Key Papers on Sea Lice and Infectious Agents

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Preamble

Below, we summarise published (or soon to be published) scientific findings, including seminal works, which pertain to the disease risks posed by salmon farms to BC's wild Pacific salmon. Much of this work has not featured prominently in recent assessments by DFO. This is not an exhaustive list of all papers relating to infectious agents on farmed and wild salmon — such a list would be unreasonably long. Instead, this list presents key peer-reviewed publications that we consider most relevant (in terms of findings and/or geographic location) for ongoing DFO open-net salmon aquaculture transition consultations. References are ordered within sections to form a coherent narrative, sometimes chronologically but sometimes not.

Sea Lice

Summary

The connection between salmon farms and sea louse infestation in juvenile salmon — pink and chum in particular — has long been unequivocally established in BC (not to mention globally), following work in the Broughton Archipelago that carefully tracked levels of developing sea lice on wild salmon as they migrated past farms[1,2]. The most ecologically comprehensive evidence from BC of population-level impacts of farm sea lice on pink and coho salmon comes (again from the Broughton Archipelago) from standard fisheries models adapted to test for the effects of observed levels of sea lice[3].

Salmon farms are major sources of sea-louse infection pressure for wild juvenile salmon, in some cases within 30 km of farms[1], and in some cases even further (depending on ocean conditions)[4]. Sea lice have profound physiological effects on wild salmon[5–9] and can cause direct mortality to their hosts, especially in smaller fish. Salmon infected with sea lice also experience increased predation[10,11] and have reduced competitive ability[12], feeding success[13], growth rate[14], and swimming endurance[15]. Salmon farms and/or sea lice have been linked to decreased population productivity of wild pink[3,16], coho[3,17], and sockeye[18] salmon born in or migrating through salmon farming regions. Outbreaks of sea lice on BC salmon farms are expected to get more frequent and more severe as the ocean warms[19], as are the effects of sea lice on their hosts[20,21]. Farm outbreaks will be more difficult to manage now that sea lice have developed resistance to emamectin benzoate[22], the main treatment that has been used to control sea lice on BC salmon farms.

Treatment resistance and climate change will worsen the sea-louse situation

Messmer et al. 2018[21]
This is the first published report of sea lice across BC showing signs of resistance to emamectin benzoate (EMB; trade name SLICE®)—the main treatment that has been used to control sea-louse outbreaks on salmon farms. Signs of resistance began in 2013.

Godwin et al. 2022[22]
Sea lice in the Broughton Archipelago appear to now be resistant to EMB.

Hamre et al. 2019[23]
Sea lice develop much faster at higher temperatures.

Bateman et al. 2016[24]
High sea lice numbers on wild and farmed salmon in the Broughton Archipelago in 2015 were probably linked to that year's marine heat wave.

Godwin et al. 2020[19]
We can expect more frequent and more severe sea-louse outbreaks as sea-surface temperatures continue to rise, and current parasite management on BC farms does not appear sufficient to control sea lice in warmer years.

Godwin et al. 2020[20]
Higher temperatures accentuate the negative effects of sea lice on juvenile Atlantic salmon survival, growth, and body condition.

Harrington et al. 2023[25]
Sea-louse populations on different salmon farms are highly connected with one another, and this connectedness depends on their distance from one another. Using DFO's state-of-the-art ocean current and environmental models from the Broughton Archipelago, this work showed that increased ocean temperatures will lead to increased levels of cross-infection.

Population-level effects of sea lice on wild salmon

a. Correlational population-level studies

Krkosek et al. 2007[26]
As of 2006, sea-louse infestations on wild juvenile pink salmon were: (1) associated with salmon farms, (2) associated with host mortality upwards of 80%, and (3) expected to cause a 99% collapse in local pink salmon populations unless management actions were taken to reduce infestations on farms. Management changes were implemented in the form of mandated treatments (see Peacock et al. 2013 below).

Connors et al. 2010[17]
As of 2007, population productivity of coho salmon exposed to salmon farms in the Broughton Archipelago was seven times lower than populations not exposed to salmon farms (populations from unexposed regions and Broughton populations pre-exposure).

Marty et al. 2010[27]
As of 2008, sea lice on farmed salmon and sea lice wild juvenile salmon were strongly linked in the Broughton Archipelago, but sea lice on farms were not associated with decreased population productivity of local wild pink salmon populations. However, it was later discovered (in Krkosek et al. 2011, below) that this study only had ~30% statistical power to detect a population-level effect if there was one.

Krkosek et al. 2011[3]
As of 2008, sea lice were negatively associated with pink-salmon population productivity in the Broughton Archipelago.

Peacock et al. 2013[16]

As of 2010, pink salmon declines in the Broughton Archipelago appeared to have been partially ameliorated by emamectin benzoate (EMB) treatments on farms (down to ~3-10% additional mortality in wild salmon due to sea lice, from >80% in the early 2000s).

Peacock et al. 2014[11]
As of 2010, sea lice were negatively associated with the population productivity of pink salmon, but not chum salmon. When juvenile pink and chum are infested with sea lice, predators preferentially eat the pink salmon, and the resulting relief in predation pressure appears to offset the negative effects of sea lice for chum (but adds to them for pinks).

Brookson et al. In prep
There is a positive relationship between the number of sea lice on salmon farms in the Broughton

Archipelago and the number of sea lice on wild juvenile salmon nearby. There is also a clear, negative association between sea louse infestation and survival of wild pink salmon at the population level. This analysis updates and reaffirms the analyses by Krkosek et al (2011)[28] and Peacock et al. (2013)[16] to include data from 2001 to 2021.

b. Experimental population-level studies

There have been no experimental population-level studies in BC because DFO has not allowed them. In Europe, many such studies have been conducted, which has established a causative link between sea lice and reductions in wild salmon returns. Two meta-analyses (which analyse many studies at one time) found that protecting juvenile salmon against sea lice during their outmigration (using EMB) increases their odds of surviving to return as adults by 18%[29] and 29%[30]. The former study also showed that the effect of sea lice was greater in years when baseline survival for salmon was lower (i.e. sea lice can exacerbate a bad situation). In the latter study, a complementary analysis predicted a 39% loss in returning adults due to sea lice. The most recent study analyzing such "paired release trial" data found, once again, that sea lice can have a large effect on wild salmon populations in Norway[31]. Notably, this type of study has been actively prevented in BC by senior DFO managers because "this work is not a priority" for DFO Science [32].

Sub-lethal effects of sea lice on juvenile salmon

Mages and Dill 2010[15]
Experimentally infested juvenile pink salmon have reduced swimming endurance relative to uninfested fish.

Krkosek et al. 2011[28]
Infested juvenile pink and chum salmon take riskier behaviour in the (thereby accepting higher predation risk) and have weakened abilities to avoid predatory strikes.

Peacock et al. 2015[10]
Infested juvenile pink and chum salmon are more likely to be eaten by predators.

Godwin et al. 2015[12]
Heavily infested juvenile sockeye salmon have decreased competitive feeding ability in western Johnstone Strait, BC.

Godwin et al. 2017[14]
Heavily infested juvenile sockeye salmon have decreased growth in Discovery Islands and Johnstone Strait. Early marine growth is of key importance for salmon survival.

Godwin et al. 2018[13]
Heavily infested juvenile sockeye salmon have reduced feeding success in western Johnstone Strait.

Atkinson et al. 2018[33]
Juvenile sockeye can reduce their sea-louse loads by leaping, which is energetically costly.

Physiological effects of sea lice on juvenile salmon

Long et al. 2019[5]
Sea lice cause a "profound physiological impact" in sockeye salmon relative to Atlantic salmon.

Long et al. 2019[6]
Sockeye salmon that are co-infected by sea lice and by infectious hematopoietic necrosis virus (IHNV) have reduced survival relative to uninfected fish and those only infected with sea lice.

Jakob et al. 2013[7]
Sockeye salmon infected by sea lice experience mortality, skin erosion, scale loss, reduced red-blood-cell counts, and higher stress levels, depending on the stage and intensity of infestation.

Jones et al. 2008[8]
In a laboratory, pink salmon develop a level of innate resistance to the direct (not sub-lethal) effects of sea-louse infestation by the time they are 0.7 grams (i.e. fish within a few weeks of marine entry).

Sutherland et al. 2011[9]
Pink salmon infected by sea lice exhibit inflammation and immune responses at small body sizes (0.3 grams), but innate immunity develops gradually as the fish get larger (to 2.4 grams).

Miscellaneous sea louse findings

Harrington et al. Preprint[34]
Specific salmon farms within a region can be disproportionate sources of sea lice, both for infecting other farms and infecting out-migrating juvenile salmon. This underlines that details can matter a great deal in sea louse infestation dynamics, and simply reducing farm numbers *may not* solve associated problems for wild salmon.

Godwin et al. 2021[35] Sea-louse counts on BC farms are probably underestimates; true counts are estimated to be ~1.18 times reported counts for *L. salmonis* and ~1.95 times reported counts for *C. clemensi*.

Krkosek et al. 2005[1]
Sea-louse infestation pressure on wild juvenile pink and chum salmon was 73 times higher near farms than background levels, and it exceeded background levels for 30 km away from the farms along the juvenile salmon migration route.

Frazer et al. 2012[36]
Density of hosts for sea lice play a fundamental component of disease dynamics in coastal seas, and are likely an influence leading to epidemics that require either rapid harvest or treatments. These factors are likely exacerbated by abiotic factors like temperature and salinity which additionally influence sea lice abundance.

Infectious Agents

Summary

Salmon farms are major sources for numerous infectious agents that can infect and cause disease in wild salmon. Other than sea lice, the two infectious agents of greatest concern in BC are the bacterium *Tenacibaculum maritimum* and the virus Piscine orthoreovirus (PRV). There is very strong evidence from BC that both *T. maritimum* and PRV are: (i) widespread in farmed salmon (especially in dead and dying fish, in the case of *T. maritimum*), (ii) transmitted from farmed to wild salmon, (iii) tightly associated with or known to cause disease in farmed and wild salmon, and (iv) linked to reduced survival in wild salmon.

***Tenacibaculum* species**

In BC, the majority of *Tenacibaculum* research has focussed around the *Tenacibaculum maritimum* as the causative agent of tenacibaculosis and mouthrot on Atlantic salmon farms[37]. Recent studies show both that salmon farms can expose nearby wild salmon to substantial *T. maritimum* infection pressure[38] and that some BC wild salmon species likely suffer population-level effects due to *T. maritimum* exposure[39]. Other recent studies (reviewed below), have recognised that other *Tenacibaculum* species detected in BC can also induce disease and mortality in both farmed and wild salmon populations[40–46].

Taken together, these studies suggest that the previous 'narrow' focus on just a single species of *Tenacibaculum* may underestimate the overall disease risks — to both wild and farmed fish — associated with farm infections of this bacterial genus.

Chen et al. 1995[47]
First identification of *Flexibacter maritimus* (since re-named *Tenacibaculum maritimum*) in the Pacific coast of North America on a variety of host species. Chinook salmon reared in marine net-pens had "severe gill lesions" colonized with the bacteria.

Avendaño-Herrera R et al. 2008[48]
Tenacibaculum maritimum samples were collected from turbot and sole on four fish farms in Spain and Portugal in 2003 and 2004. Isolates demonstrated resistance to a variety of antibacterial treatments, with farm specific resistance, indicative of evolved resistance.

Avendaño-Herrera et al. 2016[49]
Tenacibaculum dicentrarchi was identified in isolates collected from farmed Atlantic salmon in Chile following observed mortalities in 2010 and 2014. Challenge trials in Atlantic salmon and rainbow trout, exposing them to the isolate for one hour, resulted in high rates of mortality, 65 and 93% respectively. Challenged Coho salmon showed no signs of infection or mortality.

Småge et al. 2016 [50]
Tenacibaculum finnmarkense can result in tenacibaculosis in infected Altantic salmon in northern Norway, without pre-stress or other pathogens in co-infection. A whole cell inactivated vaccine of *T. finnmarkense* did not provide protection against tenacibaculosis following bath exposure.

Olsen et al. 2017[51]
89 isolates, collected across seven fish species, of *Tenacibaculum* from Norway were sequenced revealing four distinct groupings. One group represented *T. dicentrarchi*, whereas the other three groups likely represent undescribed species. The study identified *T. maritimum* and *T. ovolyticum* in new host species, as well as extended geographic ranges for *T. solea*. The overall lack of host specificity suggests that there may be multiple strains or groups that contribute to localized epidemics and that this diversity may complicate development of vaccines for tenacibaculosis.

Småge et al. 2018[52]
Tenacibaculum finnmarkense is genetically similar to *T. dicentrarchi*, grows in temperatures from 2-20°C and only in a marine environment.

Frisch et al. 2018[37]
The bacterium *Tenacibaculum maritimum* is the causative agent of 'mouthrot,' a widespread disease in Atlantic salmon on BC salmon farms. (In Pacific salmon, the general disease caused by *T. maritimum* is called "tenacibaculosis"). Mouthrot is induced by *Tenacibaculum maritimum* and can be rapidly horizontally transferred within populations of exposed salmon. Vaccines for *Tenacibaculum maritimum* did not protect salmon from exposures, subsequent infection, and mortality in this study.

Alvarez and Santos, 2018[53]
Several species of Tenacibaculum including *T. martimum, solae, discolor, gallaicum, dicentrarchi,* and *finnmarkense* are identified as the causal agent of tenacibaculosis. For aquaculture, it is key to develop techniques to determine which species are present in these fish-pathogen associations to highlight the diversity and presentations of this disease globally.

Brosnahan et al. 2019[54]
T. maritimum in farmed Chinook salmon in New Zealand, implicated as the possible cause of skin ulcers.

Klakegg et al. 2019[55]
Tenacibaculum dicentrarchi, originating from Spain, spread to a Norwegian Atlantic salmon farm and induced severe skin and muscle ulcers as well as mortality. Trials with a high challenge dose of *T. dicentrarchi* resulted in 100% mortality within 48 hours. This outbreak is the first of *T. dicentrarchi* in Atlantic salmon farmed in Norway.

Meyers et al. 2019[56]
In this Alaskan fish pathology book, *Tenacibaculum maritimum* is described as causing "mortality of juvenile Pacific salmon in seawater netpens during the winter and early spring."

Shea et al. 2020[57]
In seawater, *T. maritimum* environmental-DNA is almost exclusively detected next to active salmon farms sites (vs inactive sites) and shows one of the strongest associations with salmon farm operations among infectious agents studied.

Bateman et al. 2021[58]
T. maritimum is the most notable infectious agent present in dead and dying fish on BC salmon farms throughout the production cycle.

Valdes et al. 2021[59]
First report of *Tenacibaculum maritimum* infection in farmed rainbow trout from Chile. Challenge trials with collected isolates induced high mortality in Atlantic salmon (> 70% in 11 days), demonstrating wide ranging geographical reach for *martimum* and the ability to inflict mortality on a variety of salmonid hosts.

Nowlan et al. 2020[40]
Limited diagnostic tools exist to identify *Tenacibaculum* species outside of *maritimum* but many of the other species result in tenacibaculosis as well. Diagnostic tools should be expanded to encompass the variety of species and determine their distributions.

Nowlan et al. 2021[41]
Assays for QPCR detection of *Tenacibaculum dicentrarchi* and *finnmarkense* were developed.

Nowlan et al. 2021[42]
Tenacibaculum species (*T. maritimum*, *T. dicentrarchi*, and *T. finnmarkense* from isolates collected during outbreaks of mouthrot on BC Atlantic farms) produced clinical signs of mouthrot/tenacibaculosis and mortality in exposed Atlantic salmon. Some issues with disease challenges, but *T. maritimum* exposure resulted in the highest mortality.

Nowlan et al. 2021[43]
Tenacibaculum maritimum and *T. dicentrarchi* were both identified during disease outbreaks at commercial Atlantic salmon farms in the Broughton Archipelago. Bacterial load increased during outbreaks but did not diminish after, suggesting persistent infection. Increased mortality events and bacterial counts occurred in summer months; however, outbreaks occurred year-round.

Nowlan et al. 2022[44]
Assays for qPCR detection of *Tenacibaculum ovolyticum* were developed. The assays were deployed on

100 samples from a *Tenacibaculum* outbreak on an Atlantic salmon farm in the Broughton Archipelago and only detected *T. ovolyticum* in 2 samples, suggesting limited involvement in the outbreak.

Bass et al. 2022[39]
Tenacibaculum maritimum is associated with decreased survival in Chinook salmon and decreased body condition ("plumpness") in coho and Chinook along the BC coast.

Bateman et al. 2022[38]
Detection of *Tenacibaculum maritimum* in wild Fraser River sockeye salmon is strongly associated with salmon farms in the Discovery Islands, along their migration route northward. Evidence suggests that 6 to 56% of sockeye smolts may die as a result of farm-origin *T. maritimum* exposure. Farm-origin exposure peaked in the Discovery Islands with an approximately 13-fold increase as compared to background levels.

Spilsberg et al. 2022[45]
Tenacibaculum finnmarkense was associated with tenacibaculosis and mortality in sea farmed Atlantic salmon from Norway in the low water temperatures of late winter and early spring.

Nowlan et al. 2023[46]
Genetic sequencing of samples from Atlantic salmon with mouthrot on BC farms reveal the presence of *Tenacibaculum maritimum*, *T. ovolyticum, T. dicentrarchi, T. finnmarkense,* and two proposed novel species. Further genetic analysis highlighted the presence of genes involved in antimicrobial and antibiotic resistance, as well as toxin production and secretion systems.

Di Cicco et al. 2023 (Preprint)[60]
This clinical report represents the first diagnosed case of tenacibaculosis in wild-caught (captive) Chinook salmon in British Columbia, and highlights the possible role of *Tenacibaculum* species other than *T. maritimum* in the risks posed by salmon farms to wild salmon in BC. The report identifies *Tenacibaculum dicentrarchi* as the probable cause of clinical disease and substantial mortality of captive wild Chinook

Piscine orthoreovirus (PRV)

Piscine orthoreovirus (PRV) was first characterized in 2010[61] and, since it was first discovered in British Columbia (in diseased farmed Chinook), has been the subject of controversy for its potential to spread from salmon farms to wild salmon populations, putting wild fish at risk[39,62,63]. Initially, the origins of the virus were contentious, but it is now widely acknowledged in the scientific literature that PRV-1 originates from the Atlantic region[63–65]. Phylogenetic analyses indicate that the virus has been introduced to BC on multiple occasions[63]. Presently, PRV is known to comprise three strains, PRV-1, PRV-2, and PRV-3[66]. The PRV-1a substrain is the only lineage of PRV that has been detected in BC. Through infection with purified virus, all three strains of PRV have been identified as causative agents of disease, including the lineage found in BC (PRV-1a)[66–69].

Palacios et al. 2010[61]
Heart and skeletal muscle inflammation (HSMI) had been recorded in farmed Atlantic salmon in Norway since 1999, however the causative agent remained unrecognized. High throughput DNA sequencing studies identified piscine reovirus (PRV) as the putative causative agent and the authors urged that it must be controlled as it threatened commercial salmon production, but also wild populations.

Løvoll et al. 2012[70]

PRV was identified commonly (~36%) in pre-smolt Norwegian salmon destined for fish farms. When fish were transferred to saltwater viral load increased, declining with age. Fish were not able to clear the virus throughout the production cycle, and higher PRV loads were associated with HSMI outbreaks.

Wiik-Nielsen et al. 2012[71]
PRV is detected in wild marine fish in Norway.

Miller et al. 2012 (blocked study)[72]
First detection of PRV in the Pacific region. PRV detections were associated with jaundice/ anemia outbreaks on Chinook farms.

Kibenge et al. 2013[65]
Early phylogenetic analyses of PRV sequences generated from samples in BC and Chile concludes PRV in BC and Chile are both descendents of PRV from Norway.

Madhun et al. 2015[73]
Escaped farmed salmon in Norway implicated in exposing wild salmon to pathogens from farmed fish. Almost all escaped fish from this study were infected with PRV and salmon alphavirus. All three potential source farms shared a similar infection profile to escapees.

Siah et al. 2015[75]
Partial PRV sequence analysis across Alaska, British Columbia, and Washington State, demonstrated no identifiable geographic or temporal variation with identical sequence types found in 2001, 2005 and 2014 and across localities. World-wide comparative analysis indicates that these Canada/US Pacific sequences formed a subgroup from Norwegian sequence

Hauge et al. 2016[76]
The digestive tract of Atlantic salmon can act as a transmission point for PRV, potentially either by environmental exposure through water or contaminated faeces.

Godoy et al. 2016[77]
The first detection of HSMI lesions with presence of PRV in farmed Atlantic salmon outside of Europe. This was observed causing HSMI lesions in Atlantic and Coho salmon on Chilean farms. Additionally, some PRV strains detected here were unique, designating a new infectious strain, PRV- 3.

Garver et al. 2016[78]
This PRV challenge study aimed to examine the occurrence of clinical signs and mortality and to replicate jaundice syndrome in Chinook salmon. It concluded that PRV is not responsible for any sign of mortality or evidence of disease. Due to flaws in experimental methodology and sampling regime (tissues were not collected during the peak infection period), this study does not provide conclusive results on the role of PRV in the development of jaundice, anemia or any other PRV-related disease.

Garver et al. 2016[79]
Atlantic and sockeye salmon were challenged with PRV to examine possible disease development. The paper concluded that, “a complete lack of associated lesions and mortality in infected populations exhibiting high viral loads also indicates that western North American PRV is non-pathogenic.” Flaws in the design of the study (including an overly sparse sampling regime for histological samples) limit the value for understanding linkages between PRV and disease in Atlantic and sockeye salmon.

Lund et al. 2017[80]

PRV infected Atlantic salmon demonstrated reduced hypoxia tolerance when compared to non-infected fish both during, and following infection. Additionally PRV infected fish had lower maximum heart rate, optimum temperature at aerobic scope, signifying reduced cardiac and thermal tolerance performance.

Morton et al. 2017[62]
Occurance of PRV in wild pacific salmon around BC is correlated to exposure to salmon farms. PRV was detected in almost all Atlantic salmon (~95%) and in 37-45% of wild salmon in regions highly exposed to farms. PRV proportion was also lower following a migration event, indicating a selection of PRV on completing migration and thus reproductive capacity as well as fitness.

Di Cicco et al. 2017[81]
Heart and skeletal muscle inflammation (HSMI) diagnosed in BC farmed salmon, with PRV linked to the occurrence, severity, and timing of the diseased heart tissue.

Wessel et al. 2017[67]
PRV is internationally recognized as the causative agent of HSMI.

Purcell et al. 2018[83]
PRV RNA-positive individuals were detected at 3.4% total and ranging from 2-73% across 25 surveyed stock cohorts in a survey of various salmonids (n = 2,252; 121 stock cohorts) conducted in Washington and Alaska. PRV positive detections were most common for coho and Chinook salmon.

Di Cicco et al. 2018[84]
PRV is tightly associated with “jaundice/anemia” in farmed Chinook salmon.

Wang, 2018[85]
Wild juvenile Chinook salmon exhibit both gene expression and histopathological impacts associated with PRV

Vendramin et al. 2019[66]
Disease trials with PRV-3 demonstrate similar routes of infection, immune responses, and heart related pathology in infected rainbow trout, as compared to PRV-1 infected Atlantic salmon. Further this study demonstrates the wide ranging salmonid hosts which PRV strains can infect, resulting in similar clinical pathologies and mortality.

Dhamotharan et al. 2019[86]
Sequencing of PRV isolates over a 30 year period in Norway reveal the emergence of the PRV-1b lineage which has been associated with higher virulence. The authors hypothesise that this lineage emerged as a result of selection within the aquaculture environment.

Laurin et al. 2019[87]
PRV prevalence on farms in BC is such that it infects nearly all farmed Atlantic salmon at some point in the production cycle.

Kibenge et al. 2019 [88]
PRV prevalence is close to ubiquitous in escaped farmed Atlantic salmon following a large containment failure at a farm in northern Puget Sound. Icelandic salmon were used to stock Atlantic aquaculture sites in Washington State. The lineage of PRV detected in the escaped fish was similar to an Icelandic PRV sequence.

Zhang et al. 2019 and Correction[89,90]
Authors suggest that high-load PRV infections do not cause physiological impairment in salmon. To test

this, they used a "low-virulence strain of PRV" in a domesticated population of Atlantic salmon, noting no effect of this low-virulence strain on oxygen affinity, oxygen carrying capacity of erythrocytes, or activation of antiviral response pathways. In their original manuscript the authors failed to disclose some of their funding sources for their research, which was supported by "The British Columbia Salmon Farmers Association."

Wessel et al. 2020[68]
An isolate of PRV *from BC* (from the PRV-1a substrain) causes lesions diagnostic of HSMI in a controlled lab setting, but not as severe as other isolates tested. Differences in virulence were not consistent with phylogenetic separation of the two PRV sub-lineages (PRV-1a and 1b).

Polinski et al 2020 (Review)[91]
Three lineages of PRV (1-3) have been found across wild and farmed salmonids across North and South America, Europe and East Asia. All three genotypes can establish systemic infections which target red blood cells and cause circulatory disease.

Siah et al. 2020[64]
PRV-1 from the Pacific is distinct from that of the North Atlantic but is a direct descendant. No evidence of divergent evolution of PRV-1 between farmed and wild fish.

Mordecai et al. 2021
Phylogeographic analyses found that PRV's first introduction to BC from Europe coincided with the advent of Atlantic salmon farming in the region. Molecular surveillance and viral genome sequencing concluded that resident wild Chinook salmon are more likely to be infected with PRV the closer they are to salmon farms, and that PRV regularly transmits between farmed and wild salmon.

Polinski et al. 2021
Polinski et al. (2021) aimed to determine metabolic costs of viral infection in sockeye salmon, concluding that PRV is of little consequence to sockeye. (This was used in the media to argue that PRV is of little consequence to wild salmon.)

Mordecai et al. (*In Press* in BMC Biology; estimated date of release: 28/02/2023), peer-reviewed response to Polinski et al. 2021[92]
The upcoming peer-reviewed response found that Polinski et al. 2021 contained statistical flaws, failed to integrate knowledge about diseases caused by PRV in other salmonids, and did not consider ecological realities that likely affect disease outcomes. They conclude that the data from Polinski et al. (2021) are not adequate to support the conclusions drawn, and in some instances the findings actually suggest that PRV may cause ecologically relevant physiological impairment (i.e. disease) in sockeye.

Polinski et al. 2022[93]
Nearly all Atlantic salmon on farms became infected with PRV, a finding consistent with previous studies. Although moderate and severe heart lesions mainly arose in populations with PRV, a restrictive study design prevented the paper from being able to link disease and infection in individual fish, and to therefore corroborate previous work that identified PRV from BC as the cause of heart disease in Atlantic salmon[68,81].

Bass et al. 2022[39]
PRV is associated with decreased survival in Chinook salmon and decreased body condition ("plumpness") in both coho and Chinook.

Other relevant information

Sea lice and the infectious agents discussed above present the best-studied examples of risks posed by salmon farming to wild salmon in BC. Other work provides context, sets the stage for further research, or indicates farm impacts not tied to particular agents. We summarise a small sample of such work.

Connors et al. 2012[18]
Standard fisheries models — of the same type used by DFO to manage salmon fisheries coastwide — were used to show that farmed salmon production (as of 2009) along the Fraser River sockeye migration route exacerbated sockeye competition with pink salmon, for an overall negative effect on sockeye marine survival.

Miller et al. 2014[94]
Cumulative and synergistic effects of changing environments with spillover of aquaculture-related pathogens are increasing stress burden on wild populations, increasing their vulnerability to rapidly increasing and expanding microparasites, while ultimately leading to reduced survival of wild stocks.

Sundberg et al. 2016[95]
Intensive aquaculture may select for bacterial strains that have high virulence at both long and short term scales, which may additionally be able to outcompete other bacteria. Selective pressures on farms thus can result in rapid changes to pathogen populations, with impacts on pathogen virulence, which can result in impacts for both farmed and wild populations.

Miller et al. 2017[96]
Salmon host transcriptional biomarkers are developed to distinguish fish in an active viral disease state from those carrying a latent viral infection, and viral versus bacterial disease states. These markers are effective across RNA viral species, salmon species, and salmon tissues.

Mordecai et al. 2019[97]
Novel viruses discovered in dead and dying farmed salmon that are widespread in farmed, hatchery and wild Pacific salmon.

Mordecai et al. 2020[98]
Novel or previously undiscovered viruses revealed in dead and dying farmed Atlantic and Chinook in British Columbia. Atlantic salmon calicivirus and Cutthroat trout virus-2 were in more than half of the farmed Atlantic salmon tested. Some of the viruses first discovered in farmed Atlantic salmon were in Chinook salmon, suggesting a broad host range.

Furey et al. 2021[99]
Viral infection can increase predation of outmigrating sockeye salmon by bull trout. Infection with infectious haematopoietic necrosis virus (IHNv) increased predation risk 15-26 times. Additionally, a greater variety of infectious agents increased risk of predation.

References

1. Krkosek, M., Lewis, M. A. & Volpe, J. P. Transmission dynamics of parasitic sea lice from farm to wild salmon. *Proc. Biol. Sci.* **272**, 689–696 (2005).
2. Krkošek, M., Lewis, M. A., Morton, A., Frazer, L. N. & Volpe, J. P. Epizootics of wild fish induced by farm fish. *Proceedings of the National Academy of Sciences* **103**, 15506–15510 (2006).
3. Krkosek, M. *et al.* Effects of parasites from salmon farms on productivity of wild salmon. *Proc. Natl. Acad. Sci. U. S. A.* **108**, 14700–14704 (2011).

4. Stucchi, D. J. *et al.* Modeling sea lice production and concentrations in the Broughton Archipelago, British Columbia. *Salmon lice: an integrated approach to understanding parasite abundance and distribution* 117–150 (2011).
5. Long, A., Garver, K. A. & Jones, S. R. M. Differential Effects of Adult Salmon Lice Lepeophtheirus salmonis on Physiological Responses of Sockeye Salmon and Atlantic Salmon. *J. Aquat. Anim. Health* **31**, 75–87 (2019).
6. Long, A., Garver, K. A. & Jones, S. R. M. Synergistic osmoregulatory dysfunction during salmon lice (Lepeophtheirus salmonis) and infectious hematopoietic necrosis virus co-infection in sockeye salmon (Oncorhynchus nerka) smolts. *J. Fish Dis.* **42**, 869–882 (2019).
7. Jakob, E., Sweeten, T., Bennett, W. & Jones, S. R. M. Development of the salmon louse Lepeophtheirus salmonis and its effects on juvenile sockeye salmon Oncorhynchus nerka. *Dis. Aquat. Organ.* **106**, 217–227 (2013).
8. Jones, S., Kim, E. & Bennett, W. Early development of resistance to the salmon louse, Lepeophtheirus salmonis (Krøyer), in juvenile pink salmon, Oncorhynchus gorbuscha (Walbaum). *J. Fish Dis.* **31**, 591–600 (2008).
9. Sutherland, B. J. G., Jantzen, S. G., Sanderson, D. S., Koop, B. F. & Jones, S. R. M. Differentiating size-dependent responses of juvenile pink salmon (Oncorhynchus gorbuscha) to sea lice (Lepeophtheirus salmonis) infections. *Comp. Biochem. Physiol. Part D Genomics Proteomics* **6**, 213–223 (2011).
10. Peacock, S. J., Krkošek, M., Bateman, A. W. & Lewis, M. A. Parasitism and food web dynamics of juvenile Pacific salmon. *Ecosphere* vol. 6 art264 Preprint at https://doi.org/10.1890/es15-00337.1 (2015).
11. Peacock, S. J., Connors, B. M., Krkosek, M., Irvine, J. R. & Lewis, M. A. Can reduced predation offset negative effects of sea louse parasites on chum salmon? *Proc. Biol. Sci.* **281**, 20132913 (2014).
12. Godwin, S. C., Dill, L. M., Reynolds, J. D. & Krkošek, M. Sea lice, sockeye salmon, and foraging competition: lousy fish are lousy competitors. *Can. J. Fish. Aquat. Sci.* **72**, 1113–1120 (2015).
13. Godwin, S. C., Krkošek, M., Reynolds, J. D., Rogers, L. A. & Dill, L. M. Heavy sea louse infection is associated with decreased stomach fullness in wild juvenile sockeye salmon. *Can. J. Fish. Aquat. Sci.* 1–9 (2017).
14. Godwin, S. C., Dill, L. M., Krkošek, M., Price, M. H. H. & Reynolds, J. D. Reduced growth in wild juvenile sockeye salmon Oncorhynchus nerka infected with sea lice. *J. Fish Biol.* **91**, 41–57 (2017).
15. Mages, P. A. & Dill, L. M. The effect of sea lice (Lepeophtheirus salmonis) on juvenile pink salmon (Oncorhynchus gorbuscha) swimming endurance. *Can. J. Fish. Aquat. Sci.* **67**, 2045–2051 (2010).
16. Peacock, S. J., Krkosek, M., Proboszcz, S., Orr, C. & Lewis, M. A. Cessation of a salmon decline with control of parasites. *Ecol. Appl.* **23**, 606–620 (2013).
17. Connors, B. M., Krkošek, M., Ford, J. & Dill, L. M. Coho salmon productivity in relation to salmon lice from infected prey and salmon farms. *J. Appl. Ecol.* **47**, 1372–1377 (2010).
18. Connors, B. M. *et al.* Migration links ocean-scale competition and local ocean conditions with exposure to farmed salmon to shape wild salmon dynamics. *Conservation Letters* vol. 5 304–312 Preprint at https://doi.org/10.1111/j.1755-263x.2012.00244.x (2012).
19. Godwin, S. C., Krkosek, M., Reynolds, J. D. & Bateman, A. W. Sea-louse abundance on salmon farms in relation to parasite-control policy and climate change. *ICES J. Mar. Sci.* **78**, 377–387 (2020).
20. Godwin, S. C., Fast, M. D., Kuparinen, A., Medcalf, K. E. & Hutchings, J. A. Increasing temperatures accentuate negative fitness consequences of a marine parasite. *Sci. Rep.* **10**, 18467 (2020).
21. Messmer, A. M. *et al.* A 200K SNP chip reveals a novel Pacific salmon louse genotype linked to differential efficacy of emamectin benzoate. *Mar. Genomics* **40**, 45–57 (2018).
22. Godwin, S. C. *et al.* Salmon lice in the Pacific Ocean show evidence of evolved resistance to parasiticide treatment. *Sci. Rep.* **12**, 4775 (2022).
23. Hamre, L. A., Bui, S., Oppedal, F., Skern-Mauritzen, R. & Dalvin, S. Development of the salmon louse Lepeophtheirus salmonis parasitic stages in temperatures ranging from 3 to 24°C. *Aquaculture Environment*

Interactions vol. 11 429–443 Preprint at https://doi.org/10.3354/aei00320 (2019).
24. Bateman, A. W. *et al.* Recent failure to control sea louse outbreaks on salmon in the Broughton Archipelago, British Columbia. *Can. J. Fish. Aquat. Sci.* (2016) doi:10.1139/cjfas-2016-0122.
25. Harrington, P. D., Cantrell, D. L., Foreman, M. G. G., Guo, M. & Lewis, M. A. Timing and probability of arrival for sea lice dispersing between salmon farms. *R Soc Open Sci* **10**, 220853 (2023).
26. Krkosek, M. *et al.* Declining wild salmon populations in relation to parasites from farm salmon. *Science* **318**, 1772–1775 (2007).
27. Marty, G. D., Saksida, S. M. & Quinn, T. J., 2nd. Relationship of farm salmon, sea lice, and wild salmon populations. *Proc. Natl. Acad. Sci. U. S. A.* **107**, 22599–22604 (2010).
28. Krkošek, M. *et al.* Fish farms, parasites, and predators: implications for salmon population dynamics. *Ecol. Appl.* **21**, 897–914 (2011).
29. Vollset, K. W. *et al.* Impacts of parasites on marine survival of Atlantic salmon: a meta-analysis. *Fish Fish* **17**, 714–730 (2016).
30. Krkošek, M. *et al.* Impact of parasites on salmon recruitment in the Northeast Atlantic Ocean. *Proceedings of the Royal Society B: Biological Sciences* **280**, 20122359 (2013).
31. Vollset, K. W. *et al.* Direct evidence of increased natural mortality of a wild fish caused by parasite spillback from domestic conspecifics. *Proc. Biol. Sci.* **290**, 20221752 (2023).
32. Godwin, S, C, Bateman, A, W, Mordecai, G., Jones, S. & Hutchings, J, A. Is scientific inquiry still incompatible with government information control? A quarter century later. *In Press*.
33. Atkinson, E. M. *et al.* Oust the louse: leaping behaviour removes sea lice from wild juvenile sockeye salmon Oncorhynchus nerka. *J. Fish Biol.* **93**, 263–271 (2018).
34. Harrington, P. D., Cantrell, D. L. & Lewis, M. A. Next-generation matrices for marine metapopulations: the case of sea lice on salmon farms*. *bioRxiv* (2022) doi:10.1101/2022.04.25.489382.
35. Godwin, S. C., Krkošek, M., Reynolds, J. D. & Bateman, A. W. Bias in self-reported parasite data from the salmon farming industry. *Ecol. Appl.* **31**, e02226 (2021).
36. Frazer, L. N., Neil Frazer, L., Morton, A. & Krkošek, M. Critical thresholds in sea lice epidemics: evidence, sensitivity and subcritical estimation. *Proceedings of the Royal Society B: Biological Sciences* vol. 279 1950–1958 Preprint at https://doi.org/10.1098/rspb.2011.2210 (2012).
37. Frisch, K. *et al.* Experimental induction of mouthrot in Atlantic salmon smolts using Tenacibaculum maritimum from Western Canada. *J. Fish Dis.* (2018) doi:10.1111/jfd.12818.
38. Bateman, A. W. *et al.* Atlantic salmon farms are a likely source of Tenacibaculum maritimum infection in migratory Fraser River sockeye salmon. *Can. J. Fish. Aquat. Sci.* 1–16 (2022).
39. Bass, A. L. *et al.* Identification of infectious agents in early marine Chinook and Coho salmon associated with cohort survival. *Facets* **7**, 742–773 (2022).
40. Nowlan, J. P., Lumsden, J. S. & Russell, S. Advancements in Characterizing Tenacibaculum Infections in Canada. *Pathogens* **9**, (2020).
41. Nowlan, J. P., Lumsden, J. S. & Russell, S. Quantitative PCR for Tenacibaculum dicentrarchi and T. finnmarkense. *J. Fish Dis.* **44**, 655–659 (2021).
42. Nowlan, J. P., Britney, S. R., Lumsden, J. S. & Russell, S. Experimental Induction of Tenacibaculosis in Atlantic Salmon (Salmo salar L.) Using Tenacibaculum maritimum, T. dicentrarchi, and T. finnmarkense. *Pathogens* **10**, (2021).
43. Nowlan, J. P., Britney, S. R., Lumsden, J. S. & Russell, S. Application of Quantitative-PCR to Monitor Netpen Sites in British Columbia (Canada) for Tenacibaculum Species. *Pathogens* **10**, (2021).
44. Nowlan, J. P. *et al.* Tenacibaculum ovolyticum 16S rDNA Quantitative-PCR Assay Development and Field Testing. *Fish. Sahul* **7**, 303 (2022).
45. Spilsberg, B. *et al.* Tenacibaculosis in Norwegian Atlantic salmon (Salmo salar) cage-farmed in cold sea water is

primarily associated with Tenacibaculum finnmarkense genomovar finnmarkense. *J. Fish Dis.* **45**, 523–534 (2022).
46. Nowlan, J. P. *et al.* Genomics of Tenacibaculum Species in British Columbia, Canada. *Pathogens* **12**, (2023).
47. Chen, M. E., Henry-Ford, D. & Groff, J. M. Isolation and Characterization of Flexibacter maritimus from Marine Fishes of California. *J. Aquat. Anim. Health* **7**, 318–326 (1995).
48. Avendaño-Herrera, R., Núñez, S., Barja, J. L. & Toranzo, A. E. Evolution of drug resistance and minimum inhibitory concentration to enrofloxacin in Tenacibaculum maritimum strains isolated in fish farms. *Aquac. Int.* **16**, 1–11 (2008).
49. Avendaño-Herrera, R. *et al.* Isolation, Characterization and Virulence Potential of Tenacibaculum dicentrarchi in Salmonid Cultures in Chile. *Transbound. Emerg. Dis.* **63**, 121–126 (2016).
50. Småge, S. B. *et al.* Tenacibaculum finnmarkense sp. nov., a fish pathogenic bacterium of the family Flavobacteriaceae isolated from Atlantic salmon. *Antonie Van Leeuwenhoek* **109**, 273–285 (2016).
51. Olsen, A. B. *et al.* Multilocus sequence analysis reveals extensive genetic variety within Tenacibaculum spp. associated with ulcers in sea-farmed fish in Norway. *Vet. Microbiol.* **205**, 39–45 (2017).
52. Småge, S. B. *et al.* Induction of tenacibaculosis in Atlantic salmon smolts using Tenacibaculum finnmarkense and the evaluation of a whole cell inactivated vaccine. *Aquaculture* **495**, 858–864 (2018).
53. Fernández-Álvarez, C. & Santos, Y. Identification and typing of fish pathogenic species of the genus Tenacibaculum. *Appl. Microbiol. Biotechnol.* **102**, 9973–9989 (2018).
54. Brosnahan, C. L., Munday, J. S., Ha, H. J., Preece, M. & Jones, J. B. New Zealand rickettsia-like organism (NZ-RLO) and Tenacibaculum maritimum: Distribution and phylogeny in farmed Chinook salmon (Oncorhynchus tshawytscha). *J. Fish Dis.* **42**, 85–95 (2019).
55. Klakegg, Ø., Abayneh, T., Fauske, A. K., Fülberth, M. & Sørum, H. An outbreak of acute disease and mortality in Atlantic salmon (Salmo salar) post-smolts in Norway caused by Tenacibaculum dicentrarchi. *J. Fish Dis.* **42**, 789–807 (2019).
56. Meyers, T. *et al.* Diseases of wild and cultured fishes in Alaska. https://www.adfg.alaska.gov/static/species/disease/pdfs/fish_disease_book.pdf (2019).
57. Shea, D. *et al.* Environmental DNA from multiple pathogens is elevated near active Atlantic salmon farms. *Proc. Biol. Sci.* **287**, 20202010 (2020).
58. Bateman, A. W. *et al.* Descriptive multi-agent epidemiology via molecular screening on Atlantic salmon farms in the northeast Pacific Ocean. *Sci. Rep.* **11**, 3466 (2021).
59. Valdes, S. *et al.* First report and characterization of Tenacibaculum maritimum isolates recovered from rainbow trout (Oncorhynchus mykiss) farmed in Chile. *J. Fish Dis.* **44**, 1481–1490 (2021).
60. Di Cicco, E. *et al.* Tenacibaculosis in wild-caught, captive Chinook salmon (Oncorhynchus tshawytscha) in British Columbia, Canada. *bioRxiv* 2023.02.17.529034 (2023) doi:10.1101/2023.02.17.529034.
61. Palacios, G. *et al.* Heart and Skeletal Muscle Inflammation of Farmed Salmon Is Associated with Infection with a Novel Reovirus. *PLoS One* **5**, (2010).
62. Morton, A., Routledge, R., Hrushowy, S., Kibenge, M. & Kibenge, F. The effect of exposure to farmed salmon on piscine orthoreovirus infection and fitness in wild Pacific salmon in British Columbia, Canada. *PLoS One* **12**, e0188793 (2017).
63. Mordecai, G. J. *et al.* Aquaculture mediates global transmission of a viral pathogen to wild salmon. *Sci Adv* **7**, (2021).
64. Siah, A. *et al.* Genomes Reveal Genetic Diversity of Piscine Orthoreovirus in Farmed and Free-ranging Salmonids from Canada and USA. *Virus Evol* (2020) doi:10.1093/ve/veaa054.
65. Kibenge, M. J. T. *et al.* Whole-genome analysis of piscine reovirus (PRV) shows PRV represents a new genus in family Reoviridae and its genome segment S1 sequences group it into two separate sub-genotypes. *Virol. J.* **10**, 230 (2013).

66. Vendramin, N. *et al.* Piscine orthoreovirus subtype 3 (PRV-3) causes heart inflammation in rainbow trout (Oncorhynchus mykiss). *Vet. Res.* **50**, 14 (2019).
67. Wessel, Ø. *et al.* Infection with purified Piscine orthoreovirus demonstrates a causal relationship with heart and skeletal muscle inflammation in Atlantic salmon. *PLoS One* **12**, e0183781 (2017).
68. Wessel, Ø. *et al.* Piscine Orthoreovirus-1 Isolates Differ in Their Ability to Induce Heart and Skeletal Muscle Inflammation in Atlantic Salmon (Salmo salar). *Pathogens* vol. 9 1050 Preprint at https://doi.org/10.3390/pathogens9121050 (2020).
69. Takano, T. *et al.* Full-Genome Sequencing and Confirmation of the Causative Agent of Erythrocytic Inclusion Body Syndrome in Coho Salmon Identifies a New Type of Piscine Orthoreovirus. *PLoS One* **11**, e0165424 (2016).
70. Løvoll, M. *et al.* Quantification of piscine reovirus (PRV) at different stages of Atlantic salmon Salmo salar production. *Dis. Aquat. Organ.* **99**, 7–12 (2012).
71. Wiik-Nielsen, C. R. *et al.* First detection of piscine reovirus (PRV) in marine fish species. *Dis. Aquat. Organ.* **97**, 255–258 (2012).
72. Bailey, I. Why a federal salmon study that found viruses at B.C. fish farms took 10 years to be released. *The Globe and Mail* (2022).
73. Madhun, A. S. *et al.* Potential disease interaction reinforced: double-virus-infected escaped farmed Atlantic salmon, Salmo salar L., recaptured in a nearby river. *J. Fish Dis.* **38**, 209–219 (2015).
74. Olsen, A. B., Hjortaas, M., Tengs, T., Hellberg, H. & Johansen, R. First Description of a New Disease in Rainbow Trout (Oncorhynchus mykiss (Walbaum)) Similar to Heart and Skeletal Muscle Inflammation (HSMI) and Detection of a Gene Sequence Related to Piscine Orthoreovirus (PRV). *PLoS One* **10**, e0131638 (2015).
75. Siah, A. *et al.* Piscine Reovirus: Genomic and Molecular Phylogenetic Analysis from Farmed and Wild Salmonids Collected on the Canada/US Pacific Coast. *PLoS One* **10**, e0141475 (2015).
76. Hauge, H. *et al.* Piscine orthoreovirus can infect and shed through the intestine in experimentally challenged Atlantic salmon (Salmo salar L.). *Vet. Res.* **47**, 57 (2016).
77. Godoy, M. G. *et al.* First description of clinical presentation of piscine orthoreovirus (PRV) infections in salmonid aquaculture in Chile and identification of a second genotype (Genotype II) of PRV. *Virol. J.* **13**, 98 (2016).
78. Garver, K. A. *et al.* Piscine Orthoreovirus from Western North America Is Transmissible to Atlantic Salmon and Sockeye Salmon but Fails to Cause Heart and Skeletal Muscle Inflammation. *PLoS One* **11**, e0146229 (2016).
79. Garver, K. A. *et al.* Piscine reovirus, but not Jaundice Syndrome, was transmissible to Chinook Salmon,*Oncorhynchus tshawytscha*(Walbaum), Sockeye Salmon,*Oncorhynchus nerka*(Walbaum), and Atlantic Salmon,*Salmo salar*L. *Journal of Fish Diseases* vol. 39 117–128 Preprint at https://doi.org/10.1111/jfd.12329 (2016).
80. Lund, M. *et al.* Hypoxia tolerance and responses to hypoxic stress during heart and skeletal muscle inflammation in Atlantic salmon (Salmo salar). *PLoS One* **12**, e0181109 (2017).
81. Di Cicco, E. *et al.* Heart and skeletal muscle inflammation (HSMI) disease diagnosed on a British Columbia salmon farm through a longitudinal farm study. *PLoS One* **12**, e0171471 (2017).
82. Cartagena, J., Tambley, C., Sandino, A. M., Spencer, E. & Tello, M. Detection of piscine orthoreovirus in farmed rainbow trout from Chile. *Aquaculture* **493**, 79–84 (2018).
83. Purcell, M. K. *et al.* Molecular testing of adult Pacific salmon and trout (Oncorhynchus spp.) for several RNA viruses demonstrates widespread distribution of piscine orthoreovirus in Alaska and Washington. *J. Fish Dis.* **41**, 347–355 (2018).
84. Di Cicco, E. *et al.* The same strain of *Piscine orthoreovirus* (PRV-1) is involved in the development of different, but related, diseases in Atlantic and Pacific Salmon in British Columbia. *FACETS* **3**, 599–641 (2018).
85. Wang, Y. The physiological associations between infectious agents and migrating juvenile Chinook salmon

(Oncorhynchus tshawytscha). (University of British Columbia, 2018). doi:10.14288/1.0375894.
86. Dhamotharan, K. *et al.* Evolution of the Piscine orthoreovirus Genome Linked to Emergence of Heart and Skeletal Muscle Inflammation in Farmed Atlantic Salmon (Salmo salar). *Viruses* **11**, (2019).
87. Laurin, E. *et al.* Histopathological and novel high-throughput molecular monitoring data from farmed salmon (Salmo salar and Oncorhynchus spp.) in British Columbia, Canada, from 2011–2013. *Aquaculture* **499**, 220–234 (2019).
88. Kibenge, M. J. T. *et al.* Piscine orthoreovirus sequences in escaped farmed Atlantic salmon in Washington and British Columbia. *Virol. J.* **16**, 41 (2019).
89. Zhang, Y. *et al.* Corrigendum: High-Load Reovirus Infections Do Not Imply Physiological Impairment in Salmon. *Front. Physiol.* **10**, 1354 (2019).
90. Zhang, Y. *et al.* High-Load Reovirus Infections Do Not Imply Physiological Impairment in Salmon. *Front. Physiol.* **10**, 114 (2019).
91. Polinski, M. P., Vendramin, N., Cuenca, A. & Garver, K. A. Piscine orthoreovirus: Biology and distribution in farmed and wild fish. *J. Fish Dis.* **43**, 1331–1352 (2020).
92. Polinski, M. P. *et al.* Innate antiviral defense demonstrates high energetic efficiency in a bony fish. *BMC Biol.* **19**, (2021).
93. Polinski, M. P., Gross, L. A., Marty, G. D. & Garver, K. A. Heart inflammation and piscine orthoreovirus genotype-1 in Pacific Canada Atlantic salmon net-pen farms: 2016-2019. *BMC Vet. Res.* **18**, 306 (2022).
94. Miller, K. M. *et al.* Infectious disease, shifting climates, and opportunistic predators: cumulative factors potentially impacting wild salmon declines. *Evol. Appl.* **7**, 812–855 (2014).
95. Sundberg, L.-R. *et al.* Intensive aquaculture selects for increased virulence and interference competition in bacteria. *Proc. Biol. Sci.* **283**, 20153069 (2016).
96. Miller, K. M., Günther, O. P., Li, S., Kaukinen, K. H. & Ming, T. J. Molecular indices of viral disease development in wild migrating salmon. *Conserv Physiol* **5**, (2017).
97. Mordecai, G. J. *et al.* Endangered wild salmon infected by newly discovered viruses. *Elife* **8**, (2019).
98. Mordecai, G. J. *et al.* Discovery and surveillance of viruses from salmon in British Columbia using viral immune-response biomarkers, metatranscriptomics and high-throughput RT-PCR. *Virus Evol* (2020) doi:10.1093/ve/veaa069.
99. Furey, N. B. *et al.* Infected juvenile salmon can experience increased predation during freshwater migration. *R Soc Open Sci* **8**, 201522 (2021).