlantic salmon. Those conclusions unconventionally excluded research conducted outside of Canada, which has causally linked PRV-1, including a variant from western North America, to HSMI in Atlantic salmon (161)."
Problem: Incorrect and deceptive reporting of a cited reference.
Comment: Reference 162 is incorrectly linked to the CSAS report for viral hemorrhagic septicemia virus (VHSV), not PRV. Here is the correct reference for 162: DFO. 2019. Advice from the assessment of the risk to Fraser River Sockeye Salmon due to piscine orthoreovirus (PRV)

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transfer from Atlantic Salmon farms in the Discovery Islands area, British Columbia. DFO Can. Sci. Advis. Sec. Sci. Advis. Rep. 2019/022.

The 2019 PRV CSAS report's listing that "PRV-1 has been associated with severe heart inflammation in farmed Atlantic salmon" in BC was a "characterization," not a conclusion, and the listing was followed by important information omitted by *The Review*: "but a causal relationship has not been established."

Reference 161 is a perspectives paper, not a laboratory report that could have determined whether BC PRV causes HSMI. Reference 161 cites a 2016 email from scientist Espen Rimstad that includes, "there is no doubt that the isolate [PRV from BC] … causes HSMI." However, this email was not peer reviewed, and it does not mention whether controls were examined. Dr. Rimstad's peer-reviewed description of the effects of BC PRV ("the Canadian isolates induced only mild cardiac lesions"; Reference 86) is consistent with the 2019 CSAS PRV report (i.e., not HSMI). Reference 86 was published in 2020, so it could not have been cited by the 2019 CSAS report. Neither regulatory nor judicial decisions should rely on a preliminary email statement that is contrary to later peer-reviewed interpretation from the same scientist.

11. *The Review*: "Some challenge studies in BC and Washington (with PRV-1a in Pacific salmon species) have not observed mortality or clinical jaundice/anemia (94–96). The data and observations in some of those studies, however, did show early signs of disease progression toward jaundice/anemia, consistent with observations from PRV-infected farmed Chinook salmon in BC (15) …."

Problem: Deceptive reporting of cited references.

Comment: This quote could be interpreted to say that some, but not all, challenge studies have not observed mortality or jaundice, but that interpretation is incorrect. It would be clearer to say, "No challenge studies have reported mortality or jaundice." If the Miller laboratory has conducted a challenge study exposing Chinook salmon to PRV derived from jaundiced salmon, those results are omitted from *The Review*.

Reference 15 is an observational study of samples from moribund and freshly dead farm salmon in BC that were collected by government auditors, not by the study authors. The assignment of "early signs of disease progression toward jaundice/anemia" in fish that are all end-stage (i.e., moribund or dead) is a hypothesis that is contrary to laboratory study, which has never proceeded to jaundice/anemia. While we are confident that PRV causes minor lesions in some Pacific salmon (e.g., Reference 95), we do not know what role—if any—these lesions play in the development of jaundice, which has never occurred in controlled laboratory study with PRV.

12. *The Review*: "The abundance and density of fish in salmon farms present ideal conditions for the growth of viruses, bacteria, and parasites (4–6) (collectively "pathogens"). This can create a new source of transmission to wild Pacific salmon that would not exist naturally (6), and the associated risks are likely to be elevated whether or not a pathogen is exotic."

Problem: Omits important information.

Comment: For as long as wild salmon have existed, migrating salmon have probably been naturally exposed to millions of infected salmon. Farms add no more than a slight variation to this pattern.

The number of infectious agents per fish is similar for farm salmon and wild salmon (e.g., see References 37, 169, and 201). Based on data in *The Review*'s Figure 1B, annual salmon aquaculture production varied around 80,000 tonnes from 2010 – 2019; if harvested fish average 5 kg, then during that time about 16 million maturing farm salmon were spread among the BC farms each year. From 2010 – 2015, more than 20 years after salmon farms were established along the migration routes of Fraser River salmon, adult salmon populations returning to just the Fraser River equalled or exceeded 15 million on four occasions (Table G4 of Reference 63):

- sockeye salmon – 28 million (2010), 19 million (2014)
- pink salmon – 21 million (2011), 16 million (2013)

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These maturing fish would have been exposed to infectious disease among their cohorts—probably for several weeks—as they concentrated towards the mouth of the Fraser River. The number of sockeye salmon juveniles outmigrating from the Fraser River from just Chilko Lake exceeded 50 million in 2010, 2014, and 2015 (page 36 of Hawkshaw et al. 2020); these fish were exposed to cohorts for several weeks as they migrated out of the Fraser River and into the Pacific Ocean.

The Review cites evidence that infectious agent environmental DNA (eDNA) is elevated in the marine environment near active salmon farms (Reference 38), but we are not aware of any studies that have demonstrated that this increase is any different from what would occur from natural exposure to millions of migrating cohorts.

13. *The Review*: "Stock-recruit fisheries models that include covariates for sea lice on farms near spawning rivers, or on juvenile wild salmon themselves, have provided evidence that sea lice on salmon farms are correlated with reduced population productivity for pink and coho salmon (34, 73, 74). Seemingly contradictory studies (134) …."

Problem: Omits contrary information.

Comment: In this context, Reference 134 is not the only "seemingly contradictory" study. Omitted from *The Review*, but two of *The Review*'s coauthors (AM and MK) conducted a study (Morton et al. 2011) that compared survival of wild pink salmon populations that outmigrated as juveniles in 2007 through either (i) a corridor within the Broughton Archipelago with active salmon farms, (ii) a nearby corridor in which salmon farms were fallowed that spring, or (iii) a reference region to the north with no salmon farms:

- "The survival of the pink salmon cohort [within the Broughton Archipelago] was not statistically different from a reference region without salmon farms."
- "From the rivers assessed in the Broughton Archipelago, only the Embly River clearly corresponds to the fallow migration route. That population experienced very poor survival, with a 90% decline, although it was subject to fallow intervention."

In retrospect, the record high adult pink salmon returns to some Broughton Archipelago rivers in 2013 and 2014 are more consistent with results from the omitted contrary study (Morton et al. 2011) than with Reference 34.

[Main point #7] Did Competing Interests minimize scrutiny on publications cited by *The Review*?

We acknowledge that scientists occasionally overstate information in a cited reference or omit important information. However, we believe that the number of examples in *The Review* is excessive and biased towards potential negative effects of salmon farms on wild salmon. We question whether Competing Interests might have contributed to what we interpret as a lack of scrutiny of *The Review* and many of its cited papers.

Steven Cooke served as editor for two scientific papers in which *The Review*'s coauthor KMM is senior author: **References 15** (FACETS, 2018) and **16** (Conservation Physiology, 2023). Overlapping this period, S. Cooke also coauthored 18 scientific papers with KMM, including **References 18** (Evolutionary Applications, 2014), **179** (Molecular Ecology, 2022), **180** (Canadian Journal of Fisheries and Aquatic Sciences, 2023), and **203** (Physiological and Biochemical Zoology, 2019).

In 2022, *The Review*'s coauthor MK was named as Co-editor in Chief of the Canadian Journal of Fisheries and Aquatic Sciences. He is coauthor on two publications in this journal in 2022 (References 9 and Shea et al. 2022); both papers state that (i) MK served as an Associate Editor at the time of manuscript review and acceptance, and (ii) peer review and editorial decisions regarding the manuscripts were handled by other editors. Eight other coauthors of *The Review* have published other scientific papers in the same journal since 2022:

1. Reference 83 (2023) – AM, GM
2. Reference 161 (2023) – AWB, GM, SG

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3. Reference 180 (2023) – ALB, KMM
4. (Routledge and Morton 2023) – AM
5. (James et al. 2023) - BR
6. (Murdoch et al. 2024) – BMC

[Main point #8] *The Review* is not an independent assessment of the impacts of BC's farmed salmon industry.

The issue of Science Advances that published *The Review* also includes a separate summary (Mather 2024) that provides an opinion about the significance of *The Review* (emphasis added): "Their research, coupled with similar work, has been critical in establishing **an independent assessment** of the environmental impact of BC's farmed salmon industry on wild salmon (Figure 1) and ocean ecologies." This quote is also included in The Pacific Salmon Foundation's blog announcing publication of *The Review* (Pacific Salmon Foundation 2024).

Mather (2024) does not define "independent assessment," but he does cite *The Review*'s Reference 161, which devotes an entire section to independent review. Three coauthors of *The Review* (SG, AWB, and GM) are also coauthors of Reference 161. *The Review* does not meet two guidelines for independent review that are included in Reference 161:

1. "To ensure that science advice does not reflect 'unwarranted claims, unacceptable interpretations, or personal views,' it needs to be reviewed independently from the body seeking advice (Kelly et al. 2014)." Fisheries and Oceans Canada (DFO) is the body that licenses salmon farms. *The Review* does not meet this definition of independent review for use by DFO because two coauthors (ALB and KMM) are affiliated with DFO.
2. "For peer review to reliably assess the quality of research … for government science advice, reviewers must be independent and external from any focal party that may wish to 'tip the scales' (Hames 2008)." *The Review* does not meet this definition of independent review because most of its coauthors advocate to "tip the scales" towards salmon farm removal either directly or through organizational affiliations:
    a. **The Pacific Salmon Foundation (PSF) is an advocate** that does not meet this definition of independence. The 18 Dec 2023 message from Michael Meneer, President and CEO, PSF (Meneer 2023), includes (emphasis added), "PSF will continue to communicate our science and **advocate for the removal of open-net pen salmon farms** by 2025." Nine of *The Review*'s 16 coauthors are either affiliated with the PSF (AWB, WSB, CMD, EDC, SG, SP, and BR), or they received from the PSF research funding (KMM) or postdoctoral funding (SG, WSB, CMD, and GM).
    b. **The David Suzuki Foundation is an advocate** that does not meet this definition of independence.
        i. The 14 December 2018 press release (David Suzuki Foundation 2018) includes (emphasis added), "We'll continue to bring forward the latest, most credible science on risks to wild Pacific salmon populations, **urging governments to get these ocean-based farms out of the way of wild salmon** …."
        ii. The 24 March 2021 press release (David Suzuki Foundation 2021) includes (emphasis added), "The David Suzuki Foundation, Georgia Strait Alliance, Living Oceans Society, Watershed Watch and independent biologist Alexandra Morton say stocking farms in the [Discovery Islands area of BC] would put wild salmon at risk. **They are in court today to argue** against granting an injunction to two fish farm companies, which want to stock pens in the Discovery Islands over the coming months." Alexandra Morton is a coauthor of *The Review*.
        iii. The acknowledgements section of Reference 38 says that it "was supported by funding from the David Suzuki Foundation." The author list for Reference 38 includes six of *The Review*'s coauthors (DS, AWB, GM, BMC, KMM, and MK).
    c. **The founder and director of the Raincoast Research Society** (*The Review*'s coauthor AM) **is an advocate** that does not meet this definition of independence**.** For example, in a 10

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February 2022 article (Lewis and Morton 2022), AM writes (emphasis added), "**The DFO bureaucracy must** align with its minister and the Prime Minister's Mandate Letter to **remove salmon farms** by 2025."

Further, *The Review* does not meet a related hallmark of robust science advice that is included in Reference 161: "Funding sources (e.g., non-governmental organisations, industry, etc.) can also influence research findings; industry-supported research, for instance, displays a tendency towards pro-industry results (Bhandari et al. 2004; Sismondo 2008; Lundh et al. 2017)." *The Review* does not meet this definition of independent review for use by DFO because the aquaculture industry has funded research by coauthors of *The Review*; for example:

1. Lead author MK has published three papers reporting results from research partly funded by the aquaculture industry and coauthored with employees of DFO (Patanasatienkul et al. 2013) and the aquaculture industry (Rees et al. 2015; Rogers et al. 2013).
2. Senior author KMM has projects partly funded by the aquaculture industry:
   a. (https://www.dfo-mpo.gc.ca/aquaculture/rp-pr/acrdp-pcrda/projects-projets/P-11-02-007-eng.html) – A final report has not yet been submitted for this project. One of us (GDM) is a collaborator on this project. An earlier draft of a final report for this project was released to the public (Reference 76) without permission from GDM or three other coauthors; GDM and the three other coauthors last submitted comments on a draft final report to KMM on 2021-04-01, but KMM has not responded to these comments.
   b. (https://www.dfo-mpo.gc.ca/aquaculture/rp-pr/acrdp-pcrda/projects-projets/P-04-01-006-eng.html) – Report from this work has been published (Funk et al. 2007).

Supplementary Materials: The following supporting information can be downloaded at the website of this paper posted on Preprints.org, Dataset S1: (2025PreprintsDatasetS1.xlsx).

Author Contributions: GDM: writing—original draft, conceptualization, investigation, review and editing, methodology, data curation, and project administration. TRM and MLK: writing—original draft, reviewing, and editing. JAF, TBW, and ES: writing—reviewing and editing.

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Conflicts of Interest: GDM was employed as a diagnostic fish pathologist by the BC Ministry of Agriculture and Food from 2004 – 2023; since 2023, his work as a Senior Fish Pathology Consultant includes about 70% of revenue from the commercial salmon farming sector operating in BC; the remainder of his revenue comes from Fisheries and Oceans Canada, the BC Ministry of Agriculture and Food, the Veterinary Diagnostic Services Laboratory at the University of Prince Edward Island, and a variety of university research laboratories, commercial and public aquaculture operations, and environmental consulting firms. The other authors are employed by state government (JAF and TRM) or state-supported universities (TBW, MLK, and ES) in states that prohibit commercial marine net pen salmon aquaculture. From 1988 – 1999, MLK was employed by Fisheries and Oceans, Canada, which has a mandate to sustainably manage fisheries and aquaculture. The authors declare that they have no other competing interests.

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