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AQUACULTURE

Pathogens from salmon aquaculture in relation to conservation of wild Pacific salmon in Canada

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The spread of pathogens from farmed salmon is a conservation concern for wild Pacific salmon in British Columbia (BC), Canada. Three pathogens are prevalent in farmed Atlantic salmon in BC, spill over to wild Pacific salmon, and are linked to negative impacts on wild salmon: Piscine orthoreovirus, *Tenacibaculum* spp., and sea lice (*Lepeophtheirus salmonis*). Molecular screening of infectious agents in farmed and wild salmon and environmental DNA highlights a further 4 agents that are likely elevated near salmon farms and 37 that co-occur in wild and farmed salmon. Pathogens likely affect wild salmon indirectly by mediating migration, competition, and predation. Current net-pen aquaculture practices pose these risks to numerous populations of all species of wild salmon in BC, most of which are not covered in Government of Canada science and advisory reports. Climate change, pathogen evolution, and changes to disease management and aquaculture regulations will influence future risks.

INTRODUCTION

Salmon aquaculture has been among the fastest-growing seafood production systems over the last 30 years (*1*) and has replaced wild fisheries as the main producer of salmon both globally and in western Canada (Fig. 1). Meanwhile, wild Pacific salmon (*Oncorhynchus* spp.) have experienced widespread declines in the southern portion of their North American range, prompting fisheries restrictions and conservation listings for many populations from California through British Columbia (*2*). Salmon farming is not permitted in the western United States, but has expanded around Vancouver Island and on the central coast of British Columbia (BC), where farmed salmon (predominantly Atlantic salmon, *Salmo salar*) are raised in ocean net-pens at abundances similar to or larger than wild Pacific salmon populations that rear in or migrate through the shared marine environment (Fig. 2).

The introduction of large captive populations of domesticated Atlantic salmon, and in some cases Chinook salmon (*Oncorhynchus tshawytscha*), into coastal ecosystems inhabited by wild salmon and many other wild fish species, has created opportunities for novel ecological and evolutionary dynamics of their pathogens (*3*, *4*). 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. Examples of how salmon farms alter disease dynamics include pathogen introductions, amplification, spillover and spill-back between wild and farmed salmon (*7*–*9*), pathogen adaptation to new hosts (*10*), and the evolution of drug resistance (*11*, *12*) and virulence (*13*, *14*). These changes to disease dynamics have impacts on the health (*15*, *16*), growth (*17*), survival (*18*, *19*), and recruitment (*20*, *21*) of wild salmon. These pathogen interactions may be a primary mechanism for the association between salmon aquaculture development and wild-salmon declines observed in Europe, eastern Canada, and BC (*22*).

In BC, the possibility that salmon farms may contribute to disease, decline, and/or impaired recovery of wild Pacific salmon has resulted in concern by scientists (*23*–*25*), regulators (*26*–*28*), industry, Indigenous peoples, and the general public (*29*–*32*). Until recently, a large portion of farmed salmon production occurred along eastern Vancouver Island (Fig. 2). However, over the last few years, several initiatives led by Indigenous governments of the Dzawada'enuxw, Gwawa'enuxw, Kwiakah, Kwikwa'sutinuxw-Haxwa'mis, Mamalilikulla, 'Namgis, and shíshálh First Nations and decisions by the Canadian Federal Department of Fisheries and Oceans (DFO) (*33*) have resulted in the closure of most salmon farms in the Strait of Georgia, Discovery Islands, and Broughton Archipelago regions (Fig. 2). These closures represent nearly 50% of the salmon farms in BC, and have been implemented largely to reduce risks to wild salmon stocks from local watersheds and the Fraser River (Fig. 2), but also to address other environmental effects such as pollution, waste deposition, escapes, and interactions with marine mammals. Currently, Canadian policy on salmon aquaculture in BC is in flux, with the federal government considering options that range from re-establishing net-pen production in some decommissioned regions to transitioning the entire industry away from net-pen production.

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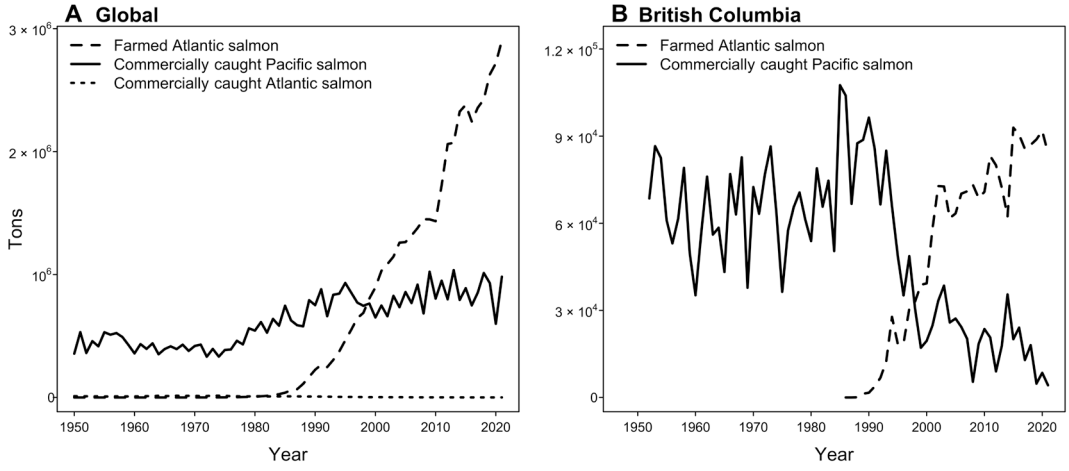

Fig. 1. Trends in production of salmon from aquaculture and commercial fisheries globally and in British Columbia. (**A** and **B**) Data are sourced from the Food and Agriculture Organization database for global aquaculture production (*193*) and Fisheries and Oceans Canada databases for aquaculture production (*194*) and commercial landings (*195*) in Canada.

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*). However, there are many more infectious agents that have been detected in farmed salmon (*36*) and wild salmon (*37*), for which environmental DNA (eDNA) is elevated in the marine environment near active salmon farms (*38*) (Fig. 3), and for which the probability of molecular detection in multiple wild salmon species increases with proximity to active salmon farms (*35*). Although such molecular detections do not guarantee infectivity (*39*), they are useful for investigating spatial and temporal patterns of pathogen distribution that are relevant for interpreting risk to wild salmon. We refer to these as infectious agents (and not pathogens) because, for some, the pathogenicity to wild salmon is unknown. We assemble data for 58 infectious agents across several high-throughput multi-infectious agent studies of farmed salmon, wild salmon, and eDNA to characterize the distributions of infectious agents among wild and farmed salmon in BC. From those data, we highlight four other infectious agents that occur in wild and farmed salmon and whose eDNA is elevated in the marine environment near active versus inactive sites (Fig. 3). We then analyze how these data and literature relate to Canadian government advisory reports published via the Canadian Science Advisory Secretariat (CSAS) on the pathogen risks to wild salmon in BC, particularly updating findings and applying them to salmon species and populations that were not encompassed in those reports. Before considering the pathogens, however, we briefly review the trends and status of wild salmon in BC in relation to salmon farming, broader regional variability, and other drivers of change.

STATUS AND TRENDS IN WILD PACIFIC SALMON IN BC

Although Pacific salmon are at historical abundance in the North Pacific Ocean due to hatcheries (*40*) and warming conditions that are favorable in the north (*41*, *42*), they have generally declined in the southern regions of their range, including BC, resulting in the collapse of commercial salmon harvests (Fig. 1B). This collapse is partly due to catch restrictions to protect small and vulnerable populations (*43*) but also low returns for commercially important stocks, including Fraser River sockeye (Fig. 2). Hypothesized drivers of declines in survival include changing ocean basin–scale environmental conditions, ocean warming at more southern latitudes, and competition among salmon at sea (*41*, *44*–*49*). Empirically, declines in smolt-to-adult survival, age at maturity, and body size (Fig. 2) have been broadly associated with increasing marine predator abundances (*50*–*52*) and competition among salmon at sea (*53*). For example, Chinook salmon return to spawn over a range of ages and the shift in age at maturity of West Coast Vancouver Island Chinook (Fig. 2) reflects a decreasing frequency of older returning individuals. In addition, degradation of freshwater habitats due to dams, land use, and climate change has contributed to declines in survival and abundance, particularly at more southern latitudes (*54*, *55*). Also in southern BC, parasite and pathogen interactions with farmed salmon are an additional stressor of wild salmon populations that migrate near salmon farms (*9*, *56*, *57*). Such is the multifactorial nature of the decline of wild salmon in BC.

The status of wild salmon populations in BC varies among regions, species, and life histories. Many populations of sockeye, Chinook, and coho salmon from the Fraser River and Vancouver Island have experienced large reductions in abundance, smolt-to-adult survival, age at maturity, and body size (*58*, *59*) and have been assessed as endangered by the Committee on the Status of Endangered Wildlife in Canada (COSEWIC) (*60*–*62*) (Fig. 2). For example, interior Fraser coho populations have not recovered from declines in the early 1990s (e.g., Fig. 2; North Thompson River coho) and sockeye salmon stocks in the Fraser River are at historic lows (*63*) (Fig. 2, Fraser River sockeye). In contrast, some populations of ocean-type Chinook, which migrate to sea in their first year of life, have increased in abundance in recent decades (*58*) (e.g., South Thompson Chinook, Fig. 2). On the central and north coasts of BC, numerous populations are also doing poorly. This includes chum salmon, which spawn in hundreds of systems and for which data from 25 well-documented systems indicate declines greater than 90% since the 1960s (*64*). Sockeye populations from large lakes up mainland

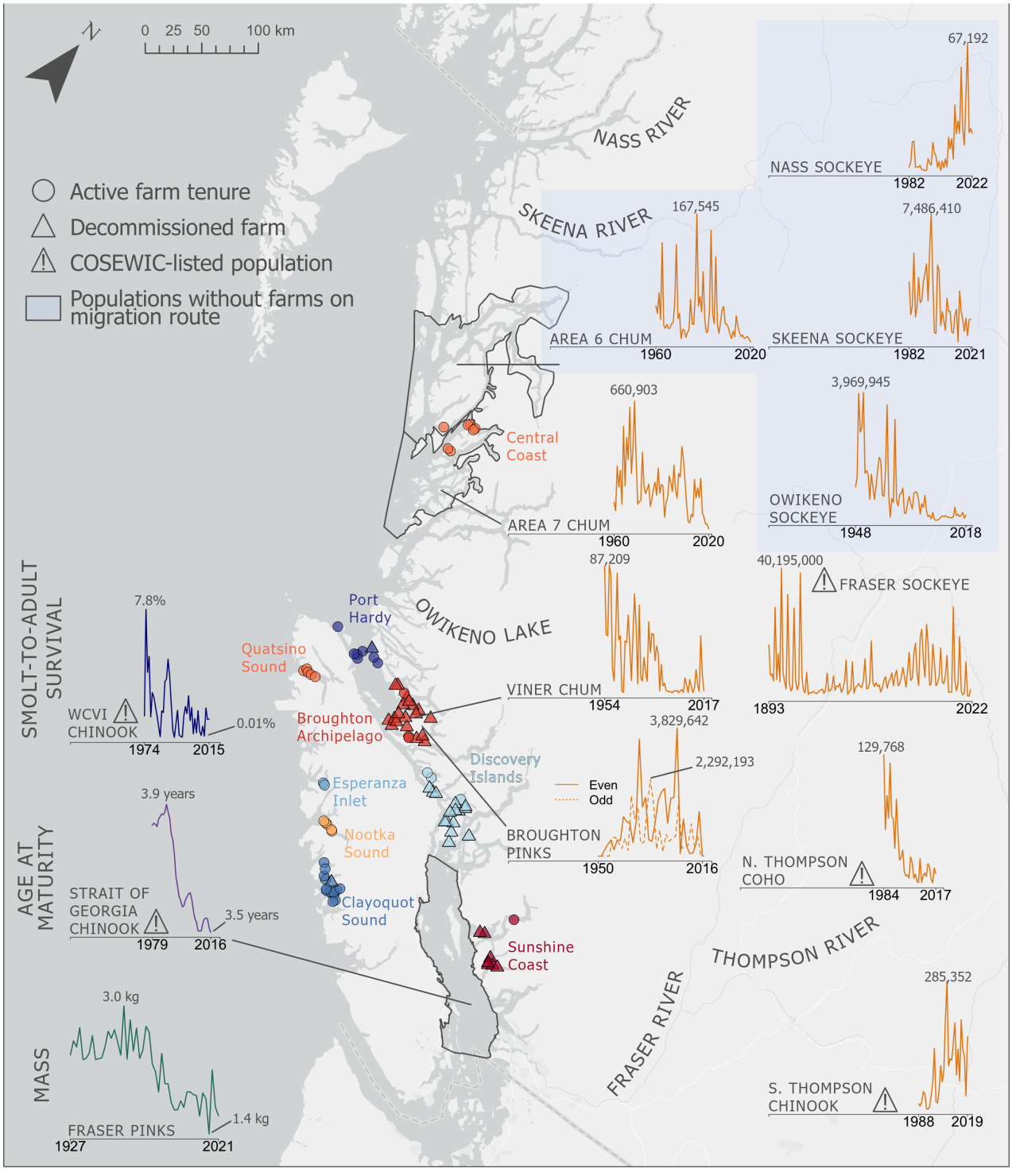

Fig. 2. Trends in spawner abundance (orange time series), smolt-to-adult survival (blue series), age at maturity (purple series), and mass (teal series) of select Pacific salmon populations in British Columbia, as well as salmon farm locations (symbols) colored by aquaculture management zone (circles = active farms, triangles = decommissioned farms). Imperiled populations designated by COSEWIC are indicated with exclamation marks and populations without active salmon farms on their migration routes are highlighted blue. Salmon populations were selected to show spatial variation in trends of stocks that are of economic or conservation importance, as well as those that likely interact with salmon farms (Area 7 chum, Viner chum, Broughton pinks, and Fraser populations). Inclusion of time series does not indicate a published relationship with salmon farming. Time series of smolt to adult survival, age at maturity, and mass illustrate changes that are likely unrelated to aquaculture but still contribute to declines of wild salmon in BC. Data on salmon abundance are sourced from the DFO NuSEDS database (*196*), except for Fraser River sockeye salmon that is sourced from the Pacific Salmon Commission. Data on life history traits are sourced from (*59*) for smolt-to-adult survival of Strait of Georgia Chinook, (*197*) age at maturity of West Coast Vancouver Island Chinook, and (*198*) body mass of Fraser River pink salmon.

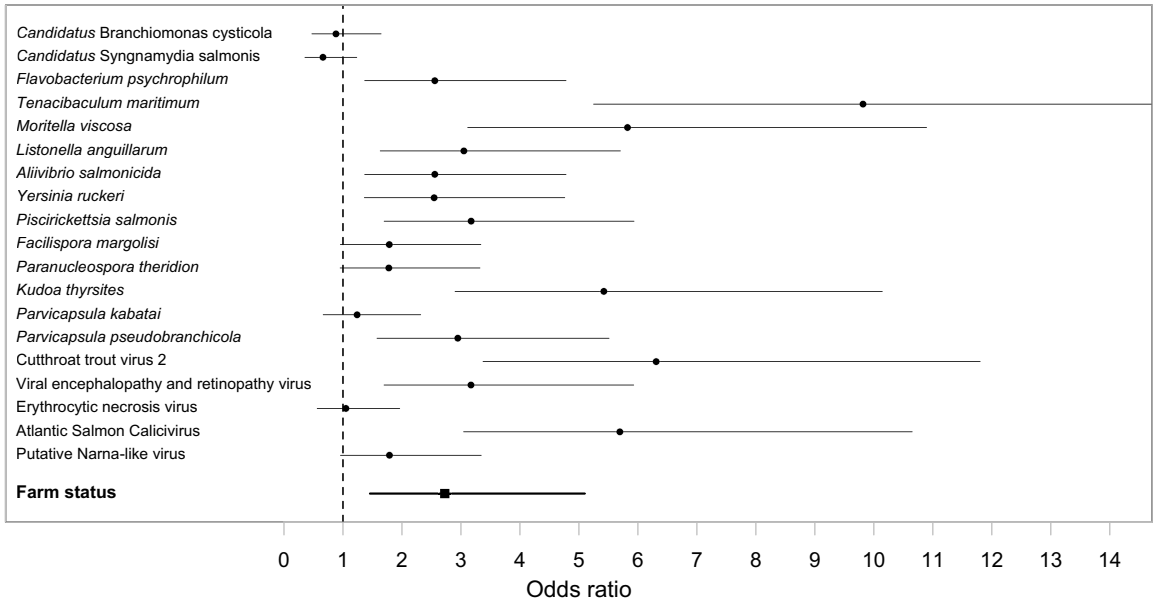

Fig. 3. Odds ratio of the occurrence of infectious agents in eDNA samples in the marine environment around active versus inactive salmon farm sites. The data are from 3 years of surveys at 58 sites in the Discovery Islands and Broughton Archipelago regions collected before the decommissioning of farms in those areas [see Fig. 2 for map, data from Shea *et al.* (*38*)]. In each of the 3 years of surveys, the status of sites as inactive versus active varied according to regular farm practices of stocking, harvesting, and fallowing. The plotted values represent post hoc best linear unbiased predictors for levels of a random effect, and should be interpreted with care (*199*). The upper confidence interval of the odds ratio for *T. maritimum* is 18.4. Model estimates and data are replotted from (*38*).

inlets on the central coast have also collapsed and been slow to recover (*65*, *66*) (Fig. 3; Owikeno Lake sockeye). However, further north, declines in sockeye have been more modest, but increasingly composed of enhanced production from Babine Lake in the Skeena watershed (*67*) (Fig. 2), and in some cases populations have increased in recent decades (e.g., lower Nass river type) (*68*).

In general, many wild salmon populations that migrate through areas of intensive salmon aquaculture (Fig. 2) are in southern BC, including populations from the Fraser River, Broughton Archipelago, and West Coast Vancouver Island, and they have been doing worse than those on the north and central coast (*34*, *61*, *69*–*71*). 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*). This has sparked research and conservation concern regarding the role of salmon aquaculture in declines and/or impaired recovery of wild salmon in BC. However, not all wild salmon systems in areas of aquaculture have declined to the same degree, and declines are not limited to areas with aquaculture (Fig. 2). Migration of wild salmon may facilitate pathogen spread beyond salmon farming regions, as has occurred for PRV, which was detected in wild salmon on the north coast of BC (*57*) and southeast Alaska (*8*), further complicating the interpretation of relationships between salmon farms and wild salmon declines. These nuances reflect the complexity of wild salmon declines in BC and that interaction with pathogens from aquaculture is one of many stressors, anthropogenic and natural, that contribute to the current state of wild salmon populations in BC. Below, we assemble and review the evidence on the risks of pathogens from salmon farms to BC's wild salmon populations.

PISCINE ORTHOREOVIRUS

For the past decade, PRV has been one of the primary infectious diseases of concern on salmon farms in terms of its risks to wild Pacific salmon (*15*). First discovered in diseased Atlantic salmon in Norway in 2010 (*75*), the virus was detected in farmed Chinook salmon in BC in 2012 (*76*), associated with disease on BC Atlantic salmon farms in 2013 (*77*), and has been documented to spread from salmon farms to wild salmon populations in BC (*8*, *35*, *57*). PRV is known to comprise three strains, PRV-1, PRV-2, and PRV-3 (*78*), of which PRV-1 is the only strain known to be present in BC. Analyses of genetic sequence data (*8*, *79*) indicate that PRV-1 originated in the Atlantic Ocean (Fig. 4B) and has been introduced to BC on more than one occasion, initially coincident with the advent of Atlantic salmon farming in the region (*8*). A few researchers assert, based on a single, very weak PCR detection in an archival liver sample of Steelhead from fresh water in BC from 1977, that the arrival of PRV predates the Atlantic salmon farming industry (*80*). Regardless, the phylogenetic evidence suggests an Atlantic origin.

Genetic diversity of PRV-1 in the Pacific regions likely reflects the transmission history among different populations. The history of Atlantic salmon introductions in the Pacific dates back to 1874 and includes decades of propagation in Columbia River hatcheries (starting in the 1970s) (*81*) and egg importation for salmon aquaculture (starting in roughly the 1980s) (*8*, *82*). Although PRV on farms is often genetically distinguishable among specific operating companies (*83*), all BC farmed Atlantic salmon are infected with a similar lineage (*8*, *79*). The unique lineage of PRV that is commonly detected in hatchery-origin Columbia River salmon is the same lineage infecting farmed Chinook salmon in BC but not the same as that infecting farmed Atlantic salmon in BC. Conversely, the lineage in farmed Atlantic salmon in BC does not appear to have become

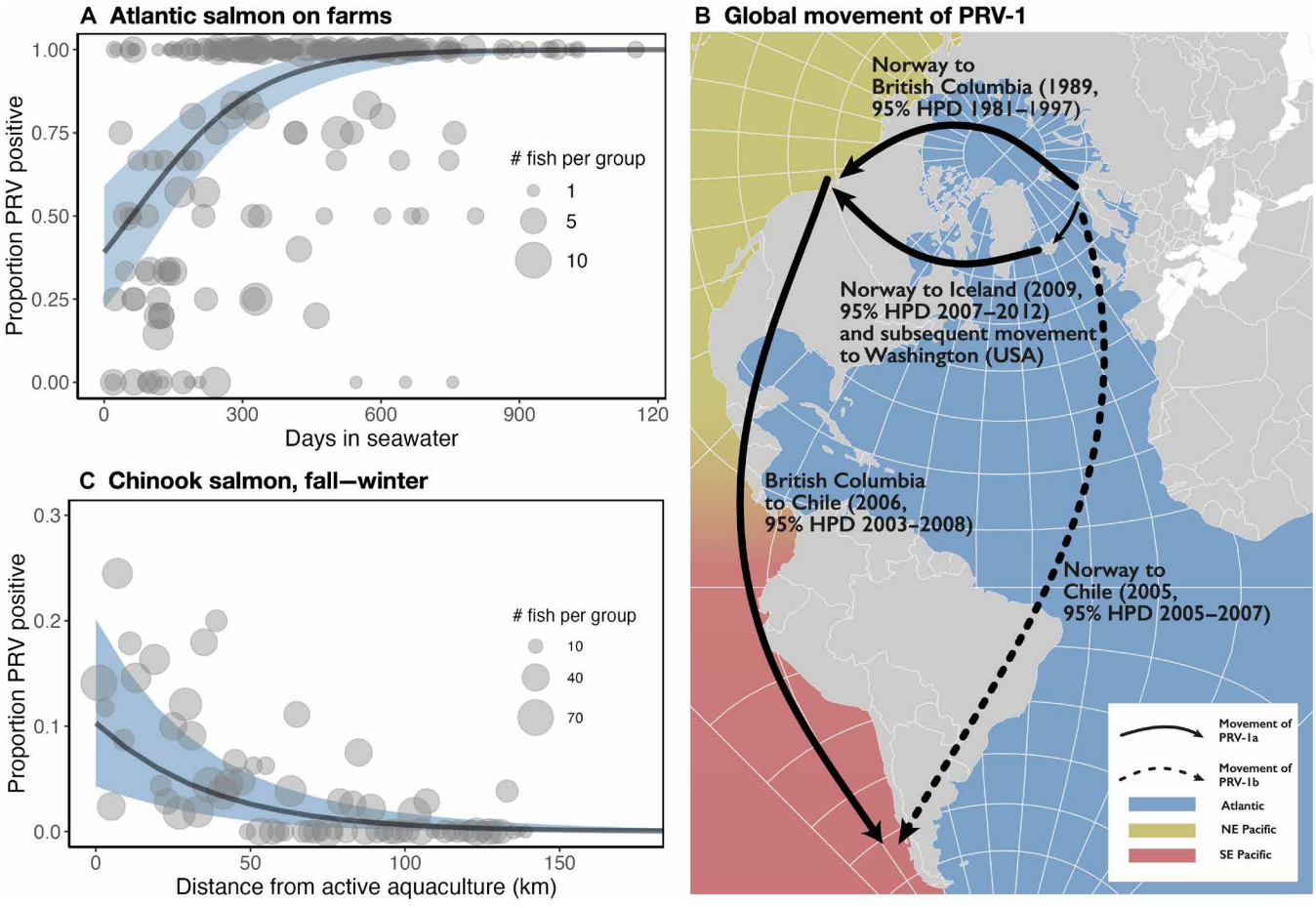

Fig. 4. Transmission dynamics of PRV. (**A**) Almost all farmed Atlantic salmon in BC become infected with PRV-1 over the course of an 18-month grow-out period. (**B**) Schematic representation of the global emergence of PRV-1. Arrows depict estimated translocations of PRV-1 lineages. Movements are determined by a maximum clade credibility time-scaled phylogenetic tree to infer the emergence of the contemporary phylogeographic distribution of PRV-1. (**C**) Probability of PRV infection for first–marine-year Chinook salmon increases closer to active salmon farms in the fall-winter period. Phylogenetic evidence (*8*) also supports ongoing transmission between farmed and wild salmon. Blue symbols in (A) and (C) indicate collections from which samples were sequenced to inform phylogenetic inference. Data and figures are sourced from (*8*).

established into BC Pacific salmon hatcheries. Rather, PRV is mostly absent in BC-origin hatchery and wild Chinook and coho salmon until late in the fall, when it is largely detected in marine-phase fish in salmon farming regions and is mostly the same lineage infecting Atlantic salmon farms (*8*).

In Atlantic salmon, PRV-1 causes heart and skeletal muscle inflammation (HSMI), a disease common on European salmon farms (*84*) that can cause variable mortality (*85*, *86*), from 0 to 20% of the farm population (in the most extreme cases) over the production cycle (*87*). 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*). Although virulence differences among isolates in this challenge study were attributed to mutations in certain viral proteins, phylogenetic placement of PRV-1 lineages was not the ultimate determinant of virulence (*86*). Farmed Atlantic salmon in BC infected with PRV-1a have also been diagnosed with HSMI, but not all Atlantic salmon infected with PRV-1a in BC exhibit HSMI (*77*, *88*), which may suggest the involvement of an environmental or host cofactor (e.g., elevated stress) in disease development and manifestation.

All three strains of PRV (i.e., PRV-1, PRV-2, and PRV-3), including the lineage found in BC (PRV-1a), have also been associated with or shown to cause disease distinct from HSMI in Pacific salmonids (*15*, *78*, *89*–*92*). All three strains can result in rupture of infected blood cells, the primary target of infection (*93*), which can lead to a jaundiced appearance and organ damage from release of toxic levels of hemoglobin (*15*, *78*, *89*–*92*). 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*), but the challenged fish did not progress to exhibit jaundice/anemia. Another challenge study conducted in sockeye salmon concluded that PRV exposure is of little consequence (*97*), although several authors of this review have questioned whether the study design was adequate to reach such a conclusion and if analytical choices made

in the study may underrepresent the true ecological impacts of PRV exposure (*98*–*100*). Together, these findings indicate that PRV-1a causes disease in Pacific salmon, but that either the most severe clinical signs of jaundice/anemia seen in Chinook salmon in BC farms (*77*, *95*) are not readily reproduced by challenge experiments or only a small proportion of individuals exhibit the most severe disease signs (*94*–*96*).

A mounting body of evidence indicates that wild salmon are also negatively affected by PRV infection. Wild BC Chinook salmon infected with PRV-1a in their first year of ocean life have gene expression patterns indicative of immune response to viral infection, while also having lesions that indicate the early signs of jaundice/anemia (*16*). Although the extent of disease progression to jaundice/anemia in wild Chinook salmon would be mediated by similar environmental conditions that can produce it in farmed Chinook (*15*), wild salmon must also contend with ecological realities from which farmed salmon are sheltered. Therefore, as with other salmon pathogens [e.g., (*18*, *101*)], 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. This is consistent with two studies on wild juvenile salmon, which found that PRV is associated with poorer body condition (in Chinook and coho) and survival (in Chinook only) (*19*), and with smaller body size and condition in Chinook (*102*). However, despite these multiple correlations, it is possible—as with all such observational field studies—that increased levels of PRV resulted from poor body condition or that the correlations were due to other unknown causal factors.

Multiple lines of evidence (genetic and epidemiological) indicate that PRV, amplified on BC salmon farms (*8*, *35*, *36*), can spill over to affect wild salmon. PRV infection of wild Pacific salmon has been correlated with exposure to salmon farms and impaired migration success (*18*, *57*). Analyses of PRV distribution among Pacific salmon in coastal BC have found that infections cluster regionally around Vancouver Island including, but not only in, areas with salmon farms (*37*, *102*). PRV is also prevalent in the biological waste (e.g., feces, tissue, and other biological materials) that drifts out of farms and in the outfall of processing plants that spills into the nearby marine environment (*83*), possibly attracting wild fish and facilitating transmission. Genetic analyses indicate continuous viral transmission between farmed and wild salmon, that the number of total PRV infections have increased in recent decades, and that Chinook salmon caught closer to farms are more likely to be PRV positive (*8*).

TENACIBACULUM SPP.

The globally distributed marine pathogen *Tenacibaculum maritimum* has long been known to cause tenacibaculosis in marine fish (*103*) and has recently come to light as posing risk to BC wild salmon (*19*). Tenacibaculosis can cause severe mortalities, and *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*). *T. maritimum* is widespread in Atlantic salmon farms throughout BC (*36*, *38*), where it commonly causes a disease, known as "mouthrot," characterized by acute oral ulcers and plaques. Mouthrot is a particular manifestation of tenacibaculosis, exhibiting a subset of the symptoms observed in diverse fish species after infection by various *Tenacibaculum* species.

Left untreated, farmed fish with mouthrot can die within days (*109*, *110*). Mouthrot is treatable with antibiotics, but treatment appears not to eliminate the pathogen from a farm. *T. maritimum* is detectable via genetic screening of farmed fish through to harvest, and detections are elevated in dead and dying fish for much of that duration (*36*). In addition, in seawater screening, *T. maritimum* eDNA is substantially elevated near active salmon farm sites, compared to inactive sites, and shows one of the strongest associations with active salmon farming of 39 salmon pathogens studied [Fig. 3; (*38*)]. Further, *T. maritimum* can form surface-attached biofilms, which may serve as reservoirs for tenacibaculosis outbreaks in aquaculture settings (*111*, *112*), e.g., possibly after net-washing or mechanical delousing dislodges bacteria (*113*). Thus, although *T. maritimum* is globally common, it appears to be amplified locally by salmon farms in BC (*6*, *48*).

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*). *T. maritimum* detection rates peaked in juvenile Fraser River sockeye as they migrated past the Discovery Islands (Fig. 5A), and spatio-epidemiological models fit to those data suggest that salmon farms in the Discovery Islands are the most plausible source of infection (*9*). In addition, sampling of wild Chinook and coho salmon in their first year of marine residence shows that *T. maritimum* infection is associated with decreased marine survival in Chinook and reduced body condition in Chinook and coho (*19*), one of the most consistent patterns across infective agents studied. Together, this evidence indicates that salmon farms, which elevate levels of *T. maritimum* in the marine environment (*36*, *38*), increase exposure levels and risk of population-level impacts for some species. These risks are likely to increase with climate change, because *T. maritimum* tends to be most common in warmer years (*36*, *38*).

Recently, previously unrecognized *Tenacibaculum* genetic diversity has come to light, with some authors describing the genus as "critically understudied" (*114*). New *Tenacibaculum* species continue to be discovered (*115*), and—relevant to Pacific salmon—multiple species can cause tenacibaculosis (and indeed clinical signs of mouthrot) (*109*, *116*–*118*). Two such species, *T. finnmarkense* and *T. dicentrarchi*, have both been isolated from mouthrot/tenacibaculosis outbreaks on Atlantic salmon farms in BC (*112*, *115*, *116*). The same species have been isolated globally from fish (including salmon) exhibiting tenacibaculosis, and their pathogenicity has been demonstrated in experimental laboratory infections of salmonids (*116*, *118*–*122*). *T. finnmarkense* has caused tenacibaculosis outbreaks in Norwegian and Chilean salmon farms (*120*, *123*). In Norway, these outbreaks have been observed at low water temperatures and in juvenile farmed salmon shortly after transfer into net-pens, suggesting ubiquity of *T. finnmarkense* in the marine environment (*117*). The closely related *T. dicentrarchi* is globally distributed (*114*) and has been identified in Chilean and Norwegian salmon farms as responsible for fin rot, gill damage, and high levels of mortality [60 to 100% in laboratory challenges; (*119*, *124*)] across multiple salmonids (*119*, *124*, *125*). Both *T. dicentrarchi* and *T. maritimum* have been identified as the causative agents of tenacibaculosis in Chinook salmon in New Zealand, demonstrating high levels of mortality [28% and 60 to 100%, respectively; (*126*)] and similar clinical presentations to those observed in tenacibaculosis outbreaks in wild-caught Chinook salmon in BC (*107*). While *T. dicentrarchi* is highly virulent in some hosts (Atlantic salmon, rainbow trout),

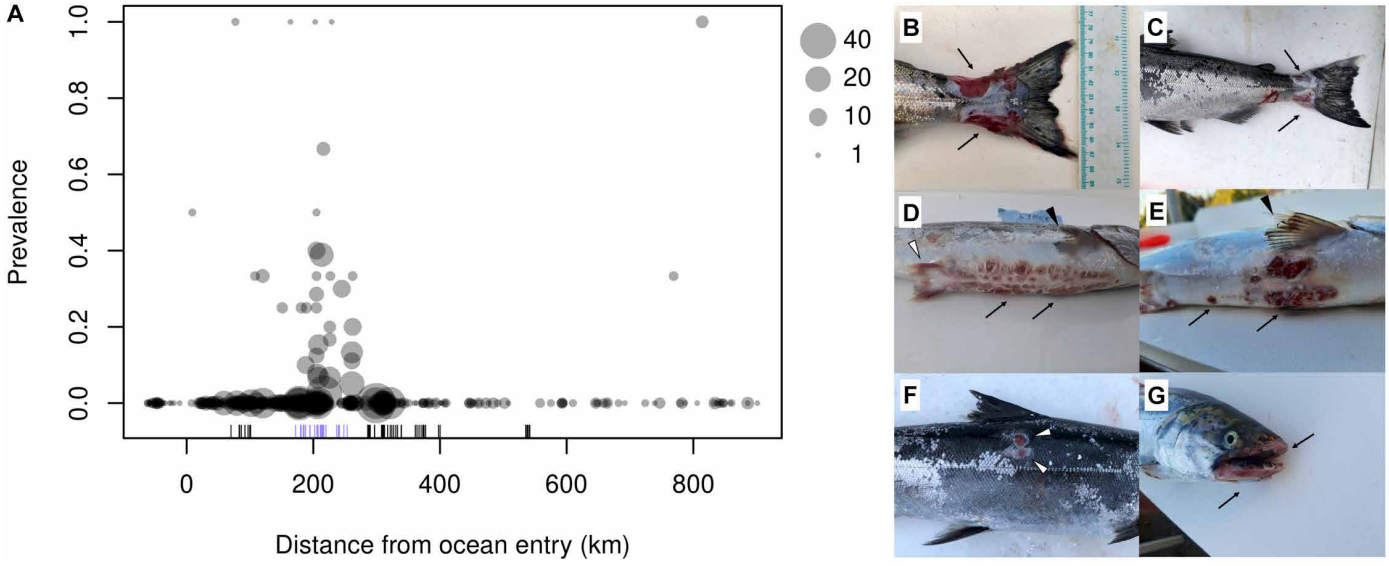

Fig. 5. *Tenacibaculum maritimum* detections and lesions of wild Pacific salmon. Molecular detections of *T. maritimum* in juvenile Fraser River sockeye salmon relative to active Atlantic salmon farm locations along the juveniles' migration route [vertical "rug" dashes along the *x* axis, blue = Discovery Islands salmon farms (**A**)]. *T. maritimum* was the only *Tenacibaculum* species screened in the study. (**B** to **G**) [reprinted from (*127*)] show typical lesions consistent with tenacibaculosis, ascribed to *T. dicentrarchi*, in infected wild-caught Chinook salmon: skin ulcers (B) and erosions (C) on the caudal peduncle (arrows) and belly [(D) and (E), arrows]; fin hemorrhages [(D), white arrowhead]; extensive scale loss associated with ulcerative lesions on the flank [(F), white arrowhead]; and severe erosion with ulceration of the mucosa and skin on both maxilla and mandible [(G), arrows]. Data and plot in (A) are from (*9*); photographs are from (*127*).

including wild and farmed Chinook in BC (*127*), it may have little effect in others, such as coho (*119*). *T. dicentrarchi* may be especially pathogenic during the stress induced by seawater transfer, and it can be harbored in nonsalmonid hosts (*124*, *125*).

SEA LICE

Sea lice (*L. salmonis* and *Caligus* spp.) are a group of ectoparasitic copepods that feed on the skin, muscle, and blood of their hosts (Fig. 6). For salmon aquaculture, sea lice cause losses due to delousing treatments, reduced growth, and survival (*128*). While sea lice naturally infest salmonids and other marine fishes around the world, salmon farms elevate sea-louse infestation pressure for nearby wild juvenile salmon (*56*, *129*–*131*), which lack the fully developed scales, immune systems, and body size that protect adult salmon from these parasites (*132*). In Europe and North America, the primary louse species of concern for wild and farmed salmon is the salmon louse (*L. salmonis*). Meta-analyses of large-scale manipulative field experiments in Europe have shown *L. salmonis* to be associated with reduced survival and recruitment of wild salmon (*20*, *133*), an effect that is stronger under poor natural conditions for survival (*20*).

In BC, the Broughton Archipelago has been the focal region for salmon farm–related ecological research. 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*) have highlighted the need to carefully consider confounding factors and account for spatial and temporal covariation in salmon dynamics when testing for evidence of correlations among salmon farms, sea louse infestations, and wild salmon productivity (*34*). Chum salmon from the Viner River in the Broughton Archipelago, which is centrally located amidst salmon farms but also a watershed heavily impacted by forestry, have collapsed (Fig. 2) but analysis of chum populations in the region do not appear to have experienced louse-associated productivity declines (*135*), possibly due to behavioral interactions between pink, chum, and their coho predators (*135*, *136*). Sea lice on wild juvenile salmon have been associated with salmon farms in the sea-louse reporting zones in BC for which analyses have been conducted, including the Broughton Archipelago (*56*, *129*, *137*), Discovery Islands (*131*, *138*), Central Coast (*130*), and Nootka Sound, Esperanza Inlet and Quatsino Sound (*139*–*141*) (see the "Canadian Government Science Advice on Salmon Disease" section for a brief meta-analysis).

Sea lice influence wild salmon physiology and behavior. Ecological studies in BC indicate that juvenile sockeye salmon infested with higher numbers of *Caligus clemensi* sea lice exhibit reduced feeding success (*142*), growth (*17*), and competitive ability (*143*). Juvenile pink and chum salmon infected with *L. salmonis* show reduced swimming endurance (*144*) and increased susceptibility to predation (*136*). Controlled laboratory trials have shown that sockeye salmon infected with *L. salmonis* experience mortality, skin erosion, scale loss, and high levels of stress (*145*). Cumulatively, these effects from *L. salmonis* have a large physiological effect on juvenile sockeye salmon relative to impacts detected in Atlantic salmon (*146*).

In the Broughton Archipelago, pink salmon populations showed evidence of recovery in the mid-2000s after changes to farm practices and implementation of regulations mandating treatment of sea lice on salmon farms when motile (i.e., adult and preadult) sea lice exceeded three per farmed salmon (*74*). As coastal waters of BC warm, however, models of sea-louse population dynamics indicate that the ability to control outbreaks will diminish due to acceleration of louse population dynamics (*147*). Further, experiments show that effects of salmon-louse infestation on survival, growth, and

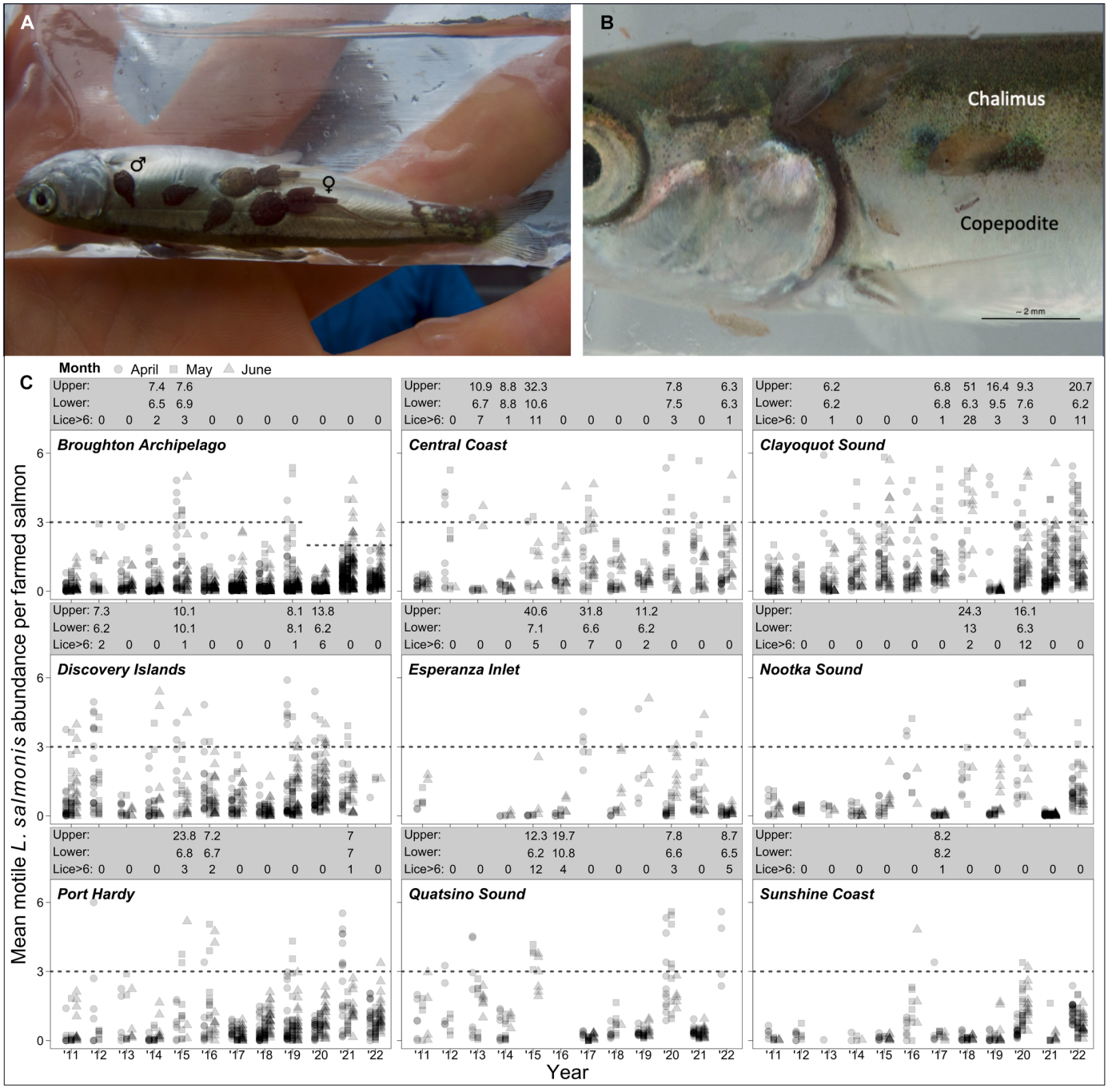

Fig. 6. *Lepeophtheirus salmonis* infestation of wild and farmed salmon. *L. salmonis* infestation of a juvenile pink salmon, showing (**A**) adult male and female lice and (**B**) copepodid and chalimus stage lice. Chalimus lice are tethered by an attachment filament and feed on epidermal tissues and sometimes blood and musculature, producing the lesions around the point of attachment. Motile lice are preadults and adults that can move around on the fish and swim from fish to fish, evading fish predators and searching of mating opportunities. Abundances (**C**) of motile stage *L. salmonis* on farmed salmon from 2011 to 2022 per farm for each sea lice reporting zone in BC. Data are shown for April, May, and June of each year, which is the peak outmigration period for most juvenile wild salmon in these areas, though some populations are resident for longer periods. The horizontal dotted line shows the regulatory limit of three motile lice per farmed fish. Tables above each plot summarize data above six motile lice per fish with the number of counts, and the lowest and highest values of those counts. Sea-louse data were sourced from Fisheries and Oceans Canada Industry sea lice monitoring database available from the Open Data program of the Canadian Federal Government (*200*). See fig. S1 for a plot of these data without the *y* axes truncated.

body condition of Atlantic salmon all become more severe at warmer temperatures (*148*). Similar to other regions, sea lice on BC salmon farms have begun to show resistance to emamectin benzoate (EMB; trade name SLICE), the main treatment used to control sea-louse outbreaks throughout the 2000s and 2010s in BC (*149*, *150*). Detecting and understanding factors affecting treatment efficacy, including the effects of changing environmental conditions, has been difficult given a lack of open data and an apparent bias in industry monitoring (*151*), highlighting the multifaceted challenges for sea-louse management in the near future.

In response to the potential for evolved EMB resistance, alternative sea-louse treatments have been implemented in BC, such as freshwater and hydrogen peroxide baths and physical removal of lice using pressurized water in a "hydrolicer" (*152*). However, these alternative treatment methods are less effective than preresistance EMB and 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*) and potentially enhance spillover of ecto-pathogens shed with mucous and scales in untreated effluent. 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). In addition, these alternative treatments can unintentionally process wild fish such as herring that aggregate within and around net-pens—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*).

INFECTION RISK FROM OTHER POTENTIAL PATHOGENS

In addition to the best-studied pathogens we have focused on above, recent high-throughput studies of >50 species of infectious agents indicate that there are many other infectious agents that are detected in farmed salmon, wild salmon, and eDNA. Many agents in juvenile Chinook, coho, and sockeye cluster along the east of Vancouver Island (*37*), and data from studies of farmed salmon and eDNA show consistent patterns for infectious agents that are prevalent in farmed salmon and water samples near active versus inactive salmon farm sites (table S1 and Fig. 3). We assembled and assessed detection data for agents that have potential transmission risks to wild salmon by meeting four criteria: (i) detection in BC farmed salmon; (ii) detection in Pacific salmon, farmed and/or wild; (iii) more frequent detections in eDNA samples near active salmon farms versus inactive salmon farm sites; and (iv) direct transmission between hosts is possible (i.e., we excluded parasites with complex life histories). The papers from which the data were assembled were found via Web of Science and at the advice of our senior author, K.M.M., who is a coauthor of all of them and runs the laboratory that generated the data. The assembled data indicate that 42 infectious agents have been detected in samples of wild Pacific salmon, of which 37 have also been detected in farmed Atlantic salmon and all of which have been detected in either farmed Chinook salmon or farmed Atlantic salmon in BC.

Beyond *T. maritimum*, four agents stand out for their prevalence in farmed salmon and occurrence in eDNA samples near active versus inactive salmon farm sites—*Moritella viscosa*, *Piscirickettsia salmonis*, Cutthroat trout virus (CTV-2), and Atlantic salmon calicivirus (ASCV). *M. viscosa* and *P. salmonis* are known pathogens of salmon that cause winter ulcer disease and piscirickettsiosis, respectively, which are managed with good husbandry, antibiotics, and vaccines (*157*). They have low prevalence (~1%) in the data on farmed salmon cohorts (table S1), although *P. salmonis* was detected in 22% of dead and dying Atlantic salmon and in 32% of dead and dying farmed Chinook salmon sampled from the DFO farm audit program (table S1). eDNA of *M. viscosa* was detected near 10 of 44 active salmon farm sites versus 1 of 66 inactive sites, whereas *P. salmonis* was detected in eDNA samples near 47 of 59 active salmon farms compared to 32 of 75 inactive sites (table S1). *P. salmonis* and *M. viscosa* have also been found to cluster in wild juvenile sockeye salmon from the Fraser River that migrated near salmon farms (*158*). CTV-2 was common in samples of farmed Atlantic salmon (54%) and detected in eDNA near 10 of 59 active salmon farms versus zero of 75 inactive sites. In Atlantic salmon, CTV-2 infection is systemic but seems to favor the brain, where it is associated with mild neuronal necrosis and degeneration (*159*). In Pacific salmon, CTV-2 has rarely been detected in wild fish, but was detected in 12% of dead and dying farmed Chinook salmon. A laboratory challenge study of CTV-2 in sockeye, pink, and Chinook salmon found persistent infections but no mortality or morbidity, and although histological analysis did not consider brain tissue, kidney, spleen, and heart tissue showed mild endocarditis in Chinook salmon (*160*). ASCV is notable for its 54% prevalence in fish in four cohorts of Atlantic salmon, 76% prevalence in dead and dying Atlantic salmon, and 10% of dead and dying farmed Chinook salmon (table S1). ASCV has rarely, however, been observed in wild Pacific salmon, and it was detected in eDNA in only 3 of 77 sites, all near active salmon farms (table S1). For ASCV, no studies exist on the physiological and ecological effects on Pacific salmon.

CANADIAN GOVERNMENT SCIENCE ADVICE ON SALMON DISEASE

In this section, we analyze how the findings of this review relate to advisory reports of the Canadian Department of Fisheries and Oceans (DFO) that have been produced to inform regulatory decisions on salmon aquaculture in BC. Our purpose is to provide an update on scientific findings published after those reports were produced and to provide advice on salmon species and populations that have not been encompassed in those reports. DFO is the regulator of salmon aquaculture in BC and is responsible for setting parasite and biomass limits on farms and approving license applications. License applications include a fish health management plan and considerations of site location, such as proximity to rivers, which must be more than 1 km. The primary formal mechanism by which DFO provides science advice to inform issues of relevance to fisheries management in Canada, including regulation of the salmon aquaculture industry, is the Canadian Science Advisory Secretariat (CSAS)—a coordinating body that produces advisory reports on analyses and risk assessments conducted by DFO scientists and invited external participants (*28*). CSAS has been criticized for procedural issues underpinning the reports and risk assessments on aquaculture-related topics, including participant selection, peer review, transparency, open data, and reproducibility, which have resulted in reports and science advice that is not fully supported by evidence (*140*, *161*). The evidence we review in this paper outlines additional information needed to fill knowledge gaps from these

CSAS reports, most of which were limited to Fraser River sockeye and salmon farms in the Discovery Islands. Here, we consider a broader scope, all salmon species in all farming regions of BC, and provide some important revisions and updates to DFO's science advice on aquaculture risks to wild salmon.

Through CSAS, DFO has conducted risk assessments of nine pathogens, known to cause disease in farmed salmon in the Discovery Islands region, for their risk to sockeye salmon from the Fraser River: PRV, infectious hematopoietic necrosis virus (IHNV), viral hemorrhagic septicemia virus (VHSV), and the bacteria *Aeromonas salmonicida*, *Renibacterium salmoninarum*, *Yersinia ruckeri*, *T. maritimum*, *P. salmonis*, and *M. viscosa*. Our review is broader, encompassing a wider range of pathogens and all Pacific salmon in all salmon farming regions of BC. Our review is also broader because we focus on pathogen occurrence in farmed salmon, rather than the ability of the pathogens to cause disease in farmed salmon, as the mechanism for creating risk to wild salmon. This latter point is important because it is a pathogen's ability to infect farmed salmon, propagate, and spill over into the environment—rather than its ability to cause disease—that is necessary for a pathogen of farmed salmon to create risks for wild salmon. This is because (i) transmission can occur in the absence of overt disease, (ii) the high abundance of farmed salmon means that spillover to wild salmon can be large even at low prevalence on farms, and (iii) the consequences of infections are likely higher in wild salmon due to the unique challenges they face, such as competition, predation, and migration (*18*), which have the potential to increase mortality in infected hosts.

Of the nine pathogens considered in DFO's risk assessments on Fraser River sockeye, three are among those that we have highlighted for impact on wild salmon (PRV, *Tenacibaculum* spp.) or likely elevated infectious risk near salmon farms (*M. viscosa*), all of which were concluded by DFO to pose minimal risk. The other five pathogens evaluated by DFO were also assessed as posing minimal risk to Fraser River sockeye. These latter five pathogens showed very low or zero prevalence in marine multipathogen molecular screening of farmed salmon, wild salmon, and eDNA (table S1). DFO has not conducted a risk assessment of sea lice, but has produced a recent statistical analysis of sea lice on wild and farmed salmon in four salmon farming regions of BC (*139*) (see below). Below, we highlight some important discrepancies in and updates to the DFO risk assessments for PRV, *T. maritimum*, sea lice, *M. viscosa*, and *P. salmonis*.

In 2019, CSAS assessed the risk to Fraser River sockeye salmon due to PRV transfer from Atlantic salmon farms in the Discovery Islands area as minimal. 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*). The consequences of transmission for Fraser River sockeye were considered negligible, with reasonable certainty for juveniles and reasonable uncertainty for adults, but participants disagreed on the applicability and abundance of the data to support uncertainty estimates. A preceding 2015 risk assessment on PRV to wild Pacific salmon in BC (not just Fraser River sockeye salmon) concluded a low likelihood that the presence of the virus in any life stage of farmed Atlantic and Pacific salmon would have an impact on wild Pacific salmon populations (*162*). The 2015 assessment was focused on whether the BC variant of PRV causes disease in Pacific and Atlantic salmon in BC, concluding that laboratory studies in Chinook, sockeye, and Atlantic salmon and rainbow trout provide evidence that infection with the lineage of PRV found along the West Coast of North America does not cause clinical disease in these species. 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*). The 2019 CSAS risk assessment further concluded that current evidence does not support the view that the lineage of PRV in BC (PRV-1a) causes disease or mortality in sockeye salmon, and that a causal relationship between PRV and jaundice/anemia in Chinook salmon has not been established. As discussed in the PRV section above, recent studies on species other than sockeye that were published after the CSAS risk assessment indicate that PRV-1a is associated with jaundice/anemia in farmed (*15*) and wild Chinook salmon in BC (*16*), that PRV is present in the biological waste that drifts from salmon farms and processing plants into the marine environment inhabited by wild fish (*83*), that transmission occurs from farmed salmon to wild Pacific salmon in BC (*8*, *57*), and that PRV is associated with lower body condition and survival in Chinook and coho (*19*).

DFO's 2020 CSAS risk assessment of *T. maritimum* from Atlantic salmon farms in the Discovery Islands assessed that the bacterium posed minimal risk to Fraser River sockeye (*110*). It concluded that it is unlikely for juveniles and very unlikely for adult Fraser River sockeye salmon to become infected with *T. maritimum* released from Atlantic salmon farms in the Discovery Islands area, but with uncertainties that ranged from high uncertainty to high certainty, respectively. However, new evidence indicates that *T. maritimum* detection rates peaked in juvenile Fraser River sockeye as they migrated through the Discovery Islands (Fig. 5), with the bacteria likely acquired from the cluster of salmon farms in that region (*9*). Further, eDNA analyses indicate that *T. maritimum* is one of the pathogens most elevated near active salmon farms (*38*). The risk assessment further concluded that the impact of *T. maritimum* on Fraser River sockeye salmon is negligible given that modeled mortality of sockeye attributable to *T. maritimum* infection from Atlantic salmon farms was less than 1%, which was made with reasonable uncertainty (*110*). However, no empirically validated models supported such a quantitative prediction, and the low impact was predicated on a low detection rate, an interpretation that subsequent work has shown to be invalid (*9*). Recent work has further shown that *T. maritimum* infection is associated with decreased marine survival in Chinook and reduced body condition in Chinook and coho salmon (*19*). 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.

A recent CSAS Science Response Report (no. 2022/045) evaluating sea lice on salmon farms and wild salmon in BC concluded that the presence of parasitic sea lice on wild juvenile salmon is not significantly associated with sea lice from nearby salmon farms (*139*). That conclusion ignores many studies, reviewed here and elsewhere (*163*), which document a large body of evidence that salmon farms increase sea-louse infection pressure on wild salmon, and that sea-louse infections affect the fitness of individual juvenile wild salmon and the overall recruitment in affected salmon populations. The conclusion of the CSAS report was based on statistical analyses that found no significant association between sea lice on wild juvenile

salmon and sea lice on nearby salmon farms within individual reporting zones. Besides the report ignoring all infection-intensity data from field collections, a key part of the analysis was an unvalidated model for sea-louse infection pressure on wild salmon that accounted for the dispersion of lice from farms to the sampling locations of wild Pacific salmon. As a result, the conclusion of a lack of association between lice on farmed and wild salmon may be the consequence of using a hydrodynamic model that did not account for asymmetric dispersal of lice due to tides and currents (*164*). However, the conclusion of the report was not tenable, even assuming a robust infection pressure model. Applying Fisher's combined test to the results from all four regions, considered separately in the report, indicates an overall significant association between sea louse infection pressure from farmed salmon and sea louse occurrence on wild juvenile salmon (Table 1).

The CSAS assessment of *M. viscosa* from salmon farms in the Discovery Islands found that the infectious agent posed minimal risk to sockeye from the Fraser River. It relied on the seasonality of winter ulcer disease, which is a cold-water disease that occurs in farmed salmon in the Discovery Islands primarily between December and February (*165*). The assessment reasoned that juvenile salmon in BC, including sockeye from the Fraser River, generally do not migrate to sea until after that time period, and so there is no temporal overlap of the disease in farmed salmon and wild salmon migrations. That reasoning, however, does not account for species, like ocean-type Chinook salmon, that remain resident in coastal waters around Vancouver Island (*166*, *167*). Further, eDNA data published just before the risk assessment, but which did not make it into the risk assessment, indicate that *M. viscosa* occurs 3 to 10.5 times more frequently in the marine environment near active salmon farms compared to inactive sites (*38*). The eDNA data were collected March to August, which suggest that salmon farms amplify *M. viscosa* in the marine environment during the migrations of wild salmon, especially during colder years (*38*), although the pathogen does not cause disease in farmed salmon during this time of year. *M. viscosa* has occurred at low prevalence in wild Pacific salmon, and for sockeye from the Fraser River, the few positive detections have come from fish that have migrated near salmon farms (*158*).

The CSAS assessment of *P. salmonis* from salmon farms in the Discovery Islands found that the infectious agent posed minimal risk to sockeye from the Fraser River. It was based on the conclusion, with reasonable certainty, that farmed salmon are unlikely to become infected (*168*). However, the data we reviewed indicate that *P. salmonis* is common in moribund farmed salmon and that *P. salmonis* is elevated in eDNA samples near salmon farms (table S1). The risk assessment's other conclusion, with reasonable to high uncertainty, that there will be minimal risk for *P. salmonis* to subsequently spread within wild sockeye populations or have ecological effects (*168*) is not empirically supported but rather reflects a lack of data to resolve uncertainty. Piscirickettsiosis has been observed in Pacific salmon species (pink, coho, and Chinook) in seawater tanks and net-pen aquaculture environments (*169*). More recent cohabitation experiments in seawater tanks have indicated that pink and chum salmon are susceptible to infection and mortality from *P. salmonis* at temperatures the fish encounter during spring and summer (*170*).

The CSAS risk assessment for VHSV found that transmission from salmon farms in the Discovery Islands to Fraser sockeye was extremely unlikely, given that sockeye appear to not be susceptible to VHSV infection, but that is not the whole story for Pacific salmon. Because of the rarity of the associated disease, transmission events coincident with the brief sockeye migration period are likely to be rare; however, the CSAS risk assessments were mandated to focus on Fraser River sockeye and so did not consider risks to other Pacific salmon species or other fish. Of particular relevance is that risk to herring-dependent species may be higher, especially for Chinook salmon, which often remain resident in nearshore habitats during their first year at sea. Herring congregate around farms, transmission from Atlantic salmon to herring is possible (*171*), and VHSV is virulent in herring (*172*, *173*). As a consequence, Chinook could be affected directly by acquiring infection from infected prey, or indirectly through a decline in prey abundance.

DISCUSSION

Currently, for wild fish, most emerging or re-emerging infectious diseases co-occur with domesticated fish populations (*6*, *174*). This aligns with the view that new or intensified transmission dynamics between wild and domesticated animals is a primary mechanism for the emergence or re-emergence of infectious diseases (*175*). It should therefore not be a surprise that for wild and farmed salmon that share the same marine environment, many infectious agents are also shared, and that some have emerged as production concerns for farmed salmon and conservation concerns for wild Pacific salmon. Our review of data from several high-throughput screening studies

Table 1. Results from (*139*) showing estimates of logistic regression models evaluating the effect of *L. salmonis* infestation pressure from Atlantic salmon farms on the log-odds of the occurrence of sea lice in sampled out-migrating juvenile chum and pink salmon. Clayoquot Sound and Quatsino Sound include chum salmon only, while Discovery Islands and Broughton Archipelago include chum and pink salmon. Fisher's combined test is shown in the bottom row. Data are sourced from the Canadian Science Advisory Secretariat Report No. 2022/045 (*139*).

Region	Coefficient (95% CI)	Odds ratio* (95% CI)	*P* value	χ^2	df
Clayoquot Sound	1.19 (−0.06, 2.43)	3.29 (0.94, 11.36)	0.06		
Quatsino Sound	1.3 (−0.34, 2.95)	3.67 (0.71, 19.11)	0.12		
Discovery Islands	0.57 (−0.03, 1.17)	1.77 (0.97, 3.22)	0.06		
Broughton Archipelago	0.12 (−0.25, 0.50)	1.13 (0.78, 1.65)	0.52		
Fisher's combined value			0.03	16.8	8

*Values greater than 1 indicate increased chances of at least one attached copepodid sea louse in samples of juvenile salmon relative to a unit increase in standardized modeled infection pressure from sea lice on farmed salmon.

for >50 infectious agents in farmed salmon and wild Pacific salmon indicates 42 infectious agents that occur in wild Pacific salmon, 37 of which co-occur in farmed Atlantic salmon, and all of which co-occur among wild Pacific salmon, farmed Atlantic salmon, and farmed Chinook salmon in BC. As our review indicates, three pathogens—PRV, *Tenacibaculum* spp., and *L. salmonis*—have emerged as a conservation concern for wild salmon due to their high prevalence on farmed salmon, strong evidence for transmission from farmed to wild salmon, and strong correlative evidence of ecological effects on the fitness and recruitment of wild Pacific salmon in BC. Several other infectious agents show evidence of high prevalence in farmed salmon and increased occurrence in eDNA samples near active salmon farms, but little else is known of their transmission dynamics or ecological effects on wild salmon.

Aquaculture environments readily enable observation of disease or mortality, as well as subsequent clinical diagnoses and laboratory studies of infection. For wild fish, the same symptoms of disease or mortality may not be easily observable, not due to lack of observation effort, but because other ecological processes, such as predation, remove those individuals from the population before overt symptoms of disease seen in aquaculture or laboratory settings can develop (*18*). The ecological context of wild fish creates an environment that changes the consequences of infection for wild fish as compared to farmed fish, and so the identification and function of disease also differs. For Pacific salmon, many studies have identified ecologically relevant but sublethal effects of infection including increased predation risk (*18*, *101*, *176*, *177*), reduced swimming endurance (*144*), reduced migration success (*57*, *178*, *179*), changes to habitat selection (*180*), and reduced foraging success, body growth, and body condition (*17*, *48*, *142*, *143*).

The spatial spread of salmon pathogens from farms into the surrounding marine environment is not empirically understood for any of the identified species, except for sea lice (*129*), resulting in key uncertainties for management. As one reference point, Atlantic salmon eDNA from salmon farms in the Broughton Archipelago was estimated to be contained relatively locally, with 95% of material ranging from 1.6 km upstream to 3.2 km downstream from farms (relative to a prevailing current) at 2 m depth, and 1.8 to 3.7 km at 8 m depth (*38*). Infectious agents can also spread when industry moves live salmon from freshwater hatcheries to marine net-pens, and among marine sites (*7*, *36*). Regulations prohibit interregional transfers of fish experiencing high levels of mortality or infectious disease outbreaks, but asymptomatic individuals can also carry and spread pathogens. The availability of alternate hosts to complete their life cycle in the nearby environment will limit transmission to wild salmon for myxozoans, whereas more easily transmissible pathogens with a broad host range could potentially spread spatially very quickly (e.g., VHSV and herring). Pathogens may also spread into the nearby environment via net cleaning (*113*), discharge from delousing vessels, and the outfall from processing plants (*83*).

In addition to the risks posed by infectious agents per se, salmon aquaculture environments promote the evolution of pathogen and drug resistance and virulence (*11*, *13*). For example, a study from Norway showed that farm-origin salmon lice were more harmful to their salmon hosts, with higher rates of skin damage, and stronger impacts on host growth when compared to wild origin salmon lice (*181*). Further, sea lice have evolved resistance to multiple treatments on salmon farms (*12*) including EMB in BC (*149*, *150*), which has been the primary chemotherapeutant used to control sea lice in BC. Selection on salmon farms has also impacted bacterial pathogens, increasing virulence (*14*, *182*) and decreasing the effectiveness of a variety of treatment options (*11*, *183*). Thus, it is not surprising that *Tenacibaculum* spp. have also developed resistance to the common antibacterial treatment oxolinic acid, with some strains of *T. maritimum* and *T. dicentrarchi* demonstrating resistance to a variety of antibiotic treatments (*115*, *184*). Similarly, there is some evidence to suggest that aquaculture-specific selective pressures have increased the virulence of PRV-1 in the Atlantic ocean and maintained the resulting lineage of the virus within farms (*185*). There is no reason to expect that salmon aquaculture in Pacific Canada is immune to the selective forces on farms that can drive evolution of pathogen virulence and treatment resistance.

Many strategies are used to mitigate the risk of pathogen spread from net-pen salmon farms, including animal husbandry, antibiotics, vaccines, and area-based management (*186*). The risks to BC's wild salmon of pathogen spread from salmon farms that we have reviewed here exist under the best practices available for net-pen salmon aquaculture. There are 41 salmon farms in BC that have been certified as sustainable by the Aquaculture Stewardship Council despite the issues outlined here, and 28 of these are among the sites in the Discovery Islands and Broughton Archipelago that were recently closed by the Federal Government and First Nations, respectively [Fig. 2, (*187*)]. Some reductions in the risk of pathogen spread from salmon farms in BC may be achievable with more formal area-based management, such as coordinated delousing efforts to minimize sea louse abundances during the outmigration period of wild juvenile salmon; however, the performance of current pathogen control strategies is unlikely to be stable due to the evolutionary dynamics of pathogens (*12*, *13*). Substantial reductions in pathogen risks beyond current practices are unlikely without the advent of new efficacious vaccines and other treatments or a transition to closed containment technology that separates the environment of farmed fish from that of wild fish.

Our review indicates that three species of pathogens—PRV, *T. maritimum*, and sea lice (*L. salmonis*)—are well documented to be amplified by farmed salmon populations in BC, transmitted between wild and farmed salmon in BC, and linked to population-level effects on wild Pacific salmon in BC. There are four further infectious agents common to farmed salmon and that are elevated in eDNA samples near salmon farms. Some are newly described (CTV-2 and ASCV), and their pathogenic potential, transmission dynamics, and effects on wild fish have not been characterized. The other pathogens *M. viscosa* and *P. salmonis* may be considered well managed, from the perspective of farmed salmon health, but they nonetheless likely increase infection pressure and ecological risk for wild salmon. 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. These pathogen and infectious agent risks to wild salmon fit into a broader conservation context for wild salmon in BC of collapsed commercial and Indigenous fisheries, risks to salmon-specialist predators such as southern resident killer whales (*Orcinus orca;* (*188*), and variable levels of salmon population decline that are likely driven by multiple anthropogenic and natural factors, including disease.

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*). For the data we reviewed, the infectious agents that co-occurred in wild and farmed salmon do not necessarily represent viable infections or pathogens that are maintained in those putative host populations. Some of the infectious agents of wild Pacific salmon that we highlighted are well-known pathogens (e.g., *M. viscosa* and *P. salmonis*), whereas others are poorly characterized (e.g., CTV-2 and ASCV). This represents an important knowledge gap to evaluate the risks to wild Pacific salmon. For all of these infectious agents, further work could help evaluate the realized transmission risk to wild salmon. Fruitful topics of study would be the relationship between molecular occurrence and minimal infectious dose, the potential for acquisition of infection by wild salmon in the environment of amplification (*35*), and the ecological effects of infection on mortality-related processes of wild salmon such as predation, competition, migration, and environmental stress. On the basis of past global findings, some risks will prove to be minimal and others will prove to be serious.

This review can be informative in several respects to Canadian science advice and policy on salmon aquaculture and fish health. The DFO, via the CSAS, has conducted risk assessments on nine pathogens and an analysis on sea lice (*110*), but for all except sea lice, they were mandated to limit those assessments to Fraser River sockeye and salmon farms in the Discovery Islands region. As reviewed above, data from many recent studies and reanalysis of the CSAS sea lice report indicate that revisions and updates are needed (e.g., for the sea lice report and the risk assessments on PRV, *T. maritimum*, *M. viscosa*, *P. salmonis*, and VHSV). There are no comprehensive risk assessments for all species of salmon in BC with respect to sea lice or the pathogens that we have highlighted in this review. The CSAS risk assessments for Fraser River sockeye do not reflect risks to other species in other areas, particularly pink, chum, and ocean-type Chinook salmon that migrate to sea at small body sizes and remain in coastal waters for several months, and particularly ocean-type Chinook that are resident in coastal BC waters during their first year (*166*).

The context of risks from salmon farms in BC is changing. Pathogen risks from salmon farms to some southern BC wild salmon populations (including in the Fraser River) have been partially mitigated by the closure of most salmon farms in shíshálh First Nation territory (Sechelt Inlet), the Discovery Islands, and the Broughton Archipelago, but disease interactions between wild and farmed salmon still occur in areas where salmon farming remains. Release of pathogens detected in the effluent from farm salmon processing plants into marine waters may be another source of infection for wild fish (*83*). While DFO plans to transition away from net-pen aquaculture in BC (*189*), of the options being considered, only full closed containment systems fully contain disease risks to wild Pacific salmon. Semiclosed containment or area-based management may improve some aspects of sea-louse management or disease control, but will continue to allow for the exchange of seawater, and infectious particles in that water, between farms and the surrounding environment.

Globally, as aquaculture continues its expansion to meet seafood demand, our review highlights that net-pen production facilitates pathogen interactions between wild and farmed fish that can create conservation risks for wild fish. With the intensification of net-pen production and its expansion to new species and new regions, it should therefore be anticipated that new disease dynamics can emerge and may require management intervention. Looking ahead, the risks of pathogen spread from farmed salmon to wild Pacific salmon in BC will likely continue to change due to changes in disease management of farmed salmon, aquaculture regulations, climate, and pathogen evolution. With climate change, for example, outbreaks of sea lice are expected to worsen (*147*, *190*) but risks of coldwater disease from *M. viscosa* to wild and farmed salmon may diminish. Geographic shifts of pathogen ranges and increased physiological stress in salmon may exacerbate many [though not all; (*191*)] existing salmon disease risks as the climate warms (*18*, *192*). Selection for drug resistance in aquaculture environments will continue to challenge pathogen management on salmon farms and influence the associated risks to wild Pacific salmon. For example, drug resistance in sea lice has shifted louse control to mechanical delousing, which removes protective mucus and scales in which surface bacteria and parasites may also be sloughed at high densities into the water column, creating new risks not yet considered by regulators. Continued surveillance that generates accessible, transparent, and unbiased data will be important to track and further evaluate pathogen risks to wild Pacific salmon in BC. For many of the infectious agents we have highlighted, the physiological effects on wild Pacific salmon are not well characterized, and there are few if any data on the agents' effects on ecological processes known to be important for wild Pacific salmon, including foraging, growth, competition, predation, and migration. Addressing these knowledge gaps will be important to resolve uncertainties regarding pathogen risks of salmon farms to wild Pacific salmon in BC, but need not preclude precautionary management. For PRV, *Tenacibaculum* spp., and sea lice, the accumulated evidence indicates that pathogen management and regulations are needed now to mitigate risks of salmon farms to wild Pacific salmon in BC.

Supplementary Materials

The PDF file includes:

Fig. S1
Legend for table S1
References

Other Supplementary Material for this manuscript includes the following:

Table S1

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