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Disease risks associated with open-net pen aquaculture in B.C.

"More than a decade of independent science has unequivocally linked open-net pen salmon farms to risks for wild Pacific salmon in British Columbia. Evidence shows that high-density farms can transmit and amplify harmful parasites and pathogens into critical migration paths of wild salmon. Canada's commitment to transition away from open-net pen farming in B.C. by 2029 represents a decisive step toward addressing a major human-driven threat to wild Pacific salmon." - Michael Meneer, CEO and President, Pacific Salmon Foundation

Launched in 2013 by the Pacific Salmon Foundation (PSF), Fisheries and Oceans Canada (DFO) and Genome B.C., the Strategic Salmon Health Initiative (SSHI) positioned PSF as a global leader in salmon health research. The ground-breaking SSHI research, led by research scientist Dr. Kristi Miller-Saunders, formerly with DFO, brought new understanding of how infectious agents affect the health of wild Pacific salmon, and the findings informed the Government of Canada's commitment to transition away from open-net aquaculture in B.C. by 2029. The PSF Salmon Health Program builds on the SSHI with the same scientific team continuing to study disease and other stressors affecting B.C.'s wild salmon. The following report outlines disease risks associated with open-net salmon aquaculture in B.C. as identified through nearly a decade of independent scientific research.

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Introduction

Salmon farming operations present many risks to wild salmon in B.C. This summary focuses on the possible impacts to wild Pacific salmon related to pathogens, including sea lice. It is not an exhaustive review of the potential risks. (For a more comprehensive review of salmon farming, please refer to the most recent Seafood Watch assessment^[1].)

Most of the evidence we summarise below relates to open-net salmon farming in B.C., but we note that this information fits within a global context of wild-salmon declines widely associated with open-net salmon aquaculture^[2]. Our research into disease covers a variety of pathogens and infectious agents. Many of these are newly discovered, or understudied in the context of wild



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Pacific salmon. This review focuses only on three pathogens for which we have the most information: sea lice, *Tenacibaculum maritimum*, and Piscine orthoreovirus (PRV), much of which was previously reviewed by Krkosek et al.^[3].

In general, the large numbers of densely packed fish on salmon farms present ideal conditions for amplification of viruses, bacteria and parasites^[4–6] (collectively “infectious agents”), offering a source of transmission to wild Pacific salmon that would not otherwise exist. Also, because farms can amplify, or “breed,” infectious agents^[4,5], associated farm risks are likely to be elevated *whether or not* any given agent has always been present in B.C. (such as sea lice) or was introduced via the farms themselves (such as PRV). Farms can raise the levels to which wild salmon are exposed, regardless of pathogen origin.

Sea lice

An established body of evidence links salmon lice, *Lepeophtheirus salmonis*, with impacts on salmon globally^[7,8]. Sea lice are native parasites whose transmission between farmed and wild salmon allows for wild juvenile salmon to be infested at abundances far above natural levels^[9,10]. Many peer-reviewed papers have reported positive relationships between infestations on farms and on wild juvenile salmon in B.C. (e.g.^[11,12]), the most recent of which was published by DFO authors^[13]. In B.C., most evidence of the impacts of sea lice comes from the Broughton Archipelago, where sea lice on salmon farms have been associated with reduced survival in pink and coho salmon^[12,14,15]. In contrast, chum salmon populations in the Broughton do not appear to have declined as a result of sea lice from farms^[16].

Sea lice also impact wild salmon physiology and behaviour. Ecological studies indicate that juvenile salmon infested with more sea lice experience increased predation^[17] and have reduced competitive ability^[18], feeding success^[19], growth^[20] and swimming endurance^[21]. Controlled lab trials provide causal evidence that is also compelling: sockeye salmon infected with salmon lice experience mortality, skin erosion, scale loss, and higher levels of stress^[22]. Cumulatively, these effects of sea lice constitute a “profound physiological impact” on sockeye salmon relative to those seen in Atlantic salmon^[23]. Further, new evidence of pathogen detections in sea lice have demonstrated a potential interaction between sea lice and pathogens such as *Tenacibaculum*^[24].

While some wild salmon populations began to recover after farm management practices improved, success appears to depend on the ability of salmon farms to manage infestations of sea lice^[12]. Outbreaks of sea lice on B.C. salmon farms are, however, expected to become more frequent and severe as the ocean warms^[25], as are the impacts of sea lice on their hosts^[26]. Inaccurate reporting of sea-louse counts on farms may further exacerbate the challenges of sea



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louse management^[25,27]. Sea lice in B.C. have developed resistance to the main chemical used to control outbreaks (emamectin benzoate, also known as EMB or SLICE®)^[28,29], indicating that sea lice will be even more difficult to manage in B.C. moving forward. Indeed, severe sea-louse outbreaks near salmon farms on the Central Coast, in Clayoquot Sound, and in the Northern Salish Sea—where the PSF Juvenile Salmon Sampling Program continues to monitor juvenile salmon—raise serious doubts about whether sea-lice can be effectively managed over the long-term.

To combat drug-resistant sea lice, the industry relies heavily on physical removal of lice, using a so-called “hydrolicer,” which employs pressurized water. This Norwegian technology is touted as an ecologically sustainable solution by industry, but it is less effective than pre-resistance EMB^[30,31]. The approach can be highly stressful on the fish, removing mucous and scales required to fend off infection and making them more vulnerable to disease^[31,32]. In addition to reports of herring ‘bycatch’ in mechanical delousing systems^[33], there are concerning observations of sea lice removed by mechanical means being released to the marine environment, and these appear capable of dispersing and infecting vulnerable juvenile wild salmon (A. Morton and M. Bartlett unpubl. data).



Figure 1. Juvenile salmon heavily infested with sea lice near open-net pen salmon farms. Left: Fish caught in the Discovery Islands, B.C., before salmon farm removal (image credit: Tavish Campbell). Right: Fish caught during the 2025 field season for PSF’s juvenile-salmon monitoring program near Port Alexander in the Queen Charlotte Strait.



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Tenacibaculum

The globally distributed marine bacterium *Tenacibaculum maritimum* has recently come to the fore as posing a substantial risk of harm to sockeye, Chinook, and coho salmon. More recently, previously unidentified genetic diversity within *Tenacibaculum* has been revealed, with new species being discovered^[34], which are frequently associated with disease outbreaks or have been shown to cause disease^[35-40]. Multiple species within the *Tenacibaculum* genus are widespread on Atlantic salmon farms in B.C., where they can cause acute fins and skin ulcers, including oral ulcers (known as "mouth rot" disease), and death within days of ocean entry^[35,41,42]. Although mouth rot is treatable with antibiotics, *Tenacibaculum* is detectable via genetic screening in farmed fish throughout their time on farms, displaying elevated levels in dead and dying fish for much of that time^[43]. Further, in seawater screening, *Tenacibaculum* environmental-DNA has been almost exclusively detected near active versus inactive salmon-farm sites, showing one of the strongest associations with active salmon farming of 39 salmon pathogens studied^[44]. A 2024 environmental DNA study confirmed these patterns in B.C., showing highly elevated concentrations of *Tenacibaculum* in waters near operating net-pen farms across multiple seasons^[45]. Thus, although *Tenacibaculum* is globally distributed, it very much appears to be associated with and amplified by salmon farms.

Note of clarification: while, in some instances, mouth rot was the only physical evidence of disease caused by *T. maritimum* in farmed Atlantic Salmon in B.C., this type of lesion is generally less common in Pacific salmon; instead, the disease caused by *Tenacibaculum* in Pacific salmon is referred to as "tenacibaculosis", with ulcers in the mouth being just one of the symptoms observed. For instance, new findings show oral detection of *Tenacibaculum* colonizing the mouth of coho^[39], and Chinook^[46]. Tenacibaculosis can cause severe mortalities, and is known to have caused substantial health issues for Pacific salmonids in California^[47], Chile^[48,49], New Zealand^[50,51], B.C.^[46] and Alaska^[52]. While infections on-farm can sometimes be managed with antibiotics, wild salmon do not have access to such veterinary care. A recent study has raised further concern by identifying antimicrobial resistance in *T. maritimum* isolates from B.C. farms^[53].



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Figure 2 Disease lesions associated and caused by *Tenacibaculum* infection in Chinook salmon: ulcers on the caudal peduncle of wild-caught chinook (left). Mouth rot is a specific form of tenacibaculosis—the disease caused by *Tenacibaculum*—that has exclusively been identified in B.C., particularly in farmed Atlantic salmon, but also observed in chinook salmon (center) experimentally exposed to *T. maritimum*. Note the yellow plaques in the mouth (black arrows). Large, deep ulcers (white arrow) and fin erosions (black arrows) in chinook salmon experimentally exposed to *T. dicentrarchi* (right).

While a 2020 CSAS review of *T. maritimum* concluded that transmission from farms posed a “minimal risk” to Fraser River sockeye in the Discovery Islands region^[42], several key shortcomings undermine that result^[54,55] and ‘scientific uncertainty’ surrounding the CSAS conclusions have been affirmed as evidence during the court rulings surrounding the transition away from open-net pen farms^[56]. First, technical flaws in details of the risk assessment resulted in unduly reducing the assessed risk (PSF’s Andrew Bateman was a participant in the relevant CSAS review meeting). Second, the risk assessment was *very* narrowly focused on only sockeye salmon, from only the Fraser River, considering only impacts from Discovery Islands salmon farms; the overall focus should be on the risk from all B.C. salmon farms to all B.C. wild salmon. Third, much new relevant evidence exists^[57,58], including a suite of laboratory disease challenge studies confirming that *T. maritimum* causes disease in Pacific salmon, which were unavailable at the time of the CSAS risk assessment. Instead, DFO’s disease assessment process focuses on the spurious absence of mouth rot in Pacific salmon to conclude that the agent is not “likely to cause disease in wild fish populations”^[55]. This statement had been disproved by the recent challenge experiments carried out by PSF and DFO (amongst others e.g. Nowlan et al.^[39]). These studies confirmed the pathogenic potential of B.C. strains of *Tenacibaculum* in Pacific salmon species (i.e. coho, Chinook, sockeye and steelhead), and also demonstrated that *Tenacibaculum* infection can result in mouth ulcers consistent with those observed in the field during mouth rot outbreaks in Atlantic salmon farms and in captive, wild-caught Chinook^[46].



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Prior to the removal of farms in the region, our research found that *T. maritimum* detection rates in juvenile Fraser River sockeye peaked as the fish migrated past the Discovery Islands^[58]. Moreover, updated spatial modeling across the B.C. coast found a consistent association between high *T. maritimum* loads and proximity to farm sites^[59]. Spatial/epidemiological models, fit to *T. maritimum* incidence data, suggest salmon farms in the Discovery Islands are the strongest source of *T. maritimum* infection along the Fraser River sockeye migration route^[58]. Further, population-level sampling of wild salmon in their first year of marine residence, shows that *T. maritimum* infection is associated with decreased marine survival for Chinook and reduced body condition (“skinny” fish) in Chinook and coho^[57]; this is one of the most consistent patterns across infective agents we studied^[57]. Altogether, this evidence suggests that salmon farms elevate levels of *T. maritimum* in the marine environment, salmon become infected with *T. maritimum* as they pass farms, and may suffer population-level impacts due to *T. maritimum* infection^[57].

Though previously less well-known than *T. maritimum*, the closely related species *Tenacibaculum dicentrarchi* has emerged as a pathogen of concern for cultured and wild Pacific salmon in B.C.^[45,46]. *T. dicentrarchi* is frequently detected on farms (often alongside *T. maritimum*), where it has been linked to outbreaks of *Tenacibaculosis*^[45,60,61]. While mouth rot remains a common (but not exclusive) symptom to Atlantic salmon caused by *T. maritimum*, both *T. maritimum* and *T. dicentrarchi*, have been shown to cause disease in Chinook salmon^[51](Figure 2) Challenge studies carried out by our own group have confirmed the pathogenic and lethal potential of B.C. strains of *T. maritimum* and *T. dicentrarchi* to Pacific salmon species. Moreover, genetic screening of B.C. wild salmon has shown that *T. dicentrarchi* is more frequently detected in fish near active salmon farms, mirroring patterns observed for *T. maritimum*^[3]. Although less is known about its precise contribution to population-level mortality, the spatial correlation with farm proximity, co-occurrence with elevated environmental stress and confirmed pathogenicity in laboratory trials suggest that *T. dicentrarchi* is likely to contribute meaningfully to disease burden in wild Pacific salmon.



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Piscine orthoreovirus (PRV)

PRV is a virus originating from the Atlantic Ocean. In Europe, it commonly causes disease issues on farms^[62]. The virus was introduced to B.C. with the advent of Atlantic salmon farming in the region (Figure 3)^[63]. It is now extremely common in Atlantic salmon on farms in B.C. Controlled laboratory challenge trials conducted in Norway and Canada have established that the lineage of PRV in B.C. can cause lesions diagnostic of the disease Heart and Skeletal Muscle Inflammation (HSMI)^[64,65], and farmed Atlantic salmon in B.C. infected with PRV can develop HSMI^[66,67]. A recent and soon-to-be-published study reveals that HSMI outbreaks on farms are relatively common in B.C., in contrast to industry and DFO reporting.

Rather than its effect on the health of farmed fish themselves, it is PRV's potential impact on B.C.'s Pacific salmon species that is of primary concern. PRV is amplified by salmon farms, and spills over to wild Pacific salmon^[63]. Various lineages of PRV have been associated with (and in some cases shown to cause) disease manifestations in Pacific salmon species, which are distinct to the ones observed in Atlantic salmon. In Pacific salmon, PRV infection can result in rupture of the blood cells, which can lead to liver and kidney failure^[68-72]. The lineage of PRV in B.C. has been tightly associated with the disease "jaundice/anemia" on Chinook salmon farms in B.C.^[68], and similar pathology has been observed in wild Chinook salmon^[73]. To date, PRV infects nearly all farmed Atlantic salmon in B.C. by the time they are ready for harvest^[43,74], and findings from our research group indicate that resident wild Chinook salmon are more likely to be infected with PRV the closer they are to a salmon farm^[63]. The same work also shows, via genetic methods, that PRV regularly transmits between farmed and wild salmon, and that the number of PRV infections has increased in recent decades. Together, this evidence paints a picture of a growing risk posed by PRV to Chinook. Further, as with *T. maritimum*, correlational analyses tie PRV to poorer survival in Chinook and coho stocks^[57].

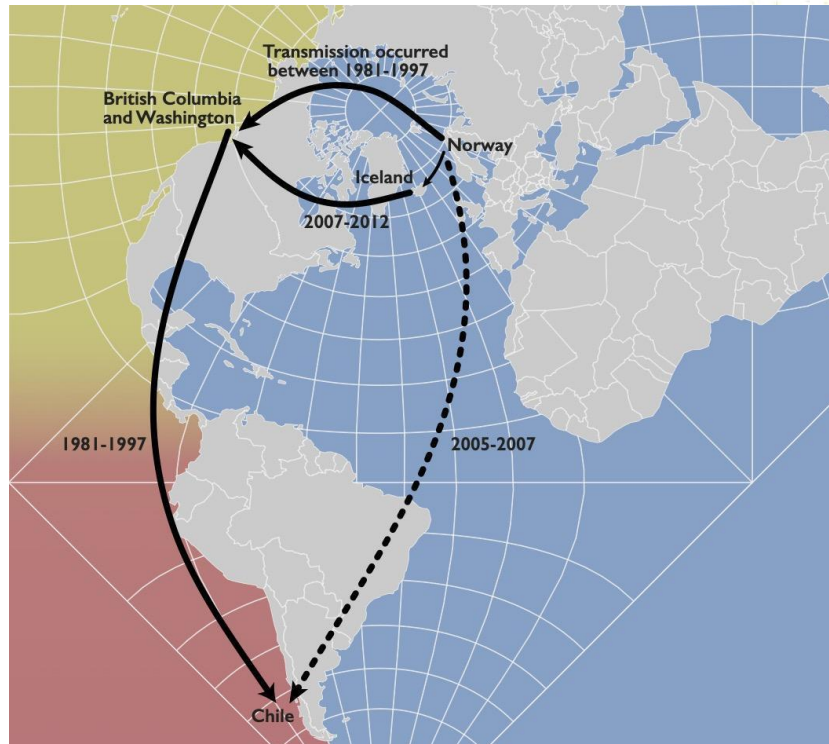


Figure 3 Schematic representation of the global emergence of PRV-1. Arrows depict estimated translocations of PRV lineages. Movements were determined by a phylogeographic analyses of PRV genome sequences^[63].

The salmon farming industry relies on movements of live salmon from freshwater hatcheries to marine net pens, and in many cases, between marine sites during the marine “grow-out” period. While regulations prohibit inter-regional transfers of fish experiencing high levels of mortality or infectious disease outbreaks, DFO does not currently assess PRV or *Tenaibaculum* as ‘disease agents’^[55], and regardless, even asymptomatic individuals can carry and spread pathogens. Pathogens are known to evolve increased virulence within aquaculture settings^[75,76]. If higher virulence pathogens were to evolve or be introduced, movements between farms could quickly spread the new strains across the southern B.C. coast, enhancing risk to both farm and wild populations.



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Salmon farm removal and pathogens

The removal of salmon farms from key migration corridors provides an opportunity to directly evaluate the relationship between aquaculture operations and pathogen exposure in wild salmon. In British Columbia, several recent farm closures—most notably in the Discovery Islands region—have created natural experiments that allow comparison of infection patterns before and after farm removal. While two recent studies^[77,78] have been used to support industry claims that the removal of salmon farms does not influence sea-louse infestation of wild Pacific salmon, these findings are:

1. published by industry-associated authors, including some that have been compensated via industry contracts,
2. in contradiction to the many studies showing the link between sea-louse infestations on farmed and wild salmon, including a recent study by some of the same authors^[13], \
3. not supported by any formal analyses,
4. interpreted in the context of a single year (2024) without regard for year-to-year coastwide trends or long-term data in the region,
5. influenced by local increases in forage-fish populations due to the conflation of multiple sea-louse species,
6. depend on data whose collection is paid for by the salmon farming industry, and
7. strongly influenced by low sample sizes and poor sampling design.

Our collaborative research group continues to monitor pathogen dynamics in wild salmon across British Columbia. The recent removal of salmon farms from the Discovery Islands (DI), alongside their continued operation on the west coast of Vancouver Island (WCVI), provides a spatial contrast that can be used to investigate the influence of aquaculture on pathogen exposure in wild salmon populations, particularly in combination with potentially interactive seasonal environmental stressors like thermal stress and food deprivation.

Using pathogen screening data from juvenile salmon and environmental samples, we plan to map pathogen prevalence and load relative to the spatial distribution of salmon farms. This work aims to test for associations between salmon farms and pathogen burden in wild fish, while accounting for other ecological and environmental drivers of infection. Together, these analyses will help clarify the extent to which salmon farms contribute to pathogen exposure along early marine migration routes and provide empirical evidence to inform ongoing monitoring and management during the transition away from open-net pen aquaculture in British Columbia.



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Note on semi-closed containment aquaculture systems

In response to challenges related to sea lice, the salmon farming industry in Norway has developed “semi-closed containment systems” (SCCSs)^[79]. The concept is that cultured fish are separated from the natural environment by a physical barrier, and water is drawn into the system from depth, aiming to avoid sea lice and harmful algal blooms, which are more common in surface water. Such systems have been shown in certain cases to provide protection against sea louse outbreaks within pens^[80]. In B.C., similar systems have been suggested as possible tools for transitioning away from open-net farms.

However, since large volumes of water are pumped into and out of the system, SCCSs do not provide direct protection from transmission of infectious agents (particularly viruses and bacteria) between farmed and wild fish^[79,81]. That is to say, farmed fish are not protected by infectious agents present in the water pumped inside the barrier, and wild fish are not protected by the water eliminated by the SCCS pens, which could contain farm-amplified pathogens. For instance, semi-closed systems may inadvertently heighten the risk of certain disease outbreaks, including amoebic gill disease^[82] as well as ulcerative diseases^[81,83–85]. Such diseases may be more easily transmitted to farmed fish through deep-water intake systems, where some pathogens are present in higher concentrations^[85,86]. Further, within these systems, there is potential for increased risk of disease outbreaks due to stressful environmental conditions (poor or fluctuating water quality, along with the development of biofilms and biofouling, and the accumulation of organic matter) that can result in increased pathogen loads and potentially severe disease outbreaks^[81,87–89].

A SCCS trial by Cermaq in Clayoquot Sound suffered die-offs, which resulted in the trial being ended early, and although the industry reports that there were no overt signs of disease, evidence of the cause has not been made public^[90]. Nevertheless, due to the lack of available data on this issue, it is precautionary to assume—until future studies prove differently—that SCCSs are not risk-free when it comes to pathogen transfer potential.



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