Chapter 4 • Summary of decline-related evidence

Although it is common to speak about Fraser River sockeye salmon as though they are a singular grouping of genetically identical fish that behave in a uniform manner, the truth is far different. Fisheries managers have traditionally clustered Fraser River sockeye into "stocks," identified principally according to their natal rivers or lakes distributed throughout the watershed. Nineteen such stocks have been the subject of most studies and monitoring. More recently, under the 2005 Wild Salmon Policy, the Department of Fisheries and Oceans (DFO) has grouped Fraser River sockeye into 20 to 30 Conservation Units (CUs) based on genetic differences. Indeed, a CU is defined as "a group of wild salmon sufficiently isolated from other groups that, if extirpated is very unlikely to re-colonize naturally within an acceptable timeframe, such as a human lifetime or a specified number of salmon generations."[1]

In addition to this genetic diversity, Fraser River sockeye exhibit significant behavioural variation. They all begin life in freshwater, grow to maturity in the North Pacific, and return to freshwater to spawn. However, some spend one to two years in nursery lakes while others do not; some migrate out through the Fraser River in days while others spend several months in the estuary; some migrate north through the Strait of Georgia while others migrate south through Juan de Fuca Strait; most return to their natal streams to spawn in their fourth year while others do so in their third or fifth year; and some move directly from the Strait of Georgia into the Fraser River while others hold off at the mouth of the river for four or five weeks before moving upstream. Fisheries managers also group returning adults according to four run-timing groups, depending on when during the summer months they begin their return migration. (For a more detailed discussion of run-timing groups, see Volume 1, Chapter 5, Sockeye fishery management.)

The decline in Fraser River sockeye that triggered the establishment of this Inquiry in 2009 can fairly be described as "a decline" when all stocks are studied on an aggregate basis. However, when individual stocks are examined, important variations in productivity (recruits returning per spawner) are evident. To complicate matters further, some (but not all) Fraser River sockeye stocks show dramatic variations in abundance over a four-year cycle (a pattern called "cyclic dominance") that consists of a dominant year, followed by a sub-dominant year, followed by two much less productive years.

To these fascinating variations in Fraser River sockeye themselves must be added similarly complex variations in freshwater and marine habitat, environmental conditions, and the myriad stressors that affect sockeye differently, depending on the geographical area and the life stage under consideration. Moreover, exposure to stressors in one life stage may not reveal their effects until a later life stage, and stressors may interact in different ways at different life stages.

In light of these complexities, it is prudent to embark on this analysis bearing in mind the caution voiced by the authors of Technical Report 1A, Enhancement Facility Diseases:

> It is tempting to think of a cause as a single entity, event or condition which inevitably leads to a specific outcome. This is rarely the case in biomedical situations, especially when population health and disease are being considered. The presence or absence of a disease typically requires a complex interplay of factors. When referring to wildlife populations, Holmes (1995) said, "Looking for a single, consistent cause for population regulation is not only wishful thinking, but also hinders our efforts to understand population dynamics. Population regulation is not only multifactorial, but interactions among those factors are important; single-factor experiments can miss important interactions. In addition, the ecological context consistently changes, so that regulatory processes track a moving target; experiments can have different results if the context differs."[2]

In the pages that follow, as much as the evidence allows, I examine the various stressors affecting Fraser River sockeye salmon according to their different life stages, adopting a five-life-stages categorization.

This chapter constitutes a summary of the evidentiary record related to the decline that is before this Commission. It is intended to accurately reflect what the researchers wrote in their reports and what witnesses said in their testimony. It should not be assumed that I necessarily agree with or endorse everything set out in this chapter of the Report. Rather, it is a summary of what I read and heard. My findings and conclusions respecting the causes of the decline of Fraser River sockeye salmon will come later, in the following chapter of this volume.

Life stage 1: incubation, emergence, and freshwater rearing

Incubation

After the long return journey to her spawning grounds, the female Fraser River sockeye selects a site for the deposit of her eggs (a redd), digs a nest in the gravel substrate, and deposits 500 to 1,100 eggs, which are simultaneously fertilized by an accompanying male or males. She then covers the eggs by further digging and repeats the digging and spawning process up to several times. Finally, she covers the completed redd, now containing two or several nests of eggs, and then guards the redd site until near death. The eggs develop in the gravel during the winter. In the early spring, after about five months' incubation, alevins emerge from the eggs. A pronounced yolk sac suspended below the body provides nourishment for the next six to 10 weeks, depending on water temperature. During this period, the alevins remain in the gravel for protection from predators and because they are poor swimmers.

Fraser River sockeye salmon are particularly vulnerable during this incubation period. The mortality rate during this seven-month period can be 80 to 86 percent.[3] In the case of a spawning female that lays 3,000 eggs, 2,580 may die during incubation or soon thereafter.[4] Mike Lapointe, chief biologist, Pacific Salmon Commission, identified the following naturally occurring stressors that contribute to egg and alevin mortality:

- Redds may be disturbed or destroyed by later-spawning females, an action that may expose the eggs within to a variety of stressors.
- High water flows may lead to scouring of redds, exposing the eggs within.

- Low water levels in natal streams and lakes can lead to desiccation (dehydration) or freezing of eggs.
- The eggs or alevins may suffocate as a result of the deposition of fine sediment in the gravel, or because of low oxygen levels in the water.
- The eggs or alevins may be exposed to diseases or parasites.
- Predators, either birds or other fish species, may attack eggs or alevins within the redds.[5]

I turn now to a consideration of the stressors that may have caused or contributed to the recent decline during this life stage.

Predation

The authors of Technical Report 8, Predation, Dr. Villy Christensen and Dr. Andrew Trites, stated that several factors must exist before a potential predator can be deemed to have a significant impact on the decline in survival rate of Fraser River sockeye. These factors include:

- The prey and predator must overlap in time and space.
- The prey has to be eaten or preferred by the predator.
- There has to be a sufficient abundance of the predator.
- The abundance of the predator must have been increasing in recent decades, or there must be indications that the predator may have shifted to feed more on sockeye.[6]

The researchers reported that cutthroat trout are known to specialize on salmon eggs between October and January.[7] However, they noted that little information is available about abundance and trend of cutthroat trout in the Fraser River system. They added, “It is our subjective evaluation ... that cutthroat trout are unlikely to be abundant enough to constitute a major factor in the decline.”[8]

The researchers also stated that rainbow trout are known to feed on sockeye eggs in Quesnel Lake, but abundance and trend information is not available for the Fraser River system. Based on Kootenay Lake studies and evidence of increased angler effort, they concluded that rainbow trout have been rather stable over the past decades and are unlikely to be a major factor for the Fraser River sockeye decline. They also concluded that steelhead are unlikely to be a major factor in the sockeye decline, given that the steelhead population is reduced throughout British Columbia, and notably in the Fraser River.[9]

Five species of sculpins are predators on sockeye eggs and alevins. A 1998 study of an Alaskan lake found that sculpins move actively to the spawning beaches before the onset of spawning, and the largest sculpins consume up to 50 eggs in a single feeding.[10] It was estimated that they consumed about 16 percent of the total number of sockeye eggs spawned, primarily immediately after the eggs were spawned. The researchers concluded, however, that

> [g]iven that there is nothing to indicate that sculpins should have increased in abundance in recent decades, and that sculpins may only be a factor on the youngest sockeye, it is not likely that sculpins should be of importance for the decline in the survival of Fraser River sockeye salmon over the last three decades.[11]

Climate change

The authors of Technical Report 9, Climate Change, Dr. Scott Hinch and Dr. Eduardo Martins, examined the possible contribution of climate change to the recent decline in abundance and productivity of Fraser River sockeye by reviewing the literature on the effects of climate-related variables, especially increased water temperatures, on sockeye biology and ecology.

The typical temperature during incubation is 5°C.[12] The researchers reported that survival of sockeye eggs (from fertilization to hatching) is highest at about 8°C, and declines under cooler and warmer temperatures. The survival of alevins (from hatching to emergence from the gravel), however, appears to be independent of temperature. The effect of temperature on survival of eggs and alevins varies among populations from interior and coastal areas. The differences are thought to reflect local adaptations to thermal conditions that the fish’s ancestors have historically experienced during incubation.[13]

Another climate change factor that can potentially decrease the survival of eggs is scouring from the spawning nest during high stream flows generated by rainfall. However, increased rainfall may also be associated with increased freshwater survival, presumably because it increases the area available for spawning and, hence, reduces mortality caused by superimposition of eggs.[14]

The researchers found that adjacent to freshwater habitats, air temperature in the Pacific Northwest has increased an average of 0.08°C per decade over the past century. Precipitation has increased by 14 percent per century, with more of it now occurring as rainfall. In British Columbia, minimum temperatures have shown the highest rate of increase at 0.17°C per decade, and precipitation has increased by 22 percent per century. The highest increases in precipitation have occurred in the interior area.[15]

Warm winters and springs since the 1950s have caused earlier snowmelt and, hence, an advance in the spring freshet by one to four weeks across a large number of rivers in the Pacific Northwest. In the Fraser River, the dates that mark one-third and one-half of the cumulative annual flow have been occurring approximately one day earlier per decade since the 1950s.[16]

There has also been a noticeable increase in water temperatures of rivers and streams. In the Fraser River, water temperatures in the summer have increased at the rate of 0.33°C per decade since the 1950s, and the river is now 2.0°C warmer than 60 years ago. No long-term records of water temperatures in the winter and spring, when sockeye salmon eggs are incubated, are available, but the Fraser River watershed has likely warmed at the highest rates during that season, since that is when the province's climate has warmed the most.[17]

The researchers made a qualitative assessment that life stage–specific survival has changed in the past 20 years owing to the recent climate patterns, particularly in temperature. Assuming that average stream temperatures through winter and spring have not become warmer than 8°C throughout the Fraser River watershed, survival of sockeye salmon eggs has possibly increased, though that of alevins has unlikely changed. However, climate warming may not have affected the early life stages of all Fraser River stocks equally – the survival of interior-spawning stocks may actually have been negatively affected because their eggs and alevins seem better adapted to colder temperatures.[18]

A confounding factor in this assessment relates to increased precipitation. Higher precipitation in the form of rain may have led to an increase in the mortality of eggs (due to scouring) in recent decades. Interior-spawning stocks would have been more affected because changes in precipitation have been greater in the interior. Also, returning adult Fraser River sockeye are now smaller than in the past and, since smaller females bury their eggs at shallower depths than larger fish, the chances of scouring is increased. On the other hand, as noted above, increased precipitation means an increased area for spawning, which may result in lower levels of mortality due to egg superimposition.[19]

Habitat impacts

I heard from DFO witnesses that habitat degradation and loss pose risks to Fraser River sockeye and that, if trends persist, there is going to be a significant decline in the productive capacity of Fraser River sockeye habitat. These trends will affect sockeye in an incremental way over time because habitat productivity has some direct bearing on the ability of an ecosystem to produce fish (although one DFO witness said habitat is not believed to be implicated in the dramatic fluctuations in sockeye returns).[20] I also heard that spawning, rearing, and all the migration routes, including the Fraser estuary, are critically important for maintaining the productivity of these stocks.[21]

Other freshwater stressors

This section considers evidence of the impact on incubating Fraser River sockeye of forest harvesting, the mountain pine beetle, surface water withdrawals and groundwater extraction, small hydroelectric projects, agriculture, and linear development. Some of the stressors discussed here could also affect the emergence and freshwater-rearing phases, which are discussed later in the chapter.

Technical Report 3: Freshwater Ecology

The authors of Technical Report 3, Marc Nelitz and others, sought to understand the potential role of freshwater stressors in recent Fraser River sockeye declines by compiling and analyzing the best available data describing six categories of human activities that have the potential to affect sockeye salmon: forestry, mining, hydroelectric projects, urbanization upstream of Hope, agriculture, and water use.

Forestry. Three core forest-harvesting activities can have a potential impact on sockeye salmon habitats and survival at different life stages. The first is road construction, which has increased by as much as 20 percent in British Columbia's interior over the past decade and interferes with the natural patterns of water flow through a watershed. As water drains across exposed road surfaces, the increased sediment is carried into streams and can cover spawning redds and reduce oxygenation of incubating eggs. Similarly the second activity, upslope harvesting, can alter hydrology of a watershed, which affects the delivery of water and gravel throughout the stream network. Finally, activities in riparian areas can affect water quality by disturbing stream bank integrity, reducing watershed inputs of nutrients and woody debris, and increasing stream temperatures through reduced streamside shading.[22]

Although our understanding of the effects of the mountain pine beetle disturbance is riddled with uncertainty, some things are known. (The mountain pine beetle is further discussed below.) Hydrologists generally agree that the resulting defoliation of pine forests leads to a decrease in interception of precipitation and loss of transpiration, factors that increase the amount of water in soils and in turn affect surface water and groundwater supplies. The loss of forest canopy will also affect the accumulation of snow and rates of snowmelt. These changes are expected to lead to an increase in total water yields and higher peak flows. Increased soil water and stream flow can lead to decreased slope stability, increased flooding, and alterations in the quality and quantity of freshwater habitats. In particular, the combined effects of beetles and salvage logging on watershed hydrology will affect the delivery of water and sediment and could consequently reduce spawner, egg, and juvenile survival. These effects will be most evident in years with intense storms.[23]

Mining. Several processes associated with mining have a potential impact on sockeye salmon–spawning habitats. For example, permanent habitat loss can occur when a mine site or tailing pond is built directly on top of a lake or stream, and mining of gravel or placer minerals from the stream bed itself leads to a less obvious disruption of the stream bed. Silt and sand from roads, pits, and gravel washing can be transported to spawning areas, thereby reducing egg survival. Mines can produce acid drainage, heavy metals, and other contaminants, and sediment from mining activities can increase lake turbidity, which can reduce light penetration and productivity or, conversely, increase nutrients and productivity.[24]

Placer mining, which targets alluvial deposits in modern or ancient stream beds, has a potentially severe impact on sockeye salmon because many alluvial deposits are closely associated with existing streams, and because water is often used to separate placer minerals from the gravel matrix. Gravel (construction aggregate) mining has a potentially severe impact on sockeye salmon populations because it targets alluvial deposits. The researchers concluded that placer mining appears to have the highest potential to reduce early freshwater survival of Fraser River sockeye. Gravel mining is thought to have less impact on Fraser River sockeye, most of which use the reaches where gravel mining occurs only as migratory corridors. The researchers reported that only one active metal mine in the Fraser River drainage is close to habitat occupied by juvenile sockeye salmon, and that no active coal mining or oil and gas production is found in the Fraser River basin.[25]

Hydroelectric projects. Independent power projects could affect sockeye salmon survival in a number of ways. For example, high total gas pressure can occur when gas or air is entrained in water and can then produce gas bubble trauma for eggs or alevins. Also, dams can disrupt the gravel supply to downstream reaches if sediment is either trapped in a reservoir or periodically removed from an intake structure. This disruption in gravel supply can have serious negative effects on channel integrity and the quality of salmon habitat in reaches downstream of dams.[26] The researchers concluded that, given the available data and the small number of independent power projects close to spawning grounds, these projects have not had a significant impact on sockeye salmon populations.[27]

Urbanization upstream of Hope. Noting that more than two-thirds of British Columbians live in the Fraser River basin, the researchers identified three ways in which urbanization and the related built environment have the potential to affect the freshwater habitats of Fraser River sockeye salmon. First, residential, business, and industrial development and road-related construction can increase the amount of impervious surfaces in urban watersheds, which affects rates of interception, patterns of runoff, and, in turn, the magnitude and timing of instream peak and low flows. Second, construction of roads and buildings along stream channels and lake foreshore areas has the potential to reduce riparian vegetation, channelize streams, and block access to habitats. Finally, roads, stormwater runoff, and municipal and industrial effluents have been known to alter water quality in watercourses across the Fraser River basin by changing concentrations of sediments, nutrients, and contaminants. Nevertheless, the researchers concluded that urban environments have a relatively small footprint within watersheds and riparian zones that influence sockeye spawning and rearing habitats, the majority of which are upstream of Hope.[28]

Agriculture. Livestock grazing and crop production can lead to physical alterations of streams, riparian zones, and flood plains. Cattle crossing through streams can potentially increase sedimentation, destroy spawning redds, and destabilize stream banks or widen stream channels. Removal and disturbance of vegetation in the riparian zone can reduce shading and increase water temperature, affecting spawners and eggs. Direct removal of water from groundwater and surface water supplies for irrigation and livestock purposes can be a significant stressor. Finally, agricultural activities can have a significant impact on the water quality of streams and lakes by increasing biochemical oxygen demand; introducing pathogens; and affecting concentrations of sediments, nutrients, and pesticides into waterways. However, the researchers concluded that agriculture has a relatively small footprint within watersheds and riparian zones that influence sockeye salmon spawning and rearing habitats.[29]

Water use. Potential impacts of water use on sockeye salmon are related to alterations in water flows and temperatures. Surface water use can reduce instream flows that constrain access to spawning habitats or, in extreme cases, de-water redds. Extraction of groundwater for irrigation can reduce flows into streams, increasing water temperatures that affect sockeye salmon adults and eggs.[30]

Nelitz and others concluded:

> Our assessment of the cumulative effect of freshwater stressors suggests that the recent declines in Fraser River sockeye salmon are unlikely to be due to changes in freshwater habitats … An important piece of evidence in reaching this conclusion is that juvenile survival has remained relatively stable across CUs where data are available (see Peterman et al. 2010 [Exhibit 748]), even though there is substantial variation in stressor intensity across CUs.* In the literature, there is strong evidence that the stressors examined here can lead to declines and extinctions of populations in a variety of species, including sockeye salmon. A consideration of individual stressors … suggests that the highest levels of overall stress are generated by forest harvesting and roads, while water use and large hydro also generate significant stress for individual CUs.[31]

During the evidentiary hearings, Mr. Nelitz testified that the timing of some stressors does not coincide with the timing of the pattern of decline in sockeye salmon.[32] For example, the mountain pine beetle infestation did not become serious until 2003, at least a decade after the Fraser River sockeye decline began. Similarly, in many watersheds the intensity of forestry disturbances has been relatively stable.[33] Mr. Nelitz also observed that, generally, the longer the migration distance of a Fraser River sockeye stock, the greater its decline.[34]

Forest harvesting impacts

Dr. Peter Tschaplinski, a research scientist with the BC Ministry of Environment, testified that a number of potential forestry-related impacts on

* The conclusion that juvenile survival has remained relatively stable across CUs is based on data from only nine stocks. See Exhibit 748, p. 2.

Fraser River sockeye habitat, including changes to watershed hydrology, can influence stream flow and processes, channel form, and erosional processes, as well as changes to riparian environments that might affect water temperature, nutrient provision, channel structure, and stream microclimates.[35]

Dr. Tschaplinski is the author of a 2010 report evaluating the effectiveness of DFO's riparian management between 2005 and 2008 (the FREP Evaluation).[36] This evaluation found that 87 percent of streams in the province were in one of three stages of properly functioning condition.[37] Dr. Tschaplinski stated that, concerning their potential to harm fish habitat, forestry practices have improved greatly during the recent 20-year decline in Fraser River sockeye and are thus unlikely to have caused the decline.[38] However, he noted the importance of watershed-based baseline research in ensuring that forestry practices do not harm sockeye habitat.[39]

Mountain pine beetle impacts

In March 2007, the British Columbia Forest Practices Board released a special investigative report entitled *The Effect of Mountain Pine Beetle Attack and Salvage Harvesting on Streamflows*.[40] This study was based on Baker Creek, a western tributary of the Fraser River at Quesnel that contains high-value salmon habitat.[41] The study found that:

- peak flows were 60 percent higher after the beetle moved through this watershed;
- total annual flows were 30 percent higher;
- after salvage logging had removed 80 percent of trees in the watershed, peak flows were even higher, at 92 percent;
- flood frequency also increased significantly, with projections that a former once-every-20-year flood would occur every three years on average; and
- the mountain pine beetle would affect flooding, channel stability, and fish habitat within similar watersheds.[42]

In 2010, the head of DFO's Fish-Forestry Research Program, Erland MacIsaac, stated that Fraser River sockeye natal watersheds are not threatened by the mountain pine beetle:

> There's relatively little pine in most of the Fraser River sockeye natal watersheds. Based on the most recent BC forest health aerial survey reports, most of the southern interior watersheds have declining rates of infestation because the mature pine is dead. Areas where there is some current Mountain pine beetle expansion are in the Skeena / Stikine watersheds and northern forest districts as the beetle moves north to more marginal pine areas, but these are areas outside of the Fraser drainage.
>
> There is always the possibility, in the future, that other conifer beetle and defoliant pests (e.g., western balsam bark beetle, western spruce budworm) may experience similar population booms in the types of forests that dominate in the watersheds of Fraser sockeye. But that's mostly speculation at this point.[43]

However, according to Peter Delaney, former chief, Habitat Policy Unit and Fish Habitat Unit and senior program adviser, DFO, significant amounts of pine are found in the catchment areas of some parts of the Fraser River watershed (for example, the Nechako River drainage).[44]

At the hearings, Dr. Tschaplinski and Ian Miller, manager, Sustainable Forest Management, BC Ministry of Forests, Lands and Natural Resource Operations, testified, and documentary evidence was presented about the potential impact of the mountain pine beetle on Fraser River sockeye. The following points were covered:

- Large-scale salvage harvesting can result in high clear-cut areas in a watershed and high levels of forest removal could mean increased water table levels because of alteration to watershed hydrology and high-energy erosional implications for both spawning and rearing habitats.[45]
- The potential exists for increased forest fires, which may result in increased water temperatures and changes in the dynamics of material delivery.[46]
- There is also the potential for increased terrain instability and landslide frequency.[47]
- Salvage-harvesting activities reduce shade and stream functioning.[48]

Dr. Tschaplinski said that provincial field assessments had not yet shown increased clear-cutting in riparian areas.[49] However, Mr. Miller stated that, under the salvage operation under way, it is reasonable to expect larger clear-cuts in the future.[50] A University of British Columbia study examined the impacts of the mountain pine beetle on channel morphology and woody debris in riparian areas.[51] It found that, in the riparian areas surveyed, there were relatively small volumes of pine. The study concluded that woody debris transferred to the streams in the next 25 years is likely to be relatively small and within the range of typical conditions.[52]

Given the unknowns about the future impact of the mountain pine beetle on fish–forestry interactions, Dr. Tschaplinski recommended researching large-scale clear-cutting impacts on fishery values.[53]

Surface water withdrawals and groundwater extraction

Dr. Michael Bradford is a research scientist with DFO and Simon Fraser University, and during the hearings he was qualified as an expert in aquatic habitat ecology.[54] He testified that the impact of removing water from a stream, for any purpose, is to reduce the magnitude of flow.[55] Low stream flows have the potential to affect salmon in various ways, for example, by limiting access to spawning and rearing habitat, interrupting the passage of adults to spawning grounds, and contributing to the problem of high water temperatures during the summer migration period.[56] In some parts of the BC interior, extensive water withdrawals occur in the summer for irrigation, at a time when streams have naturally lower flows and maximum temperatures.[57]

However, impacts from water withdrawals may be less of a concern for sockeye as compared with other species of salmon because major sockeye-producing areas such as the Adams River are generally located downstream of large lakes, which provide a buffering influence on stream flows.[56] Water withdrawals may have some effect on sockeye in the Shuswap Basin, although Dr. Bradford did not think it is as big a problem for sockeye as for coho.[59]

According to Jason Hwang, area manager, BC Interior office, Oceans, Habitat and Enhancement Branch (OHEB), his staff have generally not observed any impact to sockeye related to water withdrawals.[60] However, he said that water withdrawals could become a concern in the future as demand for water increases.[61] Dr. Bradford agreed that population growth, particularly in the drier Okanagan and Cariboo areas, could have impacts on sockeye in the future.[62]

Dr. Steve MacDonald, a research scientist with both DFO and Simon Fraser University, testified as an expert in aquatic habitat ecology. He described how groundwater has a number of functions that may contribute to the survival of salmon, which may be affected by extraction. Groundwater delivers ions and nutrients to streams and generally has a moderating influence on stream temperatures. In winter, particularly in the northern parts of the Fraser River watershed, it prevents streams from freezing and anchor ice from forming. In summer, groundwater provides a cooling influence on the spawning grounds, which is important in areas such as the Nechako watershed. Groundwater may also have a cooling influence on streams, which are used by adult sockeye as “thermal refugia” en route to the spawning grounds.[63]

Dr. Bradford indicated that groundwater extraction is potentially a concern for Cultus Lake sockeye.[64] Dr. Craig Orr, executive director of the Watershed Watch Salmon Society and qualified at the hearings as an expert in behavioural ecology with a specialty in salmon habitat ecology, said that, to maintain the Early Stuart sockeye stocks, something has to be done to protect groundwater.[65] He also said that groundwater is the “key to resilience of the salmon habitat.”[66]

Independent power projects

DFO has identified a number of potential impacts of independent power projects (IPPs) on fish and fish habitat, including the following:

- Construction and installation of powerhouses, intakes, and other hydro infrastructure may cause a harmful alteration, disruption, or destruction of fish habitat (a HADD, which is prohibited by section 35 of the *Fisheries Act*).
- Operating requirements of a facility may alter natural flow regimes and cause a flow-related HADD (where instream flows are insufficient for the protection of fish and fish habitat).
- Entrainment in pen stocks and turbines may lead to mortality of fish.[67]

According to Mr. Hwang, DFO does not consider IPPs to be a "particular imminent concern" for sockeye, given that most of the projects reviewed by DFO are not interacting or intersecting with sockeye.[68] This situation could change, however, if IPPs "continue to be something that is emphasized and development is pursued across a wide portion of the landscape."[69] Neither Dr. Bradford nor Mr. Hwang was aware of any IPPs in the Fraser River watershed that are affecting sockeye or their habitat.[70] However, Dr. Orr cautioned that "these projects are slowly creeping into anadromous fish habitat" and gave two examples of proposed projects with potential to affect steelhead and coho.[71] He also pointed out that it is difficult to assess the impacts of IPPs because there is no planning process, no public input on siting, and a lack of transparency with the monitoring program, and because the impacts are not well understood.[72]

Agriculture

Michael Crowe, section head, BC Interior office, OHEB, testified that agriculture affects fish habitat through runoff that carries pesticides and fertilizers into salmon habitat; water extraction; cattle grazing in and trampling riparian areas; ditching, diking, and stream channelization; and impediments to fish passage.[73]

Use of fertilizers is compounded by increased livestock densities that increase natural fertilizer nitrate and phosphate loading.[74] Runoff from this natural fertilizer can also be laced with chemicals and hormones deriving from animal feed made to augment growth and development.[75] Fertilizer runoff can cause the loss of aquatic plants, lowered oxygen levels, changes in local phytoplankton community structure, and increased biochemical oxygen demand in sediments.[76] Biosolids are also used in the Fraser River basin as fertilizer, and runoff from these application sites is a potential source of municipal wastewater chemicals in the Fraser River watershed.[77]

Linear development

Linear developments in the Fraser River basin include road networks, rail networks, electrical transmission lines, and seismic lines used in the oil and gas industry.[78] Road development can increase the number of stream crossings, which impede fish passage and may affect fish habitat.[79] Road and highway construction can affect local stream habitat and biota, but some of the impact will also be felt downstream.[80] The main threat is fine sediment pollution that can cause direct mortality, reduce reproductive success, and reduce food availability for fish.[81] Other threats include encroachment of development onto flood plains and riparian areas; loss of critical riparian vegetation; and modifications of the stream channel – which can alter flow characteristics, causing further impacts downstream.[82]

The construction of bridges may affect banks only minimally, but channelization and poor construction practices may destabilize channels.[83] Culverts, which are often used as alternatives to spanning structures on streams, can destabilize stream channels by disrupting the flow of woody debris, sediment, and water.[84] Culverts also tend to cause the stream channel to widen above the constriction, reducing current velocities and trapping sediment.[85]

Human development–related impacts in the Lower Fraser River

The authors of Technical Report 12, Lower Fraser Habitat, Dr. Mark Johannes and others, focused exclusively on stressors in the Lower Fraser River, from Hope to the estuary. They summarized potential human development–related impacts from 1990 to 2010, and qualitatively examined potential interactions between human development and activities in the Lower Fraser River for sockeye salmon habitats. Their findings are summarized below, under the heading Life stage 2: smolt outmigration.

Emergence and freshwater rearing

In about May, approximately eight months after spawning, the yolk sac is absorbed into the body cavity, and the alevin becomes a fry.[86] The fry, now typically about 3 cm long, migrates downstream (or more rarely upstream) into a nursery lake in search of food.[87]

The fry typically live in the nursery lake for one year (or in some cases two years), feeding on zooplankton such as *Daphnia*. They tend to remain near the surface at dawn and dusk while they are feeding, and migrate deeper during the bright daylight hours to avoid predators.[88]

Several Fraser River sockeye populations, including the Harrison River population, do not spend a year in a nursery lake and are thought to have a different outmigration pattern. They migrate downstream almost immediately after emerging from the gravel and, after spending a few months in sloughs and estuaries of the Lower Fraser River, enter the Strait of Georgia before they are one year old.[89]

Mr. Lapointe testified that, in the case of a spawning female that lays 3,000 eggs, only about 420 eggs survive to become fry.[90] He identified the following naturally occurring stressors that contribute to fry mortality: lack of food, predation, diseases, and environmental stresses such as water temperature.[91]

Several of the Commission's technical reports examined, and witnesses testified about, the stressors at these two life history stages that may have caused or contributed to the recent decline. I summarize these discussions below.

Predation

In Technical Report 8, Predation, Dr. Christensen and Dr. Trites reported that, as both coho and chinook salmon age, they increasingly prey on other fish. Because they tend to have a longer residence in freshwater, these species typically reach a size where they potentially can prey on small sockeye fry. However, Dr. Christensen and Dr. Trites concluded that the recent decline in population estimates for chinook and coho in the Strait of Georgia indicates that these species are not likely responsible for the decline in survival of Fraser River sockeye.[92]

The researchers stated that, although a 1996 study in Lake Washington found that cutthroat trout was the only important predator on sockeye fry, little information is available on the abundance and trend of coastal cutthroat trout in the Fraser River system. Dr. Christensen and Dr. Trites concluded that cutthroat trout are unlikely to be abundant enough to constitute a major factor in the decline.[93] They reported that, although steelhead can consume a significant amount of emergent sockeye smolt salmon, steelhead are unlikely to be a major factor in the Fraser River sockeye decline, given that the steelhead population is reduced in the Fraser River. They reached a similar conclusion about bull trout.[94]

Several studies in Washington and Oregon found that a significant proportion of the annual diet of large northern pikeminnows consists of sockeye salmon. A pikeminnow eradication program has been in place in Cultus Lake since 2005.[95] The researchers concluded that pikeminnow "may also be important predators of Fraser River sockeye salmon, but there does not seem to be abundance or trend estimates for the pikeminnow, and, hence, their importance cannot be quantified."[96]

During the evidentiary hearings, Jeremy Hume, research biologist, Lakes Research Program, Science Branch, testified that, in 2005, DFO removed about 45,000 northern pikeminnows from Cultus Lake, a practice that has led to increased survival of sockeye salmon.[97]

Dr. Christensen and Dr. Trites also considered several introduced fish species and made the following observations:

- A previous study had identified smallmouth and largemouth bass as predators on juvenile salmon, and considered the risk very high in small lakes. However, notwithstanding the absence of abundance and trend estimates in the Fraser River system, the researchers found it unlikely that these species were abundant enough to have had major influence on the recent decline.[98]
- Yellow perch, which were introduced illegally as live bait, have been confirmed in 59 lakes or ponds and 19 streams, including within the Lower and middle Fraser River and South Thompson River watersheds. Although yellow perch are potentially an important predator on and competitor with sockeye salmon in the Fraser River system, the researchers concluded that "the available information provides little support for the hypothesis that yellow perch were a major factor for sockeye survival trends over the last three decades."[99]

Climate change

The authors of Technical Report 9, Climate Change, Dr. Hinch and Dr. Martins, testified that scientists have identified water temperature as the master biological variable for fish.[100] Temperature directly affects metabolism, physiology, behaviour, and feeding, and indirectly can affect suitable habitat.[101] Metabolic scope defines the oxygen available for activities other than routine metabolism and is temperature-dependent.[102] Dr. Hinch and Dr. Martins reported that survival of sockeye salmon fry decreases when exposed to warm temperatures in the laboratory, although the direct effects of temperature are unlikely to cause mortality in the wild, as fry are able to move to cooler lake depths to avoid stressful temperatures. Several laboratory studies have shown that fry predation increases with higher water temperatures, and a 1985 study showed that sockeye inoculated with a parasite had higher mortality rates when held at 13°C than at 5°C.[103]

The warming trends discussed earlier in relation to eggs and alevins have also been associated with changes in sockeye nursery lakes. For example, since the 1960s Lake Washington has warmed up by as much as 1.5°C, particularly in the upper water layer. As a result of warming of Fraser River watershed nursery lakes, the timing of spring ice breakup has occurred up to 40 days earlier, and the timing of thermal stratification up to 27 days earlier.[104]

The researchers concluded that if water temperatures in Fraser River rearing lakes have paralleled warming trends of the river in the summer, then fry now experience temperatures approximately 1°C warmer than 20 years ago, and their survival has possibly decreased. However, warming is not homogenous throughout the lake volume. Since fry are able to move to cooler depths of lakes to avoid otherwise lethal temperatures at the surface, the researchers believe that warmer lake waters may not have directly affected survival of fry. They hypothesized that warmer waters may have enhanced fry mortality indirectly through increased predation rates, although the authors of Technical Report 8, Predation, provided no evidence of this type of predation.[105]

Other freshwater stressors

As discussed earlier, the authors of Technical Report 3, Freshwater Ecology, Nelitz and others, sought to understand the potential role of freshwater stressors in recent Fraser River sockeye declines by compiling and analyzing the best available data describing six categories of human activities that have the potential to affect sockeye salmon – forestry, mining, hydroelectric projects, urbanization upstream of Hope, agriculture, and water use. These activities have the potential to affect sockeye salmon during freshwater life stages, in particular by having effects on the quality and quantity of spawning habitats, on the productivity of nursery lakes for rearing juveniles, and on habitat conditions associated with smolt outmigration and adult migration. Regulation of these activities is discussed in Volume 1, Chapter 6, Habitat management. The researchers' examinations and conclusions in relation to both the incubation and nursery lake–rearing phases of the life cycle were discussed above (Incubation).

Pulp and paper

Pulp and paper mills were historically a large point source of dioxins and furans in British Columbia.[106] Toxic even in small amounts, dioxins and furans were formed as by-products in the chlorine-bleaching process used in the pulp and paper industry.[107] Dioxins and furans also have a strong tendency to adsorb to sediments and to bioaccumulate and biomagnify up the food chain.[108] The Commission's Technical Report 2, Contaminants, provides a comprehensive list of contaminants of greatest concern found in pulp and paper effluents.[109]

At the time of the hearings, seven pulp and paper mills operated in the Fraser River basin.[110] Two mills are located near Prince George, two near Quesnel, one near Kamloops, and two near Vancouver.[111] Although all seven mills are located along the migration corridor of Fraser River sockeye, Janice Boyd, a program scientist with Environment Canada, testified that, as far as she knows, none of these mills discharges into rearing lakes for Fraser River sockeye.[112] However, I was told that the Domtar Products mill near Kamloops discharges into the Thompson River, which flows into Kamloops Lake, where sockeye rear.[113]

Metal mining

Mines, and metal mines in particular, have the potential to adversely affect water quality conditions in receiving water systems (areas subject to discharges).[114] Intentional and unintentional releases from mines include the following types of contaminants:

- conventional variables (e.g., alkalinity, conductivity, hardness, pH, and total suspended solids);
- microbiological variables (e.g., fecal coliforms and enterococci);
- major ions (potassium, sodium, and sulphate);
- nutrients (e.g., nitrate, nitrite, ammonia, and phosphorus);
- metals (aluminum, arsenic, boron, barium, cadmium, copper, chromium, iron, lead, mercury, manganese, molybdenum, nickel, antimony, selenium, strontium, silver, and zinc);
- cyanides (strong acid dissociable and weak acid dissociable);
- petroleum hydrocarbons (oil and grease, alkanes, and diesel-range organics);
- monoaromatic hydrocarbons (e.g., benzene, toluene, ethylbenzene, and xylene); and
- polycyclic aromatic hydrocarbons (e.g., parent PAHs, alkylated PAHs, and total PAHs).[115]

At the time of the hearings, there were seven active metal mines in the Fraser River watershed: Endako (Prince George area), Huckleberry (Houston area), Gibraltar (between Williams Lake and Quesnel), Mount Polley (near Williams Lake), Quesnel River (near Quesnel), Highland Valley (near Kamloops), and Bralorne (Bridge River area).[116] The first six of these conduct open-pit mining while Bralorne is an underground gold mine.[117] The Endako mine discharges into a creek that drains into François Lake (a sockeye-rearing lake) and into the Endako River, which drains into Fraser Lake.[118] The Huckleberry mine discharges into the Tahtsa Reach on the Nechako Reservoir, which has two discharge points, making it unclear how much, if any, discharge ultimately enters the Fraser River.[119]

There are also closed or abandoned mines in the Fraser River watershed, not all of them known to Environment Canada or the province.[120] However, Michael Hagen, program scientist, Natural Resources Sector Unit, Environmental Protection Operations, Environment Canada, testified that a fair bit is known about most of the closed mines and, although some of these mines could be discharging into the Fraser River system, problems have been addressed where identified.[121]

Infectious diseases

The author of Technical Report 1, Infectious Diseases, Dr. Michael Kent, documented and evaluated the potential effects of diseases and parasites on Fraser River sockeye salmon during both the freshwater and the marine life stages. I summarize his findings on the freshwater period, including the incubation and fry phases.

In a confined environment, fish drink and eat in the same water in which they urinate and defecate. Pathogens can be transmitted among fish in the water environment, and the degree of transmission is greatly influenced by density of fish in water. This transmission may not be an important concern in the ocean or within large rivers, but it can come into play in small rivers, spawning channels, and hatcheries. Thus, infectious agents in particular are transmitted from fish to fish before and during spawning, or as fry.[122]

Several environmental factors can influence the impact of pathogens on salmon. For example, since fish are cold-blooded, both pathogens and hosts are strongly affected by water temperature. High water temperature has been documented to cause stress in fish, to reduce general immune status in fish, and to dramatically increase the replication rate of parasites – all of which lead to increased susceptibility to disease, especially in freshwater. Also, pollution can cause reductions in the immunocompetence of the fish host, and the addition of contaminants through fertilizers and sewage to the freshwater system can influence some fish pathogens.[123]

Dr. Kent identified the following specific pathogens that are either high or moderate risk to Fraser River sockeye juveniles.

Viruses. The infectious hematopoietic necrosis (IHN) virus often causes severe, acute systemic disease in juvenile salmonids, and one strain has been documented to cause high mortality in sockeye fry in many populations. For example, a 1989 study documented 50 percent mortality in the Weaver Creek spawning channel, in a population of about 17 million fish.[124] Dr. Kent concluded that the risk was high, adding that the virus “is deadly to fry and juvenile sockeye salmon. Sockeye in seawater are susceptible, but the virus at this stage is less virulent as older and larger fish show fewer mortalities when they become infected."[125]

Bacteria. The *Renibacterium salmoninarum* bacterium causes bacterial kidney disease (BKD) in salmonids. The infection results in acute to chronic, severe systemic disease, and fish die within a few weeks to months following infection. Infections are contracted and spread in freshwater as well as marine areas, apparently by oral-fecal transmission. Sockeye salmon are highly susceptible, and the bacterium is prevalent in British Columbia, leading Dr. Kent to rate the risk as high. *Aeromonas salmonicida* infection occurs in both wild and cultured fish in British Columbia, and can result in an acute, severe disease with high mortality. All salmonids are susceptible to it. Because the bacterium has the potential to be lethal to juvenile and adult sockeye salmon in both freshwater and seawater, Dr. Kent rated the risk as high.[126] Several members of the *Flavobacterium* genus cause disease in fish in freshwater as well as in the marine environment. Most are considered opportunists that cause significant disease only when fish are compromised by suboptimal environmental conditions. Dr. Kent rated the risk as moderate, explaining that these infections "are generally considered to cause disease mostly in hatcheries, but should not be excluded as a cause of disease in wild sockeye if water conditions are poor. There is no evidence that infections and associated mortality have increased in the Fraser River in recent times."[127]

Protozoa. *Ichthyophthirius multifiliis*, a ciliate protozoan, is a recognized serious pathogen, infecting a wide variety of freshwater fish. The parasites cause severe damage to the skin and gills, often killing fish by asphyxiation as a result of the tissue's reaction to the parasite in the gills. Heavy infections can cause high mortality in salmonids, including wild stocks of sockeye in the Fraser River system. Dr. Kent rated the risk as high, noting that severity would increase with increased water temperature and reduced water flows. The blood flagellate *Cryptobia salmositica* is common in salmonids from freshwater throughout the Pacific Northwest. Juveniles as well as adults are susceptible to the infection, and the parasite can persist in fish after they enter seawater. Infections in wild salmonids, both adults and juveniles, are often lethal. Numerous reports of the infection of sockeye from British Columbia lead Dr. Kent to rate the risk as moderate. He noted that, although the pathogen is capable of causing severe disease, there are no reports on the prevalence in Fraser River sockeye salmon.[128]

Mortality-related genomic signature

Dr. Kristina Miller is head of the Molecular Genetics Section, Salmon and Freshwater Ecosystems Division, Science Branch, DFO. I qualified her as an expert in molecular genetics, immunogenetics, and functional genetics, with a specialty in salmon.[129] She testified respecting the results of her recent investigations into a mortality-related genomic signature (explained below) identified in Fraser River sockeye salmon, some of which were reported in the journal *Science* in 2011.[130] Her research involved sampling returning adults approaching and in the Fraser River, as well as smolts before they left the Fraser River on their outbound migration. I summarize both aspects of her research here.

In 2006, sockeye salmon returning to the Fraser River to spawn were sampled at three locations – in the marine environment up to 200 km before they entered the river, in the Lower Fraser River, and at the spawning grounds. Researchers took tissue samples from the gills and inserted a transmitter in each migrating fish so that they could determine which fish made it to the spawning grounds and which ones successfully spawned. The gill tissue samples were subjected to microarray technology, through which thousands of genes are examined at once, to determine which genes are turned on and which ones are turned off. This sampling yields information about the physiological condition of the tissue; and that condition may be expressed as a pattern, known as a genomic profile or a genomic signature. The researchers also did genetic stock identification in order to look at stock-specific differences.[131]

Dr. Miller stated that, in all three tagging studies, the same genomic signature was associated with poor spawning success, whether the fish were tagged in the marine environment, in the lower river, or on the spawning grounds. In the marine environment, when fish carry this mortality-related signature, they had a 13.5 times lower probability of spawning. A similar pattern was found for returning adults tagged in the lower river and on the spawning grounds, although with not as high a difference in probability of spawning success between fish with and without the mortality-related signature.[132]

Dr. Miller also testified that in the 2006 study she found an association of the mortality-related signature with more rapid entry into the river and faster migration to the spawning grounds.[133] In a paper prepared for the June 2010 Pacific Salmon Commission (PSC) workshop (PSC workshop paper), she reported that approximately 50 percent of the returning adults tested in 2006 carried this mortality-related signature.[134] Salmon with the signature in the ocean carried a four-times-lower probability of reaching the spawning grounds, while those carrying the signature at the spawning grounds were twice as likely to die prematurely as those without this signature.[135] Dr. Miller's paper concluded:

> This study showed unequivocally that Fraser River sockeye salmon are entering the river in a compromised state, that survivorship was somewhat predictable based on gene expression [greater than] 200 km before salmon reach the river, that stocks may be affected differently, and that the freshwater environment alone may not be the sole source of the highly fluctuating mortalities of salmon in the river.[136]

This PSC workshop paper also reported that Dr. Miller's team has since observed this same mortality-related signature in brain, liver, and gill tissue (but not muscle) of adult sockeye in all years over the past decade where available (2003, 2005–2009), with the proportion of affected fish varying in different years.[137]

On the subject of smolts leaving the Fraser River, Dr. Miller's PSC workshop paper reported that the same mortality-related signature was observed in all years where samples are available (2007, 2008, and 2009). In 2008, 60 percent of smolts left the river with the unhealthy signature in the brain, and 40 percent with the signature in the liver. Overall, 82 percent of fish were affected in at least one tissue. There was a 30 percent reduction in brain prevalence of the signature from summer to fall in the ocean, and a 50 percent reduction in the liver. Overall, there were 2.4 times as many fish with the signature in the fall as in the summer. The paper concluded: "If these decreases in prevalence were due to mortality, and if we assume that 120 million smolts left the river in 2008 (there may have been more), we could account for the loss of [more than] 27 million salmon in 2008 associated with the unhealthy signature alone."[138]

Dr. Miller testified that 82 percent of outmigrating smolts have the mortality-related signature in at least one tissue, while the proportion of returning adults affected is much less, for the two years of available data.[139]

In an April 15, 2011, update to DFO scientists, Dr. Miller reported that, in June 2007 and June 2008, smolts were sampled in the marine environment.[140] Nine out of 10 from 2007 contained this mortality-related signature in liver tissue, whereas in 2008 only 40 percent of liver tissues contained it.[141] She testified that "[w]here we're looking to go is to establish whether or not it's simply the prevalence of the signature in the ocean, or whether it's the shift in prevalence that we observe over time that's more important in terms of being a predictor."[142] Dr. Miller speculated that if fish enter the ocean in poor condition, and the ocean is additionally stressed, those factors may have a more profound effect on their survivorship than if they enter, in good condition, an ocean that is in good condition.[143]

Dr. Miller stated that when a genomic signature is obtained, one can then compare the similarities with signatures observed in other controlled studies.[144] From this kind of functional analysis, the most likely explanation for this signature is that it is virally mediated (i.e., it is a response to a viral infection).[145] In addition, the fact that the signature was found in other tissues fits well with a pathogen model, but does not fit well with a general stressor or toxicant exposure.[146] In the 2011 *Science* article, Dr. Miller and her colleagues stated their hypothesis "that the genomic signal associated with elevated mortality is in response to a virus infecting fish before river entry that persists to the spawning areas."[147]

Dr. Miller stated that her finding that the fish are already conditionally challenged before they enter the river during the return migration is consistent with work done by her colleagues Dr. Scott Hinch and Dr. Tony Farrell into stress and osmoregulation.[148] She said those studies were unable to propose a mechanism for why some returning fish were more ready for freshwater and why there were so many stress indicators.[149] Genomics tries to provide a deeper level of understanding of the mechanisms that might create the kinds of patterns being observed.[150] Dr. Miller's study showed a pattern of osmoregulatory preparedness for freshwater when the returning fish were 200 km from the river – they were probably very uncomfortable in the marine environment.[151]

Dr. Kyle Garver leads the Virology Research Program in the Aquatic Animal Health Section, Salmon and Freshwater Ecosystems Division, Science Branch, DFO Pacific Region. I qualified him as an expert in molecular virology, with a specialty in viruses affecting salmon.[152] He testified that he suggested to Dr. Miller, and tried himself, several diagnostic methods to determine if there was a virus in these tissues that have the mortality-related signature.[153] He tried first a traditional virological approach in which the sample is placed onto artificially grown fish cells, and one observes these cells for virus infectivity.[154] Various lines of fish cells were tried, but Dr. Garver was unable to culture any virus. He told me that this is a broad technique and that many viruses cannot be detected using this method.[155]

Dr. Garver then tried microarray technology, using a ViroChip. The ViroChip contains bits of genetic material representing all known viruses and is used to detect the presence of virus in a sample.[156] He testified that after testing samples with the mortality-related signature using the ViroChip, he did not see any conclusive viral signature; in other words, there was no significant difference in the ViroChip results between samples with the mortality-related signature and samples without it.[157]

Dr. Miller told me that she later "background corrected" the samples and was able to report to the Pacific Salmon Commission workshop as follows:*

> A VIRAL PATHOGEN? In collaboration with BC Centre for Disease Control, we ran both healthy and unhealthy tissue RNA on a Viral Array (used to identify viral strains in humans and agricultural animals), and found the unhealthy tissue gave 6X higher intensity binding to the array than healthy tissue. There was a 3-fold over-representation of Retroviral family DNA.[158]

Dr. Miller agreed that in a meeting of DFO scientists she distributed a paper entitled "Epidemic of a novel, cancer-causing viral disease may be associated with wild salmon declines in B.C."[159] This paper was based on literature relating to the salmon leukemia virus, which was thought to involve optic tumours. Samples she examined carried very heavy vascularization on the outside of the optic lobe, but subsequent analysis showed these to be hemorrhages, not tumours.[160]

Dr. Miller said that she and Dr. Garver subsequently attempted to isolate viral material from tissues that contain the mortality-related signature. Using a sucrose gradient method, they were able to isolate DNA from tissue and found that the sequence of this DNA showed a high probability of being a parvovirus. She described her results as "a very, very powerful positive for a parvovirus."[161] Dr. Miller told me that she has observed the parvovirus sequence in tissues positive for the mortality-related signature, but not in liver samples that were negative for the mortality-related signature. At the time of the hearings in August 2011, she and Dr. Garver were in the process of testing other tissue samples, negative for the mortality-related signature, to determine if they contained parvovirus sequences or not."[162] In addition, they planned experiments to isolate the viral particle, to test the infectivity of the parvovirus, and to determine whether the parvovirus is associated with disease.[163]

Dr. Miller testified that one of the most interesting things about parvoviruses is that they require rapidly dividing cells to facilitate their own reproduction, and one can induce the proliferation of parvoviruses by stressing the cells.[164] She is interested in whether the stress associated with transitioning between freshwater and saltwater could make this virus more active and elicit more disease, although this idea is speculative.[165] She added that if it is established that there is a parvovirus, it will be the first time a parvovirus has been identified in a fish.[166]

* Background correction is a step that is not found in the ViroChip maker's protocol, which is Exhibit 1514.

Dr. Miller was asked whether it would be fair to suggest that this particular mortality-related signature, if it turned out to be the virus and if it turned out to have the mortality that she speculated about, could be a very significant explanation for the low return in 2009. She responded that "there is certainly the potential that this virus could have a major impact on salmon declines," and that "[i]t could be the smoking gun" for the 2009 low return. [167]

Dr. Miller added that she had some level of confidence that she and Dr. Garver will find disease with this virus.[168]

Dr. Miller testified that the earliest in the life cycle that she and Dr. Garver have identified this mortality-related signature was in November, before fish were going to smolt, in their natal rearing areas.[169] She added that she has no data on whether a parvovirus could be transmitted vertically (i.e., from adult fish to offspring), so it would be pure speculation.[170] Dr. Garver added that a parvovirus could be in other species in a lake and could, therefore, be transmitted horizontally (i.e., between fish of the same generation), but that is also pure speculation because it is unknown whether the parvovirus sequence is linked to disease, or how it is transmitted.[171]

In August 2011, Dr. Miller testified that industry representatives from BC Atlantic salmon farms had recently agreed to have their fish tested for the presence of parvovirus.[172] She had observed the mortality-related signature and suspected parvovirus in sockeye and chinook salmon, but she did not yet have any Atlantic salmon to test.[173] In December 2011, however, Dr. Miller explained that, shortly after testifying in August, she and Mary Ellen Walling, executive director of the B.C. Salmon Farmers Association, disagreed on when and how Atlantic salmon would be tested.[174] The result was that Dr. Miller no longer had an agreement with the salmon-farming industry to obtain Atlantic salmon samples to test for the parvovirus. She explained:

> I did not feel that what they proposed was what we originally had talked about and what I had said that we were going to do in the Cohen Inquiry and I did feel that there was no need to move forward. I didn't need them to run sockeye salmon, I needed them to provide Atlantic salmon to test.[175]

Dr. Miller rejected the proposal to have sockeye salmon tested with industry as a collaborator.[176]

Life stage 2: smolt outmigration

In about May, approximately 20 months after spawning, when the fry are about 8 cm, they begin a process called "smoltification," a physiological change facilitating the transition from life in freshwater to life in seawater. They cease their movement between shallower and deeper parts of the lake, begin to gather into schools of fish, take on a silvery body coloration, and develop an ability called "compass orientation" that aids their navigation out of the lake and downstream.[177]

Given the magnitude of the Fraser River watershed, some sockeye stocks face a daunting downstream migration. For example, smolts resident in Takla and Stuart lakes, north of Fort St. James, must cover approximately 1,200 km before reaching the ocean.[178]

Mr. Lapointe testified that while approximately 420 fry survive out of a brood of 3,000 eggs, nearly 300 of those fry will die, leaving about only 120 smolts.* This fry mortality usually occurs within the nursery lakes. I understand that these numbers are, at best, estimates. Long-term time series data exist only for two fry populations (Quesnel and Shuswap lakes) and for two smolt potpulations (Chilko and Cultus lakes). Once smolts leave their nursery lake, there was, at the time of the hearings, no standardized assessment of them during or at the end of their downstream migration.[179]

Several of the Commission's technical reports examined, and witnesses testified about, the stressors facing smolts during their downstream migration which may have caused or contributed to the recent decline.

Predation

During the marine ecology hearings, Dr. David Welch, president and CEO, Kintama Research Services, testified that tagging of smolts in Chilko and Cultus lakes showed that most of the

* In a study of Chilko salmon, Dr. Jim Irvine calculated fry-to-smolt mortality at 87.5 percent. See Exhibit 1352.

outmigration mortality occurred between release in the lake and entry into the mainstem of the Fraser River. It occurred during passage through clear river water and was possibly due to predation.[180] In Technical Report 8, Predation, Dr. Christensen and Dr. Trites reported that, although several endemic and introduced fish species are known to feed on salmonid smolts, little information is available on abundance and trend. The researchers' opinion was that none of these species is abundant enough to have had a major influence on the recent Fraser River sockeye salmon decline.[181]

As for predation by birds, the main source of bird abundance information for British Columbia is the Christmas Bird Count, conducted annually between mid-December and early January. The researchers said that the count gives information that is standardized for observer effort, which means that it can be used to evaluate trends in abundance. However, it provides no information about notable bird species that do not overwinter in British Columbia.[182]

The common merganser is an important predator on juvenile salmon during the seaward migration, as is the double-crested cormorant. The Caspian tern has recently begun breeding in British Columbia. A 2003 study estimated that, in 1998, Caspian terns consumed 12.4 million salmon smolts in the Columbia River estuary.[183] However, the researchers concluded that since there is no indication that these three species have increased in abundance in recent decades, it is therefore unlikely they have played a major role in the decline.[184] Because osprey migrate south for the winter, trend data are not available. The researchers concluded that it is unlikely that the osprey has a major predation impact on Fraser River sockeye.

The harbour seal is the only marine mammal known to occur in British Columbia that has been documented feeding on salmon smolts in freshwater and estuarine habitats. However, direct observations of feeding showed predation on chum, coho, and chinook, but not on sockeye.[185] The researchers concluded:

> Despite the shortcomings of the data, they are the best that are available and show no indications that harbour seals are a significant predator of sockeye salmon smolts … Harbour seal numbers increased during the 1980s and 1990s, but have been relatively constant for the past decade. Harbour seals should therefore have not posed an increasing threat to sockeye survival over the past decade.[186]

Climate change

The authors of Technical Report 9, Climate Change, Dr. Hinch and Dr. Martins, did not specifically address the impact of warmer river temperatures on Fraser River sockeye salmon smolts during their downstream migration, other than to observe that the Fraser River watershed has likely warmed at the highest rates during winter and spring because that is when the province's climate has warmed the most.[187]

Infectious diseases

In Technical Report 1, Infectious Diseases, Dr. Kent pointed to numerous reports of a high prevalence of the infection of the *Parvicapsula minibicornis* parasite in outmigrating Fraser River sockeye smolts as well as in adults. The chronic infection targets the kidneys and can also reduce swimming ability. Given the high prevalence in Fraser River sockeye salmon, Dr. Kent rated the risk as high.[188]

Other freshwater stressors

The authors of Technical Report 3, Freshwater Ecology, Nelitz and others, sought to understand the potential role in recent Fraser River sockeye declines of six categories of human activities that have the potential to affect sockeye salmon: forestry, mining, hydroelectric projects, urbanization upstream of Hope, agriculture, and water use. The regulation of these activities is discussed in Volume 1, Chapter 6, Habitat management.

Although most of these stressors have an impact on the incubation and nursery lakes phases of the Fraser River sockeye salmon life cycle, several are relevant to the smolt downstream migration.

Log storage / handling in the Fraser River estuary. Port Metro Vancouver estimates that 48 different tenants are distributed across 256 log storage leases and permits, covering 862 hectares within the Fraser River estuary. Logs can compact, scour, and shade nearshore habitats and smother marine plants. The reduction in primary production and growth can reduce food availability for juvenile salmon. Wood and bark debris can accumulate beneath storage areas, potentially altering the composition of food sources, smothering emergent vegetation, increasing biological oxygen demand, and increasing concentrations of potentially toxic log leachates. Studies on the effect of Fraser River estuary log storage on salmon are limited, but the researchers reported that studies in the 1980s revealed that densities of juvenile salmon (chinook, pink, and chum) and amphipods (a food source) did not differ between a large log storage site and nearby marsh areas.[189]

Hydroelectric projects. Large-scale hydroelectric projects can cause direct mortality of smolts that pass through hydro turbines or over spillways. The researchers reported that the Bridge River–Seton power project has the potential to affect Seton and Anderson Lake sockeye Conservation Units during the smolt downstream migration. A 1995 study indicated that more than 90 percent of sockeye smolts were being entrained into the power canal, with the smolt mortality rate estimated at 17 percent when the plant was fully operational.[190] A 2006 study estimated that the average number of smolts lost at the canal was approximately 200,000. However, mitigation measures introduced since 2006 have reduced smolt mortality rates to as low as 1.7 percent.[191] The researchers also concluded that, given the small number of projects in proximity to migration corridors, independent power projects have not had a significant impact on sockeye salmon populations during the downstream migration.[192] During the hearings, two witnesses testified that they were not aware of any independent power projects in the Fraser River watershed that are affecting sockeye salmon or their habitat.[193] However, a third witness cautioned that it is very difficult to assess the impacts of independent power projects because of the absence of a planning process, public input on siting, and transparency within the monitoring program, and the fact that the impacts are not well understood.[194]

Agriculture. The researchers observed that, because of the concentration of agricultural lands along the Fraser River mainstem in the Cariboo-Chilcotin, agriculture has its greatest interaction with migration corridors. The researchers found that data on type and intensity of pressure on lands and streams associated with livestock production were generally lacking.[195]

Urbanization upstream of Hope. Residential, business, and industrial development, as well as related road construction, can increase the amount of impervious surfaces in urban watersheds, which affects rates of interception, patterns of runoff, and, in turn, the magnitude and timing of instream flows. Also, roads, stormwater runoff, and municipal and industrial effluents have been known to alter water quality in watercourses across the Fraser River basin.[196]

Lower Fraser River habitat use

One technical report focused exclusively on stressors in the Lower Fraser River. In Technical Report 12, Lower Fraser Habitat, authors Johannes and others summarized potential human development–related impacts from 1990 to 2010, and qualitatively examined potential interactions between human development and activities in the Lower Fraser River (from Hope to the estuary) for sockeye salmon habitats. In testimony, several witnesses challenged some of the evidence contained in Technical Report 12. Where applicable, I set out below the contradictory evidence.

Sockeye salmon freshwater distribution in the Lower Fraser River extends to four major watersheds: the Harrison, Lillooet, Chilliwack, and Pitt rivers. Spawning, incubation, and juvenile rearing occur in these watersheds, including Cultus Lake. Residence periods extend for between four and six months for river-type sockeye such as Harrison River stocks, and one or two years for lake-type sockeye.

This 160-km portion of the Lower Fraser River and estuary is used as a migratory pathway for smolts, with a residence period of often less than seven to 10 days. Harrison River sockeye fry are an exception, using various sloughs and off-channel areas in the Lower Fraser River and estuary for

Restored salmon habitat, South Alouette River, BC, 2010

rearing over a period of two to six months. Feeding in the Lower Fraser River by Harrison fry is likely to be in micro-habitats that are attached to the main channel, where water clarity is better and where access to planktonic prey items may be better than what is available in the main channel. Disruptions or losses to these habitats may therefore have greater impact on these sockeye than disruptions to the deeper portions of the riverbed (such as through navigational dredging), because sockeye do not bottom feed.

The researchers selected seven factors as metrics to express changes over space and time in human activities and development in the Lower Fraser River between at least 1990 and 2010. They were considered potential stressors that could illustrate effects and interactions of human activities and development on the environment and show relevance for possible interaction with sockeye habitats.

Population. Population size and density provide a broad metric of potential stress on the environment and allow a generic estimate of human activities that could result in changes in land and marine areas through urban, rural, and industrial development. The researchers reported that population size and density in most regional districts and in all municipalities in the Lower Mainland increased by 150 percent over the past 20 years. Human activities related to population size and density may affect sockeye habitats through direct habitat loss and through non–point source effects associated with change in water quality and quantity.[197]

Land use. Development of residential, recreational, and industrial lands and transportation corridors in the Lower Fraser River has removed and degraded habitat areas and natural environments over the past century. Such development leads to higher levels of water pollution, nutrients, and contaminants from wastewater and stormwater runoff. Reduced natural forest, riparian, wetland, watercourse, and water body areas limit the natural capacity of landscapes to filter and buffer surface water runoff and recharge groundwater sources. The researchers observed that agricultural and forestry land use have remained stable in many regional districts, although in urban areas more than 3,000 hectares of forest and agricultural land were lost and replaced over the past two decades.[198]

Large industrial and infrastructure sites and projects. Between 1990 and 2010, 70 large industrial sites and infrastructure projects were constructed in or near the Lower Fraser River and Strait of Georgia. Thirty-six such sites or projects had potential overlap with aquatic habitat, but the researchers considered the overlap with sockeye habitats to be minimal for many of them.[199]

Waste. An estimated 80 percent of marine pollution is derived from land-based activities through liquid and solid waste. Programs to reduce the amount of solid waste disposed of were adopted in British Columbia and Metro Vancouver less than 20 years ago. Owing in part to improved best practices such as recycling and secondary or better sewage treatment, solid and liquid waste volumes, despite population growth, have not increased across the region over the past 20 years. (This last point was an assumption, not based on any analysis.) Don MacDonald, the principal author of Technical Report 2, Contaminants, gave contrary expert evidence.* He testified that the volume of discharges from wastewater treatment plants has increased over the past 20 years.[200] Johannes and others reported in Technical Report 12, Lower Fraser Habitat, that the proportion of municipalities using secondary or tertiary wastewater treatment has increased over the past 20 years – a change that has led to reduced tonnage of biological oxygen demand and total suspended solids being discharged into the environment from municipal wastewater, despite population growth.[201]

Dredging and diking activities. Fraser Valley urban areas and cities are protected by more than 400 km of dikes, built during the first half of the 20th century. No new dikes have been constructed in the past two decades, and in some cases dikes have been removed or replaced to create opportunities for salmon habitat restoration. The volume of material dredged from the Lower Fraser River has declined since the 1990s and is timed to occur outside the migration period.[202]

Contaminated materials. Contaminants such as metals and organic pollutants show a general decreasing trend over time in many organisms, presumably as a result of decreases associated with effluent discharge regulations, improved treatment, and remediation of contaminated sites. In contrast, there appears to be an increase in polybrominated diphenylethers (PBDEs, which are fire retardants found in a wide array of products) associated with increased use over the past two decades and an apparent increase in contaminants associated with personal care and pharmaceutical products. The latter may be increasing in the environment because conventional municipal wastewater treatment systems do not remove them.[203] (See Freshwater and marine contaminants, below.)

Non-indigenous species. More than 117 terrestrial and aquatic non-indigenous plant and animal species have established populations in the Lower Fraser River and Strait of Georgia. Among the nine fish species introduced into freshwater areas are the smallmouth bass and yellow perch, which are predators with the potential to directly affect sockeye survival during early life history growth in nursery habitats. However, the number of non-indigenous freshwater species has remained stable from 1990 to 2010.[204] (See Predation, above.)

The researchers noted that few data are available for quantitative evaluation and review of the amount of sockeye habitat change over time in the Lower Fraser River relative to human activities. Consequently, the potential effects from human activities on sockeye habitats were reviewed qualitatively through a hierarchical classification and ranking method that shows the substantial potential overlap with the activity and sockeye habitat, and the potential level of interaction. The researchers concluded that only one human activity, land use in Lower Fraser River watersheds, constituted a moderate risk of loss or degradation of sockeye habitats. All other human activities were ranked as low or nil risk of loss or degradation.[205]

Pathogens from salmonid enhancement facilities

In Technical Report 1A, Enhancement Facility Diseases, authors Dr. Craig Stephen and others were commissioned to evaluate the impacts of hatchery and spawning-channel diseases on wild Fraser River sockeye salmon and to determine their potential role in reduced productivity. (For detailed discussions of wild–enhanced salmon interactions and management aspects of salmon enhancement, see Volume 1, chapters 6, Habitat management, and 9, Fish health management. The British Columbia Salmonid Enhancement Program was established in 1977. As of the hearings in May 2011, there were 23 major enhancement facilities and spawning channels managed by government employees, 21 community hatcheries operated as part of the Community Economic Development Program, and about 350 public involvement projects supported by 18 DFO community advisers.[206]

* Mr. MacDonald was qualified as an expert in environmental toxicology and chemistry with particular expertise in ecological risk assessment and ecosystem-based management; water quality and water use interactions; the design and evaluation of contaminated sediments on ecology receptors, including fish; and the design and implementation of environmental quality monitoring programs. Transcript, May 9, 2011, pp. 9–10; Exhibit 828.

Weaver Creek Spawning Channel, BC, 2010

Four sockeye salmon spawning channels have been created in the Fraser River drainage. They are located in Weaver Creek, Nadina River, Horsefly River, and Gates Creek. There are also two hatchery programs for the Upper Pitt River and Cultus Lake stocks. Two spawning channels are responsible for more than 80 percent of the sockeye salmon produced in enhancement facilities – Weaver Creek (67 percent) and Nadina River (14 percent). Between 2005 and 2009, an average of 46 million sockeye salmon were released each year from enhancement facilities in British Columbia.* During that same period, approximately 97 percent of all enhanced populations of sockeye salmon released from program facilities originated in spawning channels.[207]

Movements of fish create the risk of pathogen movements. Three types of movements are relevant:

- *The movement of fish after release from hatcheries or spawning channels.* DFO has the practice of acclimating salmonids other than sockeye to seawater by short-term holding in net pens. DFO records indicated that six major facilities and 21 community facilities or sites use seawater or brackish water pens in British Columbia.
- *The transportation of fish from a hatchery to a distant receiving water body.* For example, the Freshwater Fisheries Society of BC stocks nearly 900 provincial lakes with fish produced from five facilities.
- *The transfer of fish between enhancement facilities.* No pathogen screening of fish being transferred from one enhancement facility to another takes place if both are within the same region. For these purposes, the entire Fraser River basin is one region.[208]

Another plausible exposure route occurs with the release of water or wastes contaminated with pathogens from hatcheries and spawning channels into streams, rivers, and other fish-bearing waters. The survival of fish pathogens in water can vary with environmental conditions, ranging from days to weeks and even months.[209]

The researchers reported that neither federal nor provincial regimes have been designed or funded to be a surveillance or monitoring program. Rather, they offer diagnostic support in response to active disease concerns in hatcheries. The data collected can best be used to describe the types of problems causing increased disease frequency and mortality in hatcheries but do not reflect the infection status of the hatchery population as a whole or allow the generation of information on rates of disease.[210] The quality of the data is not audited or systematically evaluated. Diagnostic laboratory records would not detect all disease events or pathogens found in an enhancement facility because the criteria used to encourage submissions to the laboratory reduce the likelihood that diseases which are endemic and familiar to the hatchery workers would be submitted, and diseases causing sporadic or a low level of mortality would not be subject to laboratory investigation.[211]

Dr. Christine MacWilliams, fish health veterinarian, Salmonid Enhancement Program, DFO, qualified as an expert on veterinary sciences with a specialty in fish health, testified that the level of screening of enhancement facilities is sufficient and that DFO “probably” does not miss

* During the habitat enhancement and restoration hearings, the evidence of Greg Savard (Exhibit 758) was that, between 2006 and 2009, the average number of Fraser River sockeye produced annually from hatcheries and spawning channels was approximately 40 million. About 90 percent of these fish were from spawning channels.

any disease outbreaks.[212] (Dr. MacWilliams's evidence on the management of enhancement facilities is summarized in Volume 1, Chapter 9, Fish health management.)

The researchers' review of hatchery records found 17 reports of fish being placed into fish-bearing waters (released into streams or rivers, or moved to lake or sea pens) with known infectious diseases, suspected infections, or clinical signs of undiagnosed disease. In some cases the fish were given a chemical treatment and released three to 10 days later, without records verifying that the treatment was effective. Most often, these releases involved myxobacterial infections or fish with symptoms consistent with myxobacteriosis. One case involved suspected but unconfirmed furunculosis.[213]

In Technical Report 1A, Enhancement Facility Diseases, Stephen and others showed that the seasonal timing of salmonid releases from enhancement facilities overlaps with the migration timing of wild fish, a practice designed to optimize survival of enhancement fish and one that creates opportunities for interactions with wild fish. However, no data were found on niche or habitat overlaps between wild sockeye salmon and enhanced salmon, or on whether the temporal co-occurrence in the same waters is sufficient to result in the exchange of pathogens between wild sockeye salmon and enhanced fish.[214]

The researchers undertook an exposure assessment to estimate the probability that Fraser River sockeye salmon are exposed to the hazards of concern. They concluded:

> Fraser River sockeye salmon reared in enhancement facilities have the most likely route of exposure to diseases present in hatcheries or spawning channels. Exposure of other Fraser River sockeye to infected enhanced fish, sockeye salmon or otherwise, has not been proven or disproven. Biologically plausible routes of exposure exist, but none have been measured. Generally, there are three variables that affect the probability of exposure; the geographic distribution of the escaped pathogen, the abundance of the pathogen in the receiving environment and the frequency with which the fish are involved in an exposure that results in transmission of the pathogen. As data are lacking for these three variables, exposure assessment is not possible.[215]

Stephen and others were also unable to clarify what effect, if any, a pathogen of enhancement facility origin might have on Fraser River sockeye salmon productivity. They stated:

> The risk assessment did establish that known fish pathogens do occur in salmonid hatcheries and spawning channels and that biologically plausible routes of exposure exist, but there was no monitoring or follow-up to establish that pathogens were transferred to sockeye salmon outside of sockeye salmon enhancement facilities and that the transfer impacted the population.[216]

The researchers concluded that the data available for the review could not prove or disprove that diseases associated with salmonid enhancement facilities have been transmitted to Fraser River sockeye salmon and, in turn, have affected their production. The portion of the Fraser River sockeye salmon population that is reared in spawning channels or hatcheries has, at times, been exposed to infectious diseases while within the enhancement operation, but there was no evidence that this exposure has medium- to long-term population-regulating effects. It could not be established whether Fraser River sockeye salmon not reared in enhancement facilities had or had not been exposed to infectious agents of enhancement facility origin.[217]

During the evidentiary hearings, Dr. Stephen agreed that the most important conclusion in his report was the following:

> We could not determine if diseases present in salmon enhancement facilities (hatcheries or spawning channels) present potential for serious or irreversible harms to Fraser River sockeye salmon. Limitations in scientific understanding, lack of ongoing surveillance of wild and cultured fishes, and deficits in data provided to us were the primary reasons for our inability to make specific cause-effect conclusions and to qualitatively or quantitatively assess risk.[218]

Freshwater and marine contaminants

Overview

A "contaminant" is a substance that can be detected, while a "pollutant" is a contaminant that has been shown to have an adverse biological effect on the environment.[219] Contaminants in the Fraser River and marine environments originate from both natural and anthropogenic sources. Natural sources include weathering and erosion of terrestrial soils, bacterial decomposition of vegetation and animal matter, and long-range transport of substances from natural combustion sources including wildfires.[220]

Anthropogenic contaminants enter the environment through a number of sources, which are discussed below. Contaminant research also shows that Pacific salmon accumulating persistent, bioaccumulative, and toxic contaminants (PBTs)* in their marine life stage transport these contaminants into spawning and lake environments.[221] These chemicals are fat-soluble, meaning they accumulate in fatty tissues and so readily accumulate in aquatic food chains and can reach relatively high concentrations in fish.[222]

Contaminant effects on sockeye can be lethal (i.e., result in an immediate fish kill), but more commonly the effects are sublethal and may cause sockeye to be more susceptible to disease, parasites, or predators.[223] Both lethal and non-lethal effects can be enhanced when fish are challenged by other environmental factors such as high temperatures, disease, or nutritional stress.[224]

DFO has acknowledged that contaminants such as pesticides and other pollutants may potentially affect Fraser River salmon.[225] Many contaminants enter sockeye habitats, and exposure to contaminants can occur at any stage of the sockeye life cycle through digestion, gills, skin absorption, or sensory exposure.[226]

Dr. Peter Ross, research scientist, Marine Environmental Quality Section, Institute of Ocean Sciences, Science Branch, testified as an expert in aquatic toxicology.[227] He explained that marine fish and fish habitat, including Fraser River sockeye habitat, can be affected by chronic low-level releases of contaminants.[228] There are two types of impacts due to exposure to two general classes of contaminants:

- *Deferred ("carry the risk") effects.* In this category, sockeye are exposed as eggs in their spawning habitat; smolts in freshwater, estuarine, or coastal habitats; or juveniles in coastal or oceanic habitat.†
- *More immediate ("gauntlet") effects.* In this category, sockeye are exposed as they transit from lake to ocean and back to lake.[229]

At the Pacific Salmon Commission's June 2010 workshop on the decline of Fraser River sockeye salmon, the Expert Advisory Panel concluded that the relative likelihood of contaminants in the Fraser River causing the 2009 decline was possible, but unlikely or very unlikely.[230] In terms of the long-term trend in declining productivity, the Expert Advisory Panel considered Fraser River contaminants and habitat conditions unlikely or very unlikely contributors to the decline.[231] However, limited site-specific data on contaminants and the often complex nature of environmental toxicological processes make any conclusion about the role of contaminants highly uncertain.[232]

In contrast to the conclusions of the Expert Advisory Panel, DFO Science contaminant researchers who participated in the 2010 PSC workshop concluded that it was plausible that contaminants were a secondary contributor to reduced productivity of Fraser River sockeye, but that direct evidence is lacking and, further, that the monitoring or assessment studies to assess any impacts are lacking.[233] Moreover, these researchers also noted that chemicals are likely to fall into the category of sockeye stressors that we can actually control.[234]

Dr. Ross told the Commission that DFO is in a very poor position to be able to rule out the effect of contaminants on the decline of Fraser River sockeye, largely because of the absence of data:

* PBTs include many well-known chemicals such as dioxins, furans, polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), polycyclic aromatic hydrocarbons (PAHs), and dichlorodiphenyltrichloroethane (DDT). See PPR 14, pp. 48–49.

† Fish, including sockeye, do not easily metabolize PBTs, so they can "carry the risk" of these contaminants with them through their entire life cycle. But as sockeye migrate home from the sea they use their fat reserves, so these chemicals can then be transferred to their reproductive tissues. See Exhibit 573, p. 31; Exhibit 73, pp. 75–76.

> An absence of data, or an absence of evidence to me is not evidence of absence, and I think it's a little bit dangerous to use an absence of data or an absence of evidence to suggest that contaminants play no role whatsoever or are indeed unlikely to play a role.
> …
> I think it gives short shrift to the examples we have from other parts of Canada with salmon that have been dramatically impacted by acid rain in Eastern Canada, and aluminum and copper and pesticides in New Brunswick. It gives short shrift to the evidence we have from our colleagues, our federal colleagues to the south of us where we see Chinook salmon returning to Puget Sound that are being affected by urban contaminants.
> …
> So these are some specific examples. Other scientists, other toxicologists might have a slightly different view, but clearly we're data deficient in terms of our current capacity to understand what's happening with the sockeye situation.[235]

Further, according to Dr. Ross, contaminants very likely contributed to the long-term decline in the sense that they may have contributed through small incidents here and there (i.e., "death by a thousand cuts") or they may have weakened the fish over time, such that when they went to sea they may have been more vulnerable.[236]

Technical Report 2: Contaminants

In Technical Report 2, Contaminants, authors MacDonald and others developed an inventory of aquatic contaminants for the Fraser River basin and evaluated the potential effects of those contaminants on Fraser River sockeye salmon. The greatest potential impact of these contaminants occurs during the downstream migration of sockeye salmon smolts. During the evidentiary hearings, Mr. MacDonald testified that virtually no data are available on spawning and rearing areas.[237] He added that he and his co-authors did not expect to see a lot of contaminated sediments within these areas, because sockeye spawn largely in headwater systems or mainstem areas farther up the Fraser River. These areas are spatially separated from the source releases of contaminants into the system.[238]

The researchers identified 15 key exposure areas within the Fraser River basin.[239] To determine if exposure to contaminants represents a causative or contributing factor in the decline, it was necessary to compare recent conditions in the watershed with those that have existed historically in the Fraser River and its tributary watersheds.[240] The temporal scope of this study was broadly defined to include the years 1965 through 2010, the period of record for which reliable water quality data are available.[241] Mr. MacDonald testified that, because of data gaps in this time period, the researchers sometimes had to make assumptions that the water quality data for river water downstream of a nursery lake were the same as for the lake water itself.[242]

The researchers identified 11 point sources (discharge of substantial volumes of wastewater from identifiable locations into receiving waters within the Fraser River basin) of contaminants.

Pulp and paper mills. Ten pulp and paper mills are located between Prince George and Greater Vancouver.[243] The researchers identified 12 categories of substances of greatest concern relative to contamination of aquatic habitats by pulp mill effluents. Several of the specific substances identified are ammonia, chlorides, mercury, benzene, toluene, and chlorophenols.[244]

Sawmills, plywood mills, and particle board mills. From the numerous mills throughout the Fraser River basin, the researchers identified nine categories of substances of greatest concern. Several of the specific substances identified are ammonia, phosphorus, sulphides, sulphates, and formaldehyde.[245]

Wood preservation facilities. At least 15 operating wood preservation facilities are located within the Fraser River basin. Among the highest-priority wood preservation chemicals are creosote and chromated copper arsenate.[246]

Cement and concrete plants. There are 17 plants operating in the basin, most in the Lower Fraser River. The contaminants of greatest concern include pH, total suspended solids, sodium, potassium, chlorine, sulphates, oil and grease, and metals such as aluminum, arsenic, copper, chromium, lead, and zinc.[247]

Seafood-processing facilities. At least 10 seafood-processing facilities are permitted to discharge effluent into the Lower Fraser River. In addition to temperature and pH, the priority contaminants include total suspended solids, residual chlorine, oil and grease, and nutrients such as nitrate, nitrite, and ammonia.[248]

Operating and abandoned mines. There are 28 operating metal and mineral mines within the Fraser River basin. These have the potential to influence water quality conditions in receiving water systems as a result of construction and operation of mine components such as camp facilities, sewage and wastewater treatment facilities, tailings containment areas, open pits, waste rock piles, roads and storage yards, airstrips, and quarries.[249] Abandoned mines may also release metals into receiving water systems in the Fraser River basin.

Bulk storage and shipping facilities. There are 24 facilities located within the Fraser River basin, most in the Lower Fraser River. Contaminants such as metals and petroleum hydrocarbons could be released through oil spills, fuel spills, or sloughing of anti-fouling paints.[250]

Other manufacturing facilities. A wide range of manufacturing facilities and operations are located throughout the Fraser River basin, including foundries and those dealing with wood pellets, rubber products, chemicals, food products, packaging, boat-building, gypsum products, cleaning compounds, and asphalt paving.[251] The contaminants of greatest interest include metals, nutrients, petroleum hydrocarbons, polymers, solvents, resins, chemical additives, volatile organic compounds, and cyanide.[252]

Contaminated sites. About 5,000 contaminated sites within the basin are registered with the provincial Land Remediation Branch. These sites include those with spills of raw or partially treated sewage as well as gasoline, oil, diesel, and other fuels.[253] Mr. MacDonald testified that there may be an additional 4,000 sites.[254]

Municipal wastewater treatment facilities. There are 31 facilities in the Fraser River basin. Eleven in the Lower Fraser River and on Iona Island account for 97 percent of volume (3.0 million cubic metres per day). A 1998 study of concentrations upstream and downstream of the Annacis Island wastewater treatment plant determined that levels of chromium, copper, iron, zinc, and PCBs downstream of the plant exceeded water quality guidelines. Effluents from wastewater treatment plants are also known to contain a variety of other substances, including pharmaceuticals, fire retardants, steroids, personal care products, and disinfectants.[255] Mr. MacDonald testified that the volume of discharges from wastewater treatment plants has increased over the past 20 years.[256] The data to evaluate them are not available, but it is assumed that the concentrations of these contaminants are increasing.[257]

Salmonid enhancement facilities. At least 37 facilities are located in the Fraser River basin, all of them generating wastewater that is discharged into receiving water systems. The highest-priority contaminants are bromine, chlorine, formalin, and formaldehyde.[258]

The researchers also identified several non-point sources (diffuse discharges of runoff from a variety of areas) and atmospheric sources of contaminants.

Runoff from forest management areas. Road building, road maintenance, and clear-cut logging can cause losses of fine sediment as a result of accelerated erosion.[259] Fertilizers, pesticides, herbicides, and insecticides applied to enhance production of timber can be lost through runoff into receiving waters. Mr. MacDonald testified that pine beetle salvage logging leads to deforestation, which in turn leads to erosion and the release of fine sediments into the receiving water systems.[260] When carried along in the flow, these suspended solids get into fish gills, causing toxicity. If they are deposited in the stream substrate, the suspended solids can create a layer on the bottom that can suffocate eggs.[261]

Runoff from agricultural operations. Cattle ranching, feed lots, chicken farms, dairy operations, and tree fruit and vegetable crops result in a wide range of herbcides, insecticides, and fungicides entering receiving waters.[262]

Runoff from municipal stormwater. Contaminants associated with this type of runoff include road salts, metals, polycyclic aromatic hydrocarbons, oil and grease, total suspended solids, nutrients, and pesticides.[263]

Runoff from linear developments. Releases of contaminants can occur during construction, maintenance, or decommissioning of roads, railways, and electrical transmission lines. The substances of greatest concern include chloride, nitrates, ammonia, arsenic, lead, mercury, petroleum hydrocarbons, and herbicides.[264]

Atmospheric sources of contaminants. These include natural sources such as forest fires (e.g., carbon monoxide, benzene) and volcanoes (e.g., silica, aluminum, potassium, sodium, iron, sulphate, and hydrochloric and sulphuric acid); human-caused sources such as vehicle, industrial, and agricultural emissions; and long-range transport of atmospheric pollutants.[265]

From this review, the researchers developed an Inventory of Aquatic Contaminants that included more than 200 substances which may be released into aquatic ecosystems in the Fraser River basin from the various land uses identified. They went on to identify which of those substances occur in surface water or sediment at concentrations sufficient to pose potential risks to aquatic organisms, including sockeye salmon. Based on water quality data from 12 of the 15 geographical areas examined, the researchers identified 23 chemicals of potential concern measured in surface water at concentrations sufficient to pose potential risks to sockeye salmon eggs, alevins, fry, smolts, or adults.[266] Data on sediment quality conditions were available for only four geographical areas in the watershed, and the researchers identified 11 substances posing potential hazards to sockeye salmon.[267]

The researchers then went on to determine if one or more of these contaminants of concern occur at concentrations sufficient to cause or substantially contribute to adverse effects on the survival, growth, or reproduction of sockeye salmon in the Fraser River basin. They refined the list of contaminants to 17 substances in surface water, five substances in sediment, and several others with the potential to accumulate in the tissues of sockeye salmon.[268] They found that water quality conditions in spawning, incubation, and rearing habitats did not exhibit any significant trend. For the migration corridors, water quality conditions generally showed a downward trend between 1965 and 1990, were consistent between 1990 and 2003, and showed improvements thereafter (although the researchers acknowledged that the reliability of post-2003 data was uncertain).[269]

The researchers concluded that the available limited data do not implicate water quality conditions (as measured by standard water quality parameters) as a major factor influencing recent trends in sockeye salmon abundance in the Fraser River basin.[270] However, numerous contaminants of concern occur in one or more habitats at concentrations sufficient to adversely affect the survival, growth, or reproduction of sockeye. These contaminants include total suspended solids, six metals (aluminum, chromium, copper, iron, mercury, and silver), and phenols. The researchers also cautioned that bioaccumulation of contaminants in fish tissues has the potential to adversely affect the productivity of sockeye salmon, although the magnitude and extent of such effects could not be determined with the available data.[271]

MacDonald and others then undertook a qualitative evaluation of the potential effects of endocrine-disrupting compounds. Many of the substances released into the environment as a result of human activities have the potential to modulate or disrupt the endocrine system of aquatic organisms. These chemicals interfere with the natural hormones responsible for the maintenance of homeostasis (metabolic equilibrium), reproduction, development, and behaviour. Exposure has the potential to cause a variety of adverse effects in fish, including abnormal thyroid function, decreased fertility, decreased hatching success, de-masculinization and feminization, defeminization and masculinization, and alteration of immune function (see below).[272]

Endocrine-disrupting chemicals include a wide variety of substances that are released into the environment from natural and human-caused sources, including:

- pharmaceutical and personal care products, including synthetic hormones, and ingredients found in cosmetics, toiletries, detergents, and cleaning products;
- industrial chemicals, including PCBs, PBDEs, ethers, esters, and bisphenol A;

- pesticides, including DDT, organophosphate pesticides, insecticides, herbicides, and fungicides;
- inorganic and organometallic compounds, including methyl mercury; and
- biogenic compounds, including several estrogen-like compounds.[273]

The researchers identified three types of effluents in which endocrine-disrupting compounds are most likely to be observed.

Municipal wastewater treatment plant effluents. For incubating sockeye eggs and alevins, exposure to wastewater treatment plant effluent is likely to be negligible for most Conservation Units. Two exceptions may be Harrison River sockeye spawning downstream of the treatment plant located at Harrison Hot Springs, and Salmon River stocks that may be exposed to diluted wastewater treatment plant effluent during incubation. No evidence was found indicating that treatment plants discharge directly into nursery lakes used for early rearing, so exposure to effluent during this phase of the life cycle is negligible for all stocks, except for the Harrison River stocks, which rear in the backwater areas and sloughs within the Lower Fraser River. However, there are numerous wastewater treatment plants located along the migration corridors. The magnitude and duration of exposure to endocrine-disrupting compounds is a function of several factors, including the level of effluent treatment used, the volume of effluent discharged, dilution capacity of receiving waters, distance travelled during downstream migration, and sockeye residence times in areas with significant effluent discharges. Since residence times are unknown, the researchers assumed that the magnitude and duration of exposure are highest for upriver stocks with the longest migration distances.[274] During the hearings on municipal wastewater treatment, three witnesses agreed that municipal wastewater potentially has harmful effects on Fraser River sockeye, in particular sublethal effects, and that it cannot be ruled out as a contributing factor to the long-term decline.[275] Dr. Ross referred to the cumulative, sublethal exposure to chemicals of concern, in particular persistent chemicals that do not break down, such as dioxins, PCBs, organic chlorine pesticides, and PBDEs.[276]

Pulp and paper mill effluents. Exposure of sockeye eggs and alevins to pulp and paper effluent is likely to be negligible during the incubation period and, with one exception, during nursery lake rearing, because none of the mills discharges into spawning streams or nursery lakes.* However, all 10 pulp and paper mills are located along the migratory corridors, and the magnitude and duration of exposure are functions of several processes, similar to those noted earlier. Since residence times are unknown for most stocks, the researchers assumed that the magnitude and duration of exposure are highest for upriver stocks with the longest migration distances.[277]

Areas with high industrial activity / chemical contamination. Exposure is likely to be negligible for salmon eggs and alevins in most Conservation Units. No information was located indicating the presence of point source industrial or other discharges into nursery lakes used for early rearing (except possibly Fraser Lake) and, accordingly, exposure during early rearing is considered to be negligible, except for Harrison River stocks. However, there are numerous point and non–point source discharges along the migration corridor. Because loadings of endocrine-disrupting compounds to the watershed from these discharges are virtually unknown, the researchers assumed that the magnitude and duration of exposure are highest for upriver stocks with the longest migration distances.[278]

The researchers concluded that exposure of smolts to endocrine-disrupting compounds at levels at or above those likely to be observed in the Fraser River basin has the potential to adversely affect adult reproduction.[279] However, it is unlikely that reproductive effects associated

* The one exception is the Harrison River stocks, which rear in backwater areas and sloughs within the Lower Fraser River. However, during the hearings on pulp and paper effluent, Robert Grace, environmental impact assessment biologist, BC Ministry of Environment, testified that there is some rearing of sockeye in Kamloops Lake and that one mill near Kamloops discharges into the Thompson River, which flows into Kamloops Lake. See Transcript, June 13, 2011, p. 49.

with endocrine-disrupting compound exposure are sufficient to explain the declines in Fraser River sockeye salmon abundance over the past two decades, or the poor returns in 2009, for several reasons: exposure to such compounds in pulp and paper mill effluents has likely decreased; exposure durations during downstream migration may be insufficient to elicit significant reproductive effects; and there is little evidence for differential response among stocks that possibly receive different exposures to such compounds.[280] However, exposure may come from other sources, such as municipal wastewater discharges, which have increased.[281]

MacDonald and others cautioned that it is nevertheless possible that exposure to endocrine-disrupting compounds is causing other types of effects that could be sufficient to adversely affect the survival, growth, or reproduction of sockeye, through compromise of the immune system. They stated:

> Collectively, the results of studies on immunosuppression indicates that exposure to endocrine disrupting compounds has the potential to adversely affect salmon during their transition to the marine environment. If the concentrations of endocrine-disrupting compounds were sufficient to elicit these types of effects in the Fraser River, then the resultant mortality of smolts during transition to the marine environment could have contributed to long-term declines in sockeye salmon abundance.[282]

Finally, the researchers undertook a qualitative evaluation of the potential effects of contaminants of emerging concern – this term describes a broad group of chemicals that were previously unknown or not previously recognized as being of concern relative to human or environmental health.[283] Contaminants of emerging concern in the Fraser River basin are likely to include veterinary and human antibiotics, prescription and non-prescription drugs, industrial and household waste products, sex and steroidal hormones, herbicides, fungicides, wood preservatives, and polychlorinated paraffins.[284] The sources for these contaminants include municipal wastewater treatment plants; and runoff from feedlots, industrial and manufacturing facilities, and wood preservation facilities.[285]

According to the researchers, exposure is likely to be negligible for most stocks of sockeye eggs and alevins, and during early rearing. Contaminants of emerging concern are most likely to be released along migratory corridors. However, during the urbanization hearings, Dr. Robie Macdonald, section head, Marine Environmental Quality, Institute of Ocean Sciences, Science Branch, testified that salmon can accumulate concentrations of fat-soluble contaminants such as PCBs which, when the salmon reach their spawning lakes or rivers, are many times higher than the water systems.[286] These contaminants can then be transferred into the watershed when the returning sockeye die. Owing to the paucity of toxicity and exposure data, it is difficult to evaluate the risks to sockeye.[287] Nevertheless, the researchers concluded that contaminants of emerging concern are a significant environmental issue that needs to be addressed and could be causing, or substantially contributing to, the decline of Fraser River sockeye observed over the past two decades.[288]

Overall, the researchers concluded:

- Exposure to measured contaminants in surface water, sediments, and fish tissues is not a primary factor influencing the productivity or abundance of Fraser River sockeye salmon over the past 20 years or in 2009.[289]
- There is a strong possibility that exposure to contaminants of concern, endocrine-disrupting chemicals, and/or contaminants of emerging concern has contributed to the decline of sockeye salmon abundance in the Fraser River basin over the past 20 years.[290]

In his testimony, Don MacDonald agreed that his report did not address contaminants in the marine environment; synergistic effects between and among contaminants; or interactive effects of temperature, disease, and contaminants.[291]

Testifying on freshwater urbanization, Dr. Robie Macdonald stated that water quality monitoring of the Fraser River provides information about general river functioning (e.g., temperature and nutrient work), but provides no information on pesticide exposure, mercury uptake, pharmaceuticals, or biomagnifying accumulating contaminants (e.g., PCBs).[292] The monitoring done is not intended to assess receiving water quality for Fraser River sockeye salmon.[293]

Specific freshwater and marine contaminant sources

Municipal wastewater

Dr. Peter Ross, Graham van Aggelen, and Dr. Ken Ashley all provided their opinion on the potential effects of wastewater effluent on Fraser River sockeye.* All agreed that municipal wastewater potentially has harmful effects, in particular sublethal effects, on Fraser River sockeye and cannot be ruled out as a contributing factor to the long-term decline of Fraser River sockeye (but is likely not the "smoking gun" concerning the poor returns in 2009).[294]

Dr. Ross explained that a number of chemicals of concern involving Fraser River sockeye are in wastewater.† In support of this view, he pointed to research in other jurisdictions that has shown effects on fish from chemicals in wastewater.[295] According to Dr. Ross, there are 90 wastewater treatment plants in the Fraser River valley. He was concerned about the cumulative, sublethal exposure to chemicals of concern – in particular, persistent chemicals that do not break down, such as dioxins, PCBs, organic chlorine pesticides, and PBDEs – by Fraser River sockeye throughout their early life and on their return migration.[296]

Mr. van Aggelen described how chemicals of emerging concern in wastewater can be present at very low levels that can cause endocrine disruption. Many municipal wastewater systems do not, or cannot, remove or treat these chemicals, and traditional fish health bioassays cannot detect them.[297] He agreed with Dr. Ross that of particular concern is the persistent, low-level continuous exposure that can have a cumulative, sublethal effect on Fraser River sockeye.[298]

Dr. Ashley agreed with Dr. Ross in expressing concern about sublethal endocrine-disrupting chemicals. He also described a potential acute toxicity issue for sockeye with some of the high concentrations of ammonia, which can be acutely lethal depending on pH and temperature discharged from the Annacis Island and Lulu Island wastewater treatment plants.[299] He said that the design of the plants at Annacis and Lulu islands are not particularly effective at converting ammonia to a non-toxic form, so they discharge it at fairly high concentrations into the effluent stream, assuming that the ammonia will be diluted to the point where it is non-toxic to salmonids.[300] Despite his concern, Dr. Ashley was not aware of any evidence that links harmful effects from ammonia from Annacis and Lulu islands to Fraser River sockeye.[301]

In the Pacific Region, DFO is not involved in monitoring or researching the impacts of municipal wastewater on salmon or Fraser River sockeye,[302] nor is anyone from Environment Canada tasked with assessing the impacts of municipal wastewater on salmon.[303] Dr. Ross testified that the lack of research on the effects of chemicals in wastewater on Fraser River sockeye makes it difficult to speak with certainty about potential effects on these fish stocks.[304]

On the related issue of stormwater, this runoff contains oil, gas, metals, PCBs, grease, antifreeze, solvents, pesticides, herbicides, fertilizer, paint, detergents, road salt, and animal feces.[305] The Commission's Technical Report 2, Contaminants, summarizes the contaminants most commonly associated with runoff of stormwater from urban centres:

- total suspended solids;
- major ions (chlorides);
- metals (arsenic, cadmium, copper, chromium, lead, mercury, nickel, and zinc);
- monoaromatic hydrocarbons;
- polycyclic aromatic hydrocarbons;
- petroleum hydrocarbons (e.g., oil and grease);
- polychlorinated biphenyls;
- organochlorine pesticides (e.g., DDT); and
- pesticides.[306]

* I qualified all three witnesses as experts in their fields. Specifically, Mr. van Aggelen, head, Environmental Toxicology Section, Pacific Environmental Sciences Centre, Environment Canada, was qualified as an expert in toxicology and toxicogenomics (Transcript, June 14, 2011, p. 3; Exhibit 1044). Dr. Ashley, senior scientist, Northwest Hydraulic Consultants, was qualified as an expert in environmental engineering, aquatic ecology, and limnology (Transcript, November 4, 2011, p. 3; Exhibit 1045). For Dr. Ross's duties and expert qualifications, see the Freshwater and marine contaminants overview, above.

† "Chemicals of concern" and "chemicals of emerging concern," as used by some witnesses, may not match exactly to the use of the terms in Technical Report 2, Contaminants. See Exhibit 826, in particular Tables 3.16 and 3.17, for a list of chemicals frequently found in municipal wastewater (pp. T-48–T-50) and a list of contaminants of emerging concern commonly present at elevated levels in wastewater treatment plant effluents (pp. T-51–T-52).

In some communities, such as Vancouver, combined systems are connected to wastewater treatment facilities, where both stormwater and sanitary sewage receive treatment.[307] One disadvantage with combined systems is that, during periods of heavy precipitation, they can become overloaded and wastewater is typically directed to combined sewer overflows (CSOs), allowing raw sewage and untreated stormwater to overflow at many exit points upstream of the treatment facility and to enter receiving waters directly without any treatment.[308] Municipalities with combined sewer systems typically experience tens of overflows of CSOs annually.[309]

Dr. Ashley testified that overflows from combined sewage and stormwater sewer systems have the potential to harm Fraser River sockeye depending on the timing and magnitude of the discharge event.[310] He stated that there is a risk of acute and chronic toxicity and accumulation of persistent contaminants in the Fraser River.[311] Dr. Ross added that research in Puget Sound, Washington, has shown that runoff from CSOs has created problems for salmon.[312]

Pesticides

The broad application of pesticides to crops, lawns, and forests results in mostly non–point source pollution in the form of runoff.[313] Pesticides can also get into surface waters from overspraying, erosion of contaminated soils, and contaminated groundwater.[314] Mr. MacDonald, lead author of Technical Report 2, Contaminants, testified that the forestry sector's use of pesticides may be one of the greatest concerns for Fraser River sockeye.[315] A 2003 Environment Canada study on pesticide use in Canada states that the majority of pesticides sold and used in British Columbia were used by the forestry sector.[316] This report also says that a number of pesticide active ingredients were used exclusively in the agriculture sector and accounted for 63 percent of total sales.[317] I also heard from Dr. Ross that agriculture and forestry pesticides are of concern with respect to Fraser River sockeye.[318] Technical Report 2 describes a number of water quality concerns associated with agriculture.[319]

Greywater

Greywater is wastewater originating from showers, baths, bathroom sinks, kitchen sinks, pools, spas, and laundry.[320] (See Volume 1, Chapter 6, Habitat management.) It gets into the environment through municipal wastewater systems, septic systems, and discharge from vessels. It can contain nutrients, bacteria, viruses, and a variety of chemicals, including endocrine disruptors associated with detergents and personal care products.[321] According to the province, the cumulative effects of multiple vessels discharging greywater may result in the long-term disruption of natural nutrient levels and subsequent impacts on the natural ecology of a water body like Shuswap Lake.[322]

Gravel removal in the Lower Fraser River

On the topic of potential gravel removal impacts on Fraser River sockeye, I heard from two witnesses qualified as experts in freshwater fish habitat in flowing waters and rivers, with an emphasis on the Lower Fraser River (for management of the potential impacts of gravel removal, see Volume 1, Chapter 6, Habitat management).[323]

Dr. Laura Rempel, a habitat biologist within OHEB, described the potential impacts to fish habitat generally resulting from gravel removal, most of which can be mitigated through planning, best practice, and due diligence (having contractors remove the gravel, and in the design of the removal itself).[324]

For most Fraser River sockeye salmon, the Lower Fraser River gravel reach appears to provide habitat during only relatively brief periods of migration. Sockeye salmon are not known to spawn regularly in the gravel reach, but at least one population has spawned sporadically in a slough habitat toward the top end of the area (Maria Slough).[325] Dr. Rempel described the occurrence of sockeye found in the gravel reach in beach seines she carried out over three years. Out of a total of more than 40,000 fish caught in these seines, on average only 0.8 percent were juvenile sockeye.[326] Dr. Rempel stated that the juvenile sockeye caught in her beach seines may have been river-type sockeye or strays from a lake-type population, but without genetic analysis there is no way to be sure which they might be.[327]

The other expert, Dr. Marvin Rosenau, is an instructor of fish wildlife and recreation technology at the British Columbia Institute of Technology.

He described the results from two single-day surveys on the gravel reach, in 2007 and in 2010.[328] Some juvenile sockeye – not outmigrating smolts – were found at two locations in 2007.[329] Juvenile sockeye were found in one of the locations in 2010 as well.[330]

Dr. Rosenau also described information on sockeye that arose out of projects by his students.[331] These projects found that sockeye were present in the winter in some isolated ponds off the mainstem of the Fraser River.[332] He summarized these findings:

> So with respect to sockeye, we're seeing this behaviour, as the floodwaters increase and then decrease, of fish moving out into the sides, the perimeters of the river, and then moving back out, in some cases clearly onto gravel bars.[333]

Dr. Rempel testified that the sites surveyed by Dr. Rosenau's students are located outside the area of gravel removal.[334] In response, Dr. Rosenau stated that one of his study sites was a gravel removal site in the 1980s. He explained that, in any event, the key piece of information is the ubiquity of sockeye throughout the flood plain and their movements during the high-discharge periods, which indicate that we do not yet understand what the fish are doing in these habitats.[335]

I also heard from Mr. Hwang that gravel removal has significantly less potential to affect sockeye than other habitat impacts such as changes to the watershed due to the mountain pine beetle, water diversions, and cumulative impacts of accelerated and high rates of foreshore and recreational property development.[336]

Both Dr. Rempel and Mr. Hwang stated that gravel removal probably has a very small potential to affect sockeye and sockeye habitat.[337] Dr. Rempel stated that DFO has adequate information to appreciate the relative use by sockeye of the gravel reach habitats.[338] However, I also heard from both Dr. Rempel and Dr. Rosenau that there are limitations to DFO's understanding of the ways that sockeye use this area.[339] Dr. Rosenau testified that the presence of juvenile sockeye in ponds that develop periodically over a number of years suggests that a lot more juvenile sockeye are in the river during high-discharge periods than previously thought.[340] He added that we need to know more about these sockeye that were found to be predominantly Late Stuart and Stellako stocks, both of which are lake-type sockeye.[341] He concluded that, in his view, we do not yet understand the role of the gravel reach with respect to sockeye salmon.[342]

Life stage 3: coastal migration

Strait of Georgia and Juan de Fuca Strait

After leaving the river, it is believed that most Fraser River sockeye juveniles turn north and migrate through the Strait of Georgia, Johnstone Strait, and Queen Charlotte Strait and into Queen Charlotte Sound.

There is some evidence that the Harrison River population, and perhaps other populations, are exceptions to this migratory pattern. For example, it appears that the Harrison River population spends the remainder of its outward migration year in the Strait of Georgia and then migrates south of Vancouver Island through Juan de Fuca Strait to the west coast of Vancouver Island.[343]

Several of the Commission's technical reports examined, and witnesses testified about, the stressors that may have caused or contributed to the recent decline during this life stage. I summarize these discussions below.

Predation

The authors of Technical Report 8, Predation, Dr. Christensen and Dr. Trites, reported that the mortality of salmonids in the ocean can be substantial – a 1968 study found early mortality of pink salmon fry to be between 2 and 4 percent per day for the first 40 days.[344] The researchers considered two categories of potential predators relevant to the postsmolt migration through the Strait of Georgia, which I will discuss in turn.

Fish

A 1991 study estimated that river lamprey in the Strait of Georgia were a major predator on postsmolt salmon, consuming an estimated 65 percent of coho and 25 percent of chinook salmon, but only 2.3 percent of sockeye production. Dr. Christensen and Dr. Trites concluded that the river lamprey may be an important predator on sockeye postsmolts; however, in the absence of estimates for trends in abundance, it is not possible to quantify the effect.[345]

Although ecosystem modelling conducted in 2001 suggested that spiny dogfish consumed approximately 145 tonnes of juvenile sockeye and pink salmon in the Strait of Georgia, there is no evidence of any clear changes in dogfish abundance between 1980 and 2005.[346] Similarly, a 2001 study estimated that, if sockeye postsmolts constituted 0.5 percent of the diet of chinook salmon in the Strait of Georgia, those chinook could consume 345 tonnes of postsmolts, or 35 million fish, per year. However, the researchers concluded that it is unlikely that chinook is of importance for the decline of Fraser River sockeye salmon, given the considerable decline in chinook abundance in the Strait of Georgia over the past decades. They reached a similar conclusion in relation to predation by coho salmon.[347]

During the evidentiary hearings, Gordon McFarlane, former head, Marine Fish Population Dynamics, Pacific Biological Station, Science Branch, testified that he agreed with the researchers' final conclusions that marine fish probably were not a major factor in the 2009 reduced returns.[348] He would, however, spend more time studying the dogfish shark.[349]

Birds

Common murres are seabirds that come ashore in summer to breed, and the Strait of Georgia has major concentrations in the late summer and the fall. They actively feed on smolts during release periods of hatcheries. However, data from the Christmas Bird Counts do not indicate any increasing trend over time. Based on this observation, the researchers concluded that the common murre may not be an important factor for the decline in survival of Fraser River sockeye salmon.[350] They also discounted the pelagic cormorant, Brandt's cormorant, and the glaucous-winged gull, given their recent declines in abundance.[351]

During the evidentiary hearings, Mr. McFarlane testified that the most vulnerable phase of marine life to predation is the first four or five weeks after entering saltwater.[352] The year 2007 saw very low productivity in the Strait of Georgia, and smolts had the lowest length:weight ratio on record – an occurrence that, over the next weeks or months, could have increased mortality and vulnerability to predation.[353]

Sockeye habitat use in the Strait of Georgia

In Technical Report 12, Lower Fraser Habitat, authors Johannes and others summarized potential human development–related impacts over the recent 1990–2010 period and examined potential interactions between human development and activities in the Lower Fraser River (from Hope to the estuary) for sockeye salmon habitats. Some witnesses challenged the evidence contained in Technical Report 12, and I set out the conflicting evidence below. The researchers addressed stressors and marine conditions specific to the Strait of Georgia, including land use, shipping and vessel traffic, and water and biological properties.

Land use. Development of residential, recreational, and industrial lands and transportation corridors in the Strait of Georgia has removed and degraded habitat areas and natural environments over the past century. Larger population size and density lead to higher levels of water pollution, nutrients, and contaminants from wastewater and stormwater runoff.[354]

Shipping and vessel traffic. Shipping and marine vessels are a source of noise, contaminants, accidental spills, and non-indigenous species into the marine areas of the Strait of Georgia through hull fouling and ballast water exchange. However, port vessel traffic and ferry traffic across the Strait of Georgia have remained relatively stable during the past two decades. Although cruise ship traffic has nearly tripled between 1990 and 2005, it remains a small proportion of total ship movements. The researchers concluded that shipping and vessel traffic have limited direct interaction with sockeye habitats.[355]

Strait of Georgia water properties. Water circulation and other properties in the Strait of

Georgia – such as sea surface temperature, sea surface salinity, and nutrient properties and distribution – are determined by a number of factors, primarily the seasonality of freshwater discharge from the Fraser River, variation and strength of prevailing winds and tidal mixing, and currents influenced by climate and Pacific Ocean conditions.[356] For example, freshwater discharge from the Fraser River controls local circulation and helps stratify the upper layers of the water column in the strait.[357] Time series of sea surface temperatures showed that Juan de Fuca Strait was cooler at all times than the Strait of Georgia, and that a gradual warming of waters has occurred. Such changes in the physical water properties in the strait, which are linked to biological production, have implications on the distribution, growth, and survival of sockeye salmon.[358]

Strait of Georgia biological properties. Biological conditions in the Strait of Georgia show a large range of variation over seasons and years. Phytoplankton primary production supports the marine ecosystem and food chain in the Strait of Georgia. The onset of the spring phytoplankton bloom (late February to early April) appears to be associated with changes in wind, solar radiation, and Fraser River discharge.[359] Harmful *Heterosigma* algal blooms can potentially cause mortality in various marine species, including salmon, through altered ability to uptake oxygen and diminished respiratory function. However, because sockeye smolt distribution, timing, and migration tend to be outside the nearshore coastal areas and time periods normally associated with the generation and spread of *Heterosigma* blooms, the researchers concluded that no observations are currently available to assert or reject causal links between sockeye mortality and harmful algal blooms.[360] (However, I heard expert testimony regarding the potential impact of harmful algal blooms on Fraser River sockeye during the marine ecology hearings that asserted otherwise [see below].) *Neocalanus*, a dominant zooplankton species and preferred prey for Fraser River sockeye smolts while migrating through the Strait of Georgia, started a large decline in abundance in the 1990s, with a further decline between 2003 and 2007 associated with a warming trend. It is a subarctic copepod, and during warming conditions it is unable to complete its life cycle. Other copepods have become more abundant in the food web with the decline of *Neocalanus*.[361]

The researchers found that few data are available to be used for quantitative evaluation and review on the amount of sockeye habitat change over time in the Strait of Georgia relative to human activities. Nevertheless, concerning the impact of major project development, the researchers concluded that there has been a net gain of habitat:

> The habitat protection strategies used in the lower Fraser River and Strait of Georgia appear to be effective at supporting sockeye habitat conservation during project review and project-related activities (e.g., construction impacts of a specific project). More broadly, a hypothesis that the declines in Fraser River sockeye production are the result of major (or even moderate and minor) project development is not supported by the likely net gains in habitat that have occurred over the period of review.
>
> Overall, the development of major projects and resource restoration efforts during the period 1990–2010 has resulted in a net gain of sockeye habitat and these gains have been substantially added to through efforts to restore historically lost or damaged fish habitats.[362]

The researchers' summary of potential links to Fraser River sockeye declines based on interaction and effects of human activities identified Strait of Georgia water properties as a moderate risk, and Strait of Georgia biological properties as a high risk. They concluded (without a quantitative analysis):

> Potential interactions between biophysical conditions in the Strait of Georgia and sockeye (habitat and habitat use) are suggested as representing moderate and high risk to sockeye in the data compiled here, but limits in existing studies and data prevent an analysis of the causality of these interactions. Our review suggests that there may be an association between changes in biophysical conditions (temperature and food availability and/or quality) in the Strait of Georgia, sockeye habitat use and potentially production. This observation is not supported by conclusive causal linkages, but is supported by other studies which suggest that

> Fraser sockeye production is expected to be higher when sockeye growth and condition are high, compared with poorer sockeye production in years where the sockeye have lower growth and condition. Cooler years in the Strait of Georgia are expected to result in habitats with higher abundance and availability of preferred sockeye prey and lower levels of competitors and predators … The observations of association in time and space between sockeye declines and water and biological conditions in the strait are unlikely to be solely responsible for the declines observed in sockeye populations. The cause is likely much more complex, although the observations do suggest that research in these areas is warranted.[363]

During the evidentiary hearings, Dr. Johannes testified that his conclusion regarding net habitat gains was really an assumption based on several conditions – the available literature, his own experience as part of a team working on environmental assessments and reviews, and his professional experience throughout his career.[364] The Harper and Quigley study he relied on for his conclusion of net gain in sockeye habitat dealt with habitat across all of Canada, not just Fraser River habitat, and that study found that a No Net Loss determination could not be made for 86 percent of the projects.[365]

Dr. Johannes said that when other species of salmon and other fish are taken into consideration, habitat is in fact being lost.[366] However, based on records of distribution, he stated: "I would say that in the Lower Fraser for those races and sub-populations of sockeye that might use the Lower Fraser, there's probably been no net loss."[367] Regarding the requirement to restore lost habitat on a two-for-one basis, Dr. Johannes said that he did not know the extent to which the 2:1 requirements for habitat replacement are complied with or completed: "Conceptually, I would say that … during the last time period, there is no net loss conceptually. If I had the opportunity to look at the compliance records and the audits and the detailed information, I don't know what the answer might be."[368] He agreed with the suggestion that one has to consistently monitor projects to ensure that developers are in fact doing a proper job when they say they are restoring habitat.[369]

Other witnesses disagreed with the conclusion that there has been no net loss in habitat. Patrice LeBlanc, director, Habitat Management Policy Branch, Program Policy Sector, testified that Canada is not achieving No Net Loss and that there is an inability to measure the losses occurring nationally.[370] Mr. Hwang testified that, at the operational level, all indications are that Canada is not meeting the No Net Loss principle.[371] Rebecca Reid, former regional director, OHEB, and, at the time of the hearings in April 2011, regional director, Fisheries and Aquaculture Management, agreed that Canada is probably not achieving No Net Loss, but there is insufficient information to know for sure.[372] Randy Nelson, regional director, Conservation and Protection, Pacific Region, said that, in his experience from working on the Fraser River for 20 years, there probably has been a loss of fish habitat in many areas.[373] (For a more detailed discussion of the No Net Loss principle, see Volume 1, Chapter 6, Habitat management.)

Climate change

The authors of Technical Report 9, Climate Change, Dr. Hinch and Dr. Martins, reported that sea surface temperature in the Strait of Georgia has increased at about 0.25°C per decade since the 1950s. Waters are now 1.5°C warmer than 60 years ago, and 0.5°C warmer than 20 years ago.[374]

Thermal conditions experienced by smolts during their first months of marine life are closely related to their first-year survival. Although warmer temperatures have been frequently associated with increased early marine survival in Alaskan sockeye, the same conditions have been associated with poor survival of Fraser River sockeye migrating along the British Columbia coast. Warm sea surface temperatures are associated with reduced upwelling and, hence, low food availability (zooplankton) for young sockeye. As well, the peak timing of the main zooplankter in the Strait of Georgia has advanced up to 30 days in the past decades, and the peak duration has shortened in response to warming. The observed advance in timing of the Fraser River spring freshet may also be contributing to an earlier peak in zooplankton density in the Strait of Georgia, which might mean that sockeye smolts miss the peak of zooplankton (their food) abundance.[375]

The researchers concluded that warm sea surface temperatures lead to high metabolic rates in sockeye salmon and can change food availability, an occurrence that can reduce early marine growth. Warm waters can increase the abundance of non-resident predatory fish and can also increase the metabolic rate of resident predator fish, leading to increased food consumption.[376] These changes suggest that survival of juvenile Fraser River sockeye salmon during their coastal migration has likely decreased in the past two decades, although there were no data for Fraser River sockeye on which to base the analysis.[377]

Marine ecology

In Technical Report 4, Marine Ecology, authors Dr. Stewart McKinnell and others referred to studies from the 1960s which found that the earliest sockeye migrants into the Strait of Georgia had the least diverse diets – copepods were the dominant prey in April, but their contribution to sockeye diet diminished in later months. *Neocalanus*, the dominant copepod in the Strait of Georgia, experienced an 87 percent decline in abundance between 2001 and 2006, and this decline was accompanied by longer-term declines in copepod zooplankton biomass in the strait. During the years of *Neocalanus* decline, the composition of the phytoplankton spring bloom varied annually among diatoms, flagellates, and dinoflagellates.[378]

A review of studies from the 1970s found that the average size of sockeye postsmolts caught in the Fraser River plume and in the Gulf Islands did not increase with time through April, May, and June, a conclusion that led to speculation that they spend too little time in the Strait of Georgia to allow them to increase significantly in size.[379] (However, a major problem with such a conclusion arises from the high probability of a variable composition of the catch – fish caught in June may not be from the same stock as those caught in April.) The researchers concluded that sockeye salmon emerging from rivers in the southern part of their range, such as the Fraser River, have lower growth rates and lower average marine survival than their counterparts in southeastern Alaska. It is therefore not unreasonable to consider the initial period of their postsmolt migration as a race northward to find better feeding conditions in coastal Alaska.[380]

In addressing the low 2009 return of Fraser River sockeye, the researchers examined conditions in the Strait of Georgia during mid-2007, when postsmolts that would return in 2009 were migrating north. The Fraser River creates a large plume of fresh and brackish nutrient-rich water that spreads thinly across the strait and is mixed with salty water below by tides, winds, and currents. The sun's radiant energy, which provides the most warmth at the surface layer, reinforces the density gradient. In the spring of 2007, the daily volumes of freshwater entering the Strait of Georgia were often in the upper quartile, but not extreme. Similarly, an analysis of daily sea surface temperatures and salinity at four stations in the Strait of Georgia indicates that 2007 was not extreme in the historical record.[381]

Phytoplankton and nitrate concentrations during the winter and spring of 2007 in the Strait of Georgia were found to be similar to what had been observed from 2002 to 2006. During the summer, phytoplankton concentrations were higher at most stations but then dropped during the autumn. Surface temperatures in the Strait of Georgia remained warmer than average or average through the sockeye outmigration in 2007 and 2008. However, the deep waters in the strait began cooling in mid-2007, a trend that continued and intensified into 2008.[382]

During the evidentiary hearings, the Government of Canada tendered four reports that considered juvenile Fraser River sockeye ecology in the Strait of Georgia and the adjacent marine areas of Queen Charlotte Sound and Hecate Strait. Those reports can be summarized as follows, based on the reports' abstracts.

Preikshot and others. Based on catches of juvenile Fraser River sockeye salmon in a trawl survey and smolt sampling, it was estimated that the average residence time of juveniles in the Strait of Georgia was 35 days.[383]

Beamish, Neville, and Sweeting. The synchronous poor early marine survival of virtually all the salmon species in the surface waters of the Strait of Georgia in the spring of 2007 indicates that there was a collapse of prey production for these species, resulting in the poor 2009 Fraser River sockeye returns. It is likely that climate and ocean conditions within the Strait of Georgia were responsible for the synchronous poor production of prey and resulting poor survival of Pacific salmon and herring.[384]

Beamish and others. Large abundances of juvenile Pacific salmon of all species enter the Strait of Georgia between late April and late May, but smaller abundances (e.g., Harrison River sockeye and South Thompson chinook populations) enter the strait six to eight weeks later. These late ocean-entry populations have higher productivity. Although the reasons for this higher productivity are not known, the researchers proposed that in recent years feeding conditions in the strait improved in July after many of the other juvenile Pacific salmon have emigrated or died.[385]

Thomson and others. The low Fraser River sockeye returns in 2009 indicated poor early marine survival of juvenile salmon in 2007, likely due to low food levels arising from unfavourable wind and river discharge conditions in the Strait of Georgia and the Queen Charlotte Sound–Hecate Strait region in the spring of 2007. Conversely, the high returns in 2010 were associated with a large smolt output from the Fraser River and good early marine survival in 2008, which was likely due to adequate food levels arising from favourable wind and runoff conditions in the spring of 2008. Even though the 2008 entry stocks were negatively influenced by the 2008–9 winter, the winter conditions in 2009–10 had a positive effect on these fish.[386]

During the evidentiary hearings, Dr. Richard Beamish, retired research scientist, Salmon and Freshwater Ecosystems, Pacific Biological Station, Science Branch, testified that his research indicated that Fraser River sockeye take about 35 days to migrate through the Strait of Georgia. He also described the Preikshot study, which concluded that the average residence time of juveniles in the Strait of Georgia was approximately 35 days.[387] In Dr. Beamish's view, this figure was consistent with several other estimates, although he agreed that there are still some uncertainties with this estimate.[388]

Dr. Welch testified that acoustically tagged smolts move about a body length a second, or 10 km a day.[389] On that basis, it takes a smolt 15–20 days to swim from the mouth of the Fraser River to the north end of the Strait of Georiga, and about the same time to reach the Queen Charlotte Sound.[390] He thought that the Preikshot estimate of 35 days to clear the Strait of Georgia (Exhibit 1305) was an overestimate.[391]

Dr. Beamish testified that in 2001 he and Dr. Connie Menken published an article (Exhibit 1308) espousing a critical size / critical period hypothesis:

> So in general, then, what we're saying is that juvenile salmon enter the ocean and have to grow quickly. There's large mortalities in that first up to six week period, and the fish that grow the fastest are the ones that are the larger ones, store energy and continue to store energy through the summer and survive the harsher conditions when feeding is less available, and prey are less available in the winter.[392]

Dr. Beamish also referred to his more recent study documenting a synchronous failure in juvenile Pacific salmon and herring production (Exhibit 1309). He said that during a 2007 trawl survey in the Strait of Georgia, he and his colleagues encountered extremely low abundances of herring and five salmon species (pink, sockeye, chinook, chum, and coho).[393] He could not think of another situation, anywhere, of such a synchronous failure in year-class strength.[394] They also found that coho and chinook had a high percentage of empty stomachs, although Dr. Beamish agreed that the sample sizes were small.[395] Dr. Beamish was directed to a study by Dr. Angelica Peña in which she stated that the distribution of phytoplankton and nitrate concentration during winter and spring of 2007 was similar to those observed in previous years.[396] He responded that he did not think that Dr. Peña, or any other scientist, was saying that phytoplankton production in the Strait of Georgia in 2007 was normal.[397]

Dr. Welch stated that his concern about focusing on the Strait of Georgia before establishing where the problem is means one may spend a great deal of energy on an area that is not the problem.[398] Dr. McKinnell agreed with the suggestion that we may never know what caused the 2009 decline.[499] Dr. Welch added that multiple explanations are still on the table.[400] Dr. Beamish said that sockeye entering the Strait of Georgia experienced very poor conditions for growth and survival, and then those conditions were exacerbated when they moved through Queen Charlotte Sound.[401]

Dr. McKinnell testified that, since completing his technical report, he has done further research showing that the British Columbia rivers which had the highest peak five-week discharge in the spring of 2007 were from Queen Charlotte Strait north.[402] The Fraser River was the 17th highest in the record.[403] He said he could use the term "extreme" for physical conditions in Queen Charlotte Strait and Sound, but not for anything he found when looking at the Strait of Georgia.[404]

Dr. Beamish also summarized Dr. Richard Thomson's study, which found extremely anomalous conditions in the Strait of Georgia in the spring of 2007: exceptional freshwater discharge, low surface salinity, a very shallow mixing layer depth, and winds blowing up the strait retaining the freshwater.[405] Dr. Beamish testified that these conditions match perfectly with what would affect prey production and result in the very poor survival that he and his co-authors had identified.[406] Conditions in Queen Charlotte Sound were also anomalous in 2007, an occurrence which resulted in Dr. Beamish's interpretation that juvenile sockeye entering the Strait of Georgia experienced very poor conditions for growth and survival, and that those conditions were exacerbated when the fish moved through Queen Charlotte Sound.[407] In fact, the poor conditions extended into that winter.[408] He agreed that conditions in Queen Charlotte Sound or in the Gulf of Alaska contributed to the extremely poor returns in 2009, and added that the critical size / critical period hypothesis acknowledges that mortality does not have to actually occur in the area where those conditions initially started.[409] Dr. Beamish was referred to the 2010 Pacific Salmon Commission hypothesis that "[o]cean conditions (physical and biological) inside Georgia Strait are important indicators of contributors to the Fraser sockeye situation," and the conclusion that the relative likelihood that this hypothesis caused observed changes in productivity during the long-term decline was "likely" and during 2009 was "very likely."[410] He agreed with this conclusion.[411]

Dr. Jim Irvine, research scientist, Salmon and Freshwater Ecosystems, Pacific Biological Station, Science Branch, testified that low returns for Chilko Lake in 2009 occurred despite huge freshwater survivals two years earlier, and that the low returns were caused by anomalously low ocean survivals – or at least smolt-to-adult survivals.[412] On the other hand, he explained, the good returns in 2010 were the result of high freshwater survivals in 2008; the ocean survivals were only average.[413] The anomalously low survivals for the 2007 ocean-entry-year fish could be due to some sort of major catastrophe occurring in some specific location (for which there is no evidence), or a cumulative effect of subnormal conditions at multiple life history phases – the sort of thing one expects to see occasionally in times of climate change.[414]

Harmful algal blooms

The term "harmful algae" includes any phytoplankton species that is harmful to marine organisms, humans, other animals, or the environment.* Blooms of species of the toxic alga *Heterosigma akashiwo* occur annually in British Columbia waters, particularly within the Strait of Georgia and Barkley Sound, and have been identified as a cause of net-pen salmon losses in British Columbia.[415] However, mortalities of sockeye salmon have not been directly attributed to this alga, so no causal link has been established.[416]

Dr. Jack Rensel provided expert testimony on harmful algal blooms (HABs).† He said there is evidence that HABs in the Strait of Georgia could have contributed to the poor 2009 Fraser River sockeye return and may have contributed to the longer-term decline in Fraser River sockeye productivity.[417] Dr. Rensel also said that there is general agreement in the fields of marine ecology and algal bloom science that a major worldwide increase is occurring in the frequency and intensity of harmful algal blooms.[418]

Dr. Rensel suggested that exposure of juvenile Fraser River sockeye to *Heterosigma* blooms could result in direct, acute effects or in chronic effects such as infections, making the fish more susceptible

* Harmful algae do not necessarily cause harm in low concentrations, and they are sometimes important components of the marine food web. See PPR 19, p. 72.

† Dr. Rensel was qualified as an expert in algal zooplankton and marine and freshwater habitats; harmful algal bloom dynamics; monitoring and mitigation studies; and fish physiology studies, bioassays, and fish kill assessments (Transcript, August 17, 2011, p. 4). His curriculum vitae is Exhibit A to Exhibit 1363.

to poor food supply conditions and predation.[419] Adult Fraser River sockeye, Dr. Rensel suggested, are at risk of acute or chronic effects from *Heterosigma* because they swim at shallower depths on return to the river.[420] Dr. Rensel cautioned that *Heterosigma* blooms could have an impact in combination with diseases, low food availability, and other stressors.[421] There are a number of ecotypes of *Heterosigma*, as well as other algal species that may be harmful to Fraser River sockeye.[422]

In the June 2010 PSC workshop proceedings, harmful algal blooms in the Strait of Georgia were rated as a "possible" contributor to the poor 2009 return and as an "unlikely to possible" contributor to the longer-term decline.[423] Dr. Rensel did not agree entirely with the conclusions reached by the workshop because he had already left it when the ratings were applied and there was no expert present to argue for the HABs theory.[424] I note, however, that David Marmorek, who worked on algal blooms and contaminants with Dr. Rensel at the PSC workshop, testified that a lot of consideration went into the ratings.[425] According to Dr. Rensel, all the evidence presented for the various hypotheses was correlational, so if voting were based just on correlations, the harmful algal bloom hypothesis should have had a "very likely" rating.[426]

One of Dr. Beamish's expert reports appears to discount Dr. Rensel's *Heterosigma* theory.[427] Dr. Rensel explained that after this report was tendered into evidence, he corresponded with the lead author, Dr. Beamish.[428] According to Dr. Rensel, Dr. Beamish's discounting of the Rensel theory was based on a misunderstanding about where the sampling for *Heterosigma* occurred in 2007, and Dr. Beamish would be revising his paper to account for this misunderstanding.[429]

Although harmful algal blooms were seen in the Strait of Georgia in 2008, the 2010 Fraser River sockeye return was very good. In response, Dr. Rensel said that the timing of the bloom in 2008 was later than in 2007, so most Fraser River sockeye would have already left the strait.[430]

Pulp and paper mills

Pulp mills also operate on the shores of the Strait of Georgia and in other marine areas through which Fraser River sockeye may migrate. Six pulp and paper mills operated in the Strait of Georgia between 1990 and 2010.[431] In 2003, mills were still operational at Port Mellon and Squamish on the mainland as well as at Elk Falls, Gold River, Harmac, and Port Alberni on Vancouver Island.[432] The Squamish and Elk Falls mills closed in 2006 and 2009, respectively.[433] All these mills are subject to the *Pulp and Paper Effluent Regulations*.[434]

Infectious diseases

As discussed earlier for the freshwater life history stages, Dr. Kent, the author of Technical Report 1, Infectious Diseases, documented and evaluated the potential effects of diseases and parasites on Fraser River sockeye salmon during both the freshwater and the marine life stages. He identified the following pathogens as either high or moderate risk to Fraser River sockeye salmon during the marine life stages.

Viruses. The infectious hematopoietic necrosis (IHN) virus causes severe, acute systemic disease in juvenile salmonids. According to studies in 2002 and 2006, it is highly pathogenic to Atlantic salmon in seawater pens, and outbreaks occur occasionally in British Columbia. The virus has been detected in adult sockeye in seawater. Dr. Kent concluded that the risk was high, noting that sockeye in seawater are susceptible, although the virus at this stage is less virulent because older and larger fish show fewer mortalities when they become infected.[435] During the evidentiary hearings, Dr. Kent testified that the infectious salmon anemia virus (ISAv) occurs in other parts of the world and can cause a serious disease in salmonid fish, but at the time of his report it had never occurred in British Columbia.[436] Dr. MacWilliams added that ISAv has been shown to cause natural infections in marine-farmed Atlantic salmon. Under laboratory conditions, it has been possible to experimentally infect other species, such as rainbow trout, but work done on Pacific salmon has shown that they are relatively resistant to the disease.[437] (See also the discussion below on infectious salmon anemia virus.)

Bacteria. Renibacterium salmoninarum causes bacterial kidney disease (BKD) in salmonids. Infections are contracted and spread in marine as well as freshwater, apparently by oral-fecal transmission, and fish die from a few weeks to months following infection. Sockeye are highly susceptible. The researcher rated the risk as high, noting that the infection progresses after infected smolts migrate to seawater.[438] During the evidentiary hearings, Dr. MacWilliams testified that this bacteria infects the host's immune cells, which can make the host susceptible to other diseases.[439] With *Aeromonas salmonicida*, the infection occurs in both wild and cultured fish in British Columbia, and can result in an acute, severe disease with high mortality. The bacterium is transmissible in seawater from fish to fish, and is recognized as a potentially serious disease in net pen–reared Atlantic salmon. Dr. Kent rated the risk as high. The *Vibrio anguillarum* marine bacterium causes vibriosis in unvaccinated smolts shortly after entry into seawater. It can cause severe disease in seawater pen–reared fish, whereas documentation of mortality in wild salmonids in seawater is less definitive. Infections are exacerbated by chemical pollutants or co-infections by parasites. The researcher rated the risk as high.[440]

Myxozoa. There have been numerous reports of a high prevalence of the infection of the *Parvicapsula minibicornis* parasite in adult sockeye in the Fraser River, as well as in outmigrating smolts. The infection is chronic, and it targets the kidneys. The researcher rated the risk as high, stating that it occurs in smolts shortly after seawater entry but is not detected in older fish in seawater. Thus, assuming that fish do not spontaneously recover, this myxozoan is linked to parasite-associated mortality in seawater.[441]

Helminths (worms). The adult stages of tapeworms of the *Eubothrium* genus are found in the intestine and stomach of juvenile sockeye salmon. Heavy infections may reduce swimming, stamina, growth, survival, and saltwater adaptation, and may alter migration orientation. The researcher rated the risk as moderate.[442]

Parasitic crustaceans (sea lice). Marine parasitic copepod *Lepeophtheirus salmonis* has for many years been reported on all salmon species from the oceans. Articles claim that it causes significant mortality in pink and chum salmon in British Columbia, particularly associated with fish farms, although other research has failed to support the claims of fish farm–caused mortality. A 2010 paper found an association between sea lice on farms and on wild juvenile salmon, but not a negative association with overall survival of the latter.[443] A 2008 study concluded that, based on experimental studies with pink salmon, elevated risk associated with *L. salmonis* occurs only in fish weighing less than 0.7 grams, while sockeye smolts are much larger when they enter seawater.[444] Hence, reports of infections on sockeye are not a direct indication that the parasite causes significant mortality in this species. *Caligus clemensi* is another caligid copepod that infects both farmed and wild salmon in British Columbia. Being smaller, on a per parasite basis it is considered less pathogenic. Another concern with parasitic copepods is that they are potentially vectors for other pathogens. *Caligus* spp. are capable of moving from host to host, and a 2008 study showed that *L. salmonis* may move to predator salmon when infected prey are eaten. The researcher rated the risk as moderate, noting that significant mortality has not been documented in sockeye salmon.[445] (See also the discussion below on sea lice and Fraser River sockeye.)

The researcher said that the state of the science for understanding the impacts of pathogens on wild salmon in British Columbia is minimal. It is also difficult to study the impacts of diseases on wild fish, particularly in the marine environment.[446] He added, "[W]e cannot conclude that a specific pathogen is the major cause of demise to the Fraser River sockeye salmon. However, pathogens cannot be excluded at this time as adequate research on the impacts of disease on this population has not been conducted."[447]

Dr. Kent did not consider the question of whether diseases found in fish farms are transmitted to wild fish because he was instructed that those issues would be explored by the technical reports on aquaculture.[448]

Infectious salmon anemia virus

As discussed in Volume 1, Chapter 9, Fish health management, infectious salmon anemia virus (ISAv) is an orthomyxovirus that infects fish and can cause a systemic and lethal disease known as infectious salmon anemia (ISA) in Atlantic salmon.[449] ISAv is most commonly found in farmed Atlantic salmon, though it has also been found in other species of wild fish and has caused infection in experiments with steelhead trout, chum, and coho.[450] Outbreaks of ISA have occurred on Atlantic salmon farms around the world, including in eastern Canada, the eastern United States, Norway, Chile, the United Kingdom, and the Faroe Islands.[451] The virus may transmit from one fish to another through exposure to organic material (such as blood) containing ISAv, exposure to sea lice from ISAv-infected fish, and exposure to ISAv particles shed into seawater.[452]

To assess what role, if any, ISAv may have had in relation to the decline of Fraser River sockeye salmon, two issues must be considered: (1) whether ISAv is present in BC waters, and (2) whether ISAv is capable of causing disease and death in Fraser River sockeye.

Whether ISAv is present in BC waters

During the hearings on disease in August 2011, I heard testimony from Dr. MacWilliams, Dr. Kent, and Dr. Gary Marty, fish pathologist, Animal Health Centre, BC Ministry of Agriculture, that there had been no confirmed cases of ISA or ISAv in British Columbia.[453] Scientific technical reports prepared for the Commission by Dr. Kent, Dr. Lawrence Dill, professor of biological sciences at Simon Fraser University, and Dr. Donald Noakes, professor of mathematics and statistics and the associate vice-president of research and graduate studies at Thompson Rivers University, reflect a similar understanding.[454] Indeed, in Technical Report 1, Infectious Diseases, Dr. Kent does not list ISAv in his review of pathogens of concern to Fraser River sockeye at all.[455] Its absence reflected the understanding that this pathogen had not been identified as being present in BC waters.

I reopened the hearings in December 2011 to hear additional evidence related to reports of ISAv in British Columbia salmonids, including Fraser River sockeye. A panel of experts on molecular genetics and salmon diseases (including ISAv) appeared before me and could not come to agreement on whether or not ISAv or an ISAv-like virus is present in BC salmon:

- Dr. Frederick Kibenge, chair, Department of Pathology and Microbiology, Atlantic Veterinary College, University of Prince Edward Island, told me that there was "overwhelming" evidence that there is an orthomyxovirus present, and that the reports of ISAv may be "ISA virus sequences or it may be ISA virus-like."[456]
- Dr. Miller expressed her "clear belief" that there is a virus present, which is "very similar to ISA virus in Europe" although she acknowledged that further work would be required to understand just how similar.[457] She also told me that the ISAv or ISAv-like sequences are present in fish archives dating back to 1986 and hypothesized that they have been in Pacific waters longer than that.[458]
- Dr. Are Nylund, professor, University of Bergen, told me that although there had been "a lot of indications" that ISAv could be present in Pacific salmon, he had not yet seen any "hard evidence" of its presence.[459]
- Nellie Gagné, molecular biology scientist and laboratory supervisor, Gulf Fisheries Centre, DFO, expressed her view that there was not enough information available to conclude that ISAv or another orthomyxovirus was present. More work would be necessary to make such a determination.[460]

Although they did not agree on whether ISAv or an ISAv-like virus was present, all agreed that more research was necessary and that, whether ISAv is present or not, there had been no confirmation of ISA in any of the Pacific salmon tested by the time of the December hearings.[461] As stated in Volume 1, Chapter 9, Fish health management, I find that the evidence does not allow me to conclude whether ISAv or an ISAv-like virus currently exists in Fraser River sockeye. However, I accept the opinion of the expert panel (Dr. Kibenge, Dr. Nylund, Ms. Gagné, and Dr. Miller) that, as of December 2011, there was no evidence that fish tested for ISAv (the virus) suffered from ISA (the disease) as that disease was then understood.

Whether ISAv is capable of causing disease in Fraser River sockeye

Evidence of whether ISAv, if present, is capable of causing disease in Fraser River sockeye is mixed. Dr. Dill wrote in Technical Report 5D, Dill Salmon Farms Investigation, that "a close watch should be kept for indications of this disease [ISA], and biosecurity [on salmon farms] rigidly enforced, since ISA could be devastating to BC wild salmon populations."[462] In contrast, Dr. Kibenge, whom I qualified as an expert on ISAv, testified that the disease ISA can be found only in farmed Atlantic salmon and that, as far as he knew, Pacific salmon are not known to develop ISA.[463]

Published scientific literature in evidence before me appears to support the contention that existing, known strains of ISAv do not pose a significant risk to Pacific salmon. In a 2003 report by Rolland and Winton published in the *Journal of Fish Diseases*, the authors injected steelhead trout, chum, chinook, coho, and Atlantic salmon with a known dosage of Norwegian-strain ISAv.[464] The authors found that Pacific salmon are "considerably more resistant to ISAv compared with their Atlantic counterparts" and suggested that "Pacific salmon species are at a relatively low risk should ISA spread to the west coast of North America where these species are endemic."[465] However, they also cautioned that "the potential for ISAv to adapt to *Oncorhynchus* spp. should not be ignored."[466]

A more recent (2007) article by MacWilliams and others, published in the journal *Diseases of Aquatic Organisms*, similarly reported that another member of the *Oncorhynchus* species, rainbow trout, was "highly resistant to developing ISA" following injection with a known ISAv strain.[467] However, this report also documented a unique histopathic lesion occurring in some of the rainbow trout infected with ISAv, which was not previously associated with ISA in Atlantic salmon.[468] The authors report their "considered opinion that this unique lesion is characteristic of fatal ISAv-infection of rainbow trout."[469] The lesions found in rainbow trout in response to ISAv infection were so unique that "if these lesions were viewed in rainbow trout under field conditions, it is unlikely that ISA would have been considered as a differential diagnosis."[470] I take this report as another example of the relative resistance of Pacific salmon to known strains of ISAv, and that ISAv may produce different symptoms among the various species of salmon.

Because the experiments by Rolland and Winton and the article by Dr. MacWilliams involved known strains of ISAv, they provide limited insight into how an endemic or "Pacific-region" strain of ISAv, if it exists, may affect Pacific salmon. Further, neither of these studies involved sockeye.

Unpublished genomic expression research by Dr. Brad Davis, a post-doctoral fellow in Dr. Miller's molecular genetics laboratory, suggests that the Pacific salmon for which Dr. Miller obtained presumptive positive ISAv tests during the fall of 2011 may be exhibiting an influenza-like host response.[471] Dr. Davis's report suggests that "we cannot at this point assume that this virus does not cause disease to these fish" and recommends follow-up laboratory experimentation.[472] According to Dr. Miller, Dr. Davis's preliminary results indicate that fish testing presumptively positive for ISAv sequences in her lab were exhibiting "some level of damage" even though disease and mortality had not been demonstrated.[473] I note that by focusing on the host response, Dr. Davis's results and Dr. Miller's interpretation do not appear to hinge on whether the ISAv sequences detected are associated with a known strain, or newly discovered strain, of ISAv.

Finally, I also heard of the potential for ISAv, if present, to mutate into a form that may cause Pacific salmon mortality. Dr. Nylund told me that, although there was insufficient information to know if a Pacific ISAv or North Atlantic ISAv strain was present in British Columbia waters, "if it is a North Atlantic ISA virus, of course it can mutate into a pathogenic strain."[474] However, both Dr. Kibenge and Dr. Miller stated that, with the information currently available, it was not possible to say what degree of damage such a mutation could do.[475] The 2003 Rolland and Winton article, described above, suggests that the ability of Pacific salmon to carry ISAv in those trials "indicates that it would be unwise to overlook the possibility of ISAv replicating in, or establishing a carrier status among these species should they be exposed to the virus" and that genetic mutations "may result in the evolution of strains with differences in host ranges, virulence or immune response to vaccines."[476]

Sea lice

Fraser River sockeye smolts enter seawater free of sea lice. In the marine environment, they are exposed to sea lice from infected fish, either wild or farmed.

Sea lice are small marine parasitic copepods, visible to the naked eye. As noted above, there are two species that infect Fraser sockeye: *Lepeophtheirus salmonis* (the "salmon louse," or "*Leps*"), and *Caligus clemensi* (the "herring louse," or "*Caligus*"), shown in Figure 2.4.1. The sea lice life cycle includes non-parasitic stages in which the sea lice disperse in the water; several infective stages during which the louse is tethered to its host by a frontal filament; and two pre-adult stages and one adult stage, which are motile (i.e., the louse can move on the surface of the fish and even move from fish to fish).[477] Sea lice feed on the fish's superficial tissues (mucus and skin) and blood.[478] A document in evidence written by Dr. Simon Jones, research scientist, Aquatic Animal Health Section, Science Branch, described their impact: "The impact of sea lice on individual fish is highly variable and ranges from mild stress, osmotic imbalance due to skin damage, increased susceptibility to other infections, impaired swim performance and in severe cases, death."[479]

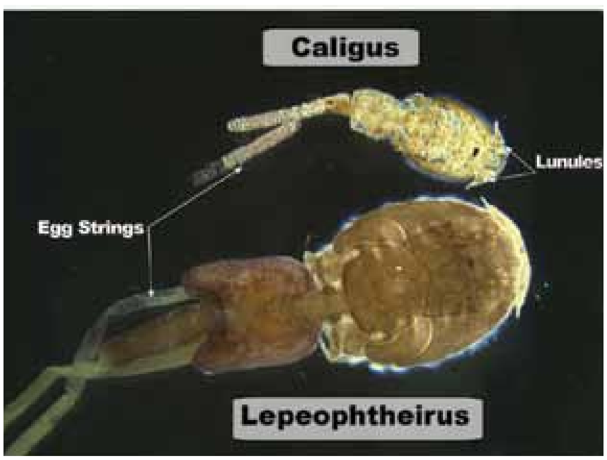

Figure 2.4.1 Pacific sea lice species (enlarged; scale unknown)

Source: Exhibit 1780, p. 3.

Dr. Kent, author of Technical Report 1, Infectious Diseases, rated sea lice as a "moderate" risk to Fraser River sockeye.[480] He cited work by DFO researchers showing that, in a sockeye survey conducted in May and June 2010, the sockeye had a 4 percent prevalence of *Leps* infection (with an abundance of less than one louse per fish) and 70 percent prevalence (number of fish infected) of *Caligus* infection (with an abundance or intensity of between one and 16 lice per fish).[481] Dr. Kent also noted that sea lice are possibly vectors for other pathogens, and are possibly responsible for significant mortalities in pink salmon.[482]

The authors of technical reports 5C, Noakes Salmon Farm Investigation (Dr. Noakes) and 5D, Dill Salmon Farm Investigation (Dr. Dill), both of which are described below (Salmon farms), discussed sea lice in relation to salmon farms. Additionally, during the hearings on aquaculture, I heard from a panel of expert witnesses on sea lice: Dr. Jones; Dr. Orr; Michael Price, biologist with Raincoast Conservation Foundation; and Dr. Sonja Saksida, a fish veterinarian with a private practice and the executive director of the Centre for Aquatic Heath Sciences.* These experts told me about genetic differences between Pacific and Atlantic sea lice; the sources of sea lice that infect Fraser River sockeye; the effect of sea lice infection on Fraser sockeye (at individual and population levels); the potential for sea lice to act as vectors to transfer other pathogens among fish; and the overall risks posed to Fraser River sockeye from sea lice.

Genetic differences in sea lice

The species of *Caligus* that infects salmon and other fish species in British Columbia occurs only in the northeastern Pacific Ocean.[483] In contrast, the species of *Leps* is present in both the Pacific and Atlantic oceans. Dr. Jones told me that there are genetic differences between *Leps* from the Pacific and Atlantic oceans.[484] Farmed Atlantic salmon raised in net pens in the Pacific Ocean have a lower instance of pathology and disease when infected with *Leps* when compared with farmed salmon in the Atlantic Ocean.[485] Dr. Saksida also testified that, based on her observations as a veterinarian working at aquaculture facilities, sea lice in the Atlantic Ocean cause more damage to farmed Atlantic salmon than lice in the Pacific Ocean.[486] Dr. Jones also testified that the Pacific salmon louse behaves differently on different

* I qualified all four witnesses as experts in their field. Specifically, Dr. Jones was qualified as an expert in parasitology and immunology with a specialty in sea lice and diseases of salmon, including as this relates to farmed and wild salmon (Transcript, September 6, 2011, p. 2); Dr. Orr was qualified as an expert in ecological sciences with a research focus on sea lice affecting farmed and wild salmon; Mr. Price was qualified as an expert in juvenile salmon ecology in relation to sea lice infestation; and Dr. Saksida was qualified as an expert in veterinary medicine and veterinary epidemiology with a specialty in fish health.

species of Pacific salmon and on Atlantic salmon.[487] All these differences require "research in British Columbia that is distinct and separate from research that's undertaken in Europe."[488] Dr. Orr said differences in pathogenicity between Atlantic and Pacific *Leps* is based on speculation and that experimentation would be useful to clarify this hypothesis.[489]

Sources of sea lice on Fraser River sockeye

Salmon are the primary hosts for *Leps,* though some life stages of *Leps* have also been found on three-spine stickleback.[490] Dr. Dill, the author of Technical Report 5D, Dill Salmon Farms Investigation, said that, in the Broughton Archipelago region of the British Columbia coast, it is uncontroversial to say that salmon lice on wild fish are coming from the salmon farms.[491] Dr. Noakes, author of Technical Report 5C, Noakes Salmon Farms Investigation, agreed, saying, "I don't think there's any question that some of those lice are coming from the farms and onto pink salmon, but there are other hosts there, as well."[492] Dr. Noakes said that, while no adult lice have been found on sticklebacks, "that doesn't mean that they aren't competent for infecting other fish, in terms of having motile lice."[493] Dr. Orr commented that there is no evidence that alternative sources of lice, such as sticklebacks, are "anywhere near the magnitude of the source of lice from the farms."[494] In relation to the Fraser sockeye migration route, Atlantic farmed salmon are a source of *Leps* infecting Fraser River sockeye in the Discovery Islands.[495]

Although salmon farms may be a source of *Leps* infecting Fraser River sockeye, both Dr. Jones and Mr. Price testified that *Caligus* is the dominant louse species infecting juvenile sockeye migrating through the Strait of Georgia and the Discovery Islands.[496] *Caligus,* while found on salmon farms, is also found on other hosts besides salmon – herring, in particular.[497]

Mr. Price described a peer-reviewed study he conducted to look at whether salmon farms caused increased sea lice infection on migrating Fraser River sockeye in the Discovery Islands.[498] He testified that, in 2007, lice levels (for both species of lice) on Fraser River sockeye found downstream of salmon farms were an order of magnitude higher than on sockeye in a comparison area on the North Coast (an area with no salmon farms).[499] Further, within the Discovery Islands, lice levels on juvenile sockeye were significantly higher *after* they passed fish farms.[500] In 2008, he noticed a similar pattern, though "the differences between upstream and downstream [of salmon farms] were not as clear."[501] Mr. Price said the "lice levels on farmed fish at the time within the Discovery islands, inter-annual variation between the lice levels of *Caligus clemensi* … and *Lepeophtheirus salmonis* … matched the inter-annual variation we saw on juvenile sockeye."[502] He testified that weight or size of the fish did not seem to be a factor relating to lice level, and that salinity and temperature were accounted for in his modelling.[503] He concluded that "position relative to farms was the best predictor of lice levels on juvenile sockeye."[504]

Dr. Jones and Dr. Saksida were both critical of Mr. Price's paper, as were Dr. Noakes in Technical Report 5C, Noakes Salmon Farm Investigation, and Dr. Stewart Johnson, who testified during the hearings on disease.[505] Dr. Jones said he "felt that the conclusions that farms were the only source of the infections … were not always supported by the observations that I saw presented in the paper."[506] More specifically, while there were similar levels of *Caligus* and *Leps* on the farms, after the sockeye passed the farms, their *Caligus* levels appeared to be 4.8 times higher but their *Leps* levels only 1.14 times higher.[507] Also, there were very few *Caligus* on the farms in 2008. Dr. Jones proposed an alternative hypothesis: that the sockeye were infected with *Caligus* from another fish source, such as herring, possibly in the Strait of Georgia before reaching the fish farms.[508] In response, Mr. Price said that if herring or other fish were a source of the lice on the fish farms, then they would "need to assume a similar spatial distribution as the salmon farms in this region. We see no evidence of that."[509] Mr. Price also noted the possibility that sockeye are more resistant to *Leps* and more susceptible to *Caligus,* explaining the differences in lice species abundance on sockeye downstream of farms.[510]

Although Dr. Saksida agreed with Mr. Price's suggestion that sockeye salmon may be more resistant to the salmon louse (*Leps*) than to *Caligus,* she criticized Mr. Price's paper for excluding data from an "outlier" sample site upstream of the fish farms.[511] In 2008, the outlier had some of the highest sea lice prevalence (number of fish infected) and intensity (number of lice per infected fish).[512]

In response, Mr. Price said he ran the analyses with and without the outlier site, and the results remained the same.[513]

Dr. Jones testified that DFO began a "field surveillance effort" on Fraser River sockeye in 2010 (see Volume 1, Chapter 9, Fish health management) as well as laboratory studies. He said that in 2010, as noted by Dr. Kent (above), DFO looked at 300 sockeye and found that 70 percent of them were infected with *Caligus*, and 3 to 4 percent of them were infected with *Leps*.[514] He said that early analysis of the data suggests that, for *Caligus*, "there is a strong relationship between the level of lice on the sockeye and the distance that they've migrated from the Fraser River. In other words, the time spent in the ocean seems to be a strong determinant of the level of infection."[515]

Effects of sea lice on Fraser River sockeye

As noted above, the effect of sea lice on individual fish is variable; fish can exhibit mild stress to mortality. *Leps* infections of unusually high intensity (e.g., an average of 300 lice per fish) can result in significant pre-spawn mortality for adult returning sockeye.[516] (See the discussion in Life stage 5: return migration, below.) The stages of salmon lice that have the most pathogenic effect on individual fish are the sub-adult and adult lice – the motiles.[517] No studies have been done to show the effects of *Caligus* on sockeye, though researchers hypothesize they have a lesser impact on sockeye than do *Leps*.[518]

Dr. Jones emphasized that sea lice infections on sockeye in the Strait of Georgia are very different from those on juvenile pink and chum in the Broughton Archipelago.[519] More research has been conducted in the Broughton Archipelago, and several scientific papers were presented to me in evidence.[520] *Caligus* as opposed to *Leps* is the dominant species on Fraser River sockeye.[521] Dr. Jones also said that sockeye salmon are in most cases a year older than pinks when they enter the marine environment: "They're a larger fish with a more mature scaled skin, and I would expect that that would confer to the sockeye salmon some level of resistance. I would be hesitant to extrapolate further."[522] Laboratory studies on such subjects as critical thresholds of infection and resilience related to size of the fish are not available for sockeye.[523] However, scientists have done some work on other species of salmon.[524] Dr. Jones emphasized that "you can't treat all salmon equally," and that that to determine whether a particular species of salmon is resistant or susceptible to infection by sea lice requires doing the work in relation to that species.[525] Still, Dr. Jones testified, "Certainly the fact that 70 percent of juvenile sockeye salmon that we've seen in our surveys have *Caligus* infestations to me tells me that there will be a cost associated with those infestations."[526]

A presentation by Dr. Jones suggests the following specific research needed to test the hypothesis that sea lice contributed to the decline of Fraser River sockeye:

- Establish inter-annual variations in sea lice levels for juvenile sockeye in the Strait of Georgia.
- Establish inter-annual variations of infections with bacteria, virus, or other parasites.
- Determine lethal and other impacts of sea lice on individual sockeye salmon in controlled laboratory experiments.
- Integrate and analyze health data from farmed and wild salmon in the Strait of Georgia and elsewhere to obtain a global assessment of pathogen dynamics.[527]

Dr. Orr said he would be more concerned about the effects of sea lice on sockeye "if we saw higher numbers of *Leps*," but that we cannot "discount the issue of *Caligus*."[528] He noted that sea lice can have behavioural influences on fish and referred to work on pink salmon which shows lice can cause infected fish to be more vulnerable to predators.[529] He also noted the need for more research into the effects of *Caligus* on sockeye.[530]

Sea lice as a disease vector

Dr. Saksida said that sea lice "are potentially a mechanical vector" for bacteria or viruses, but that these micro-organisms do not likely replicate on the sea lice – just sit on the animal or pass through their digestive tracts.[531] She also testified that the fish diseases of main concern are transmitted through water: "So if the fish are close enough to have a sea louse jump between one fish and another fish, if they're motile sea lice, then there would be water-borne exposure. So I would say that the water-borne exposure is far more significant than any effect that a sea louse would have."[532] Similarly, both Dr. Noakes and Dr. Dill agreed that there is potential for sea lice to serve as vectors for other pathogens, but this is more of an "accidental" vectoring because pathogens do not need the lice as a means of transmission.[533] Dr. Jones said most of the research for lice as vectors has been done on *Leps*.[534]

Level of risk and contribution to the decline

Most of the researchers whom I heard from agreed that sea lice are not directly implicated in the decline of sockeye salmon.[537]

Dr. Jones said sea lice pose a "low risk to moderate risk to sockeye salmon associated with all species of sea lice."[536] He said that, while *Caligus* probably does not pose a threat, *Leps* "has a greater potential to cause harm, and that every effort to manage *L. salmonis* on salmon farms would be appropriate in terms of minimizing that risk."[537] Dr. Saksida also said the risk to sockeye from *Caligus* is "low to moderate" and that more research is required on the distribution of *Caligus*.[538] She thought it unlikely that sea lice contributed to the decline in sockeye productivity.[539] Dr. Orr agreed that there is a "low to moderate risk of mechanical damage" from sea lice to sockeye.[540] However, he also said the possibility for sea lice to act as a disease vector means there is "a fairly high risk" if the lice levels on salmon farms are not controlled.[541]

Mr. Price said the risk from sea lice to sockeye salmon is "fairly high" in combination with other factors such as food limitations or increased predation risk."[542] Although Mr. Price said, "I don't believe sea lice acting in isolation are responsible for the decline in sockeye productivity,"[543] he stated that "factors rarely act in isolation on the population dynamics of a species," and *Leps* or *Caligus* acting with other factors to stress sockeye juveniles "may be a contributing factor to not only productivity declines but also during that 2009 return or the low return."[544]

Salmon farms along the migratory route

Outmigrating smolts encounter salmon farms during their migration through the Discovery Islands toward Johnstone Strait. They may also encounter salmon farms if migrating up the west coast of Vancouver Island. The authors of technical reports 5C, Noakes Salmon Farm Investigation, and 5D, Dill Salmon Farm Investigation, Dr. Noakes and Dr. Dill, independently reviewed and evaluated the effects of salmon farms on Fraser River sockeye salmon.* They considered impacts on Fraser River sockeye from sea lice exposure, from farm wastes that affect benthic and pelagic habitat quality, from Atlantic salmon escapees, and from disease.

To facilitate their work, the author of Technical Report 5A, Salmon Farms and Sockeye Information, Dr. Josh Korman, fisheries ecologist with Ecometric Research,† summarized the "spatial and temporal trends for some important elements of the salmon-farming data, such as sea lice abundance and the frequency of bacterial and viral diseases, which could affect wild sockeye salmon."[545] Dr. Korman used data disclosed to the Commission from past BC Ministry of Agriculture (BCMAL) salmon farm audits and the B.C. Salmon Farmers Association (BCSFA) fish health database, and DFO data on Atlantic salmon escapes from salmon farms.[546]

Also to assist in the analyses of Dr. Noakes and Dr. Dill, the author of Technical Report 5B, Salmon Farms and Sockeye Relationships, Dr. Brendan Connors, post-doctoral fellow in the School of Resource and Environmental Management at Simon Fraser University,‡ statistically examined the "relationship between salmon aquaculture in British Columbia and Fraser River sockeye salmon

* Dr. Noakes was qualified as "an expert in salmon population dynamics, including wild salmon / farmed salmon interactions, fisheries climate interactions and in statistical analysis including time series analysis" (Transcript, August 25, 2011, p. 69). Dr. Dill was qualified as "an expert in behavioural ecology, predator / prey relationships and ecological factors affecting wild fishes, including parasites and fish farms" (Transcript, August 25, 2011, p. 71).

† Dr. Korman was qualified as "an expert in salmonid stock assessment and in statistical analysis, in particular of population level fisheries data" (Transcript, August 25, 2011, p. 62).

‡ Dr. Connors was qualified as "an expert in statistical analysis, fish population dynamics with a particular research emphasis on wild salmon / farmed salmon interactions" (Transcript, August 25, 2011, p. 74).

population dynamics."[547] Dr. Connors used sockeye production data compiled by Dr. Randall Peterman and Dr. Brigitte Dorner in Technical Report 10, Production Dynamics, as well as various salmon farm variables obtained through the Commission's disclosure from BCMAL, BCSFA, and the BC Ministry of Environment.[548]

Although Dr. Noakes and Dr. Dill came to similar conclusions on the effect of many of the specific potential stressors related to salmon farms, they disagreed about the risks posed from disease on salmon farms and about the significance of Dr. Connors's conclusions.

Effects of specific stressors

In their reports and during testimony, Dr. Dill and Dr. Noakes discussed a number of specific stressors stemming from salmon farms, summarized below.

Physical infrastructure. In his report, Dr. Dill wrote that "[f]arm structures themselves can create backeddies in fast flowing channels, perhaps encouraging juvenile sockeye to rest there during migration."[549] Lights from farms (used to encourage growth of farmed fish) could attract sockeye as well as other fish that are their predators or competitors.[550] If farmed fish were infected with disease or sea lice, it could increase the risk of infection to wild sockeye by bringing them into closer proximity to farmed salmon.[551] In his report, Dr. Noakes did not comment on physical infrastructure of salmon farms.

Effect of farm waste on benthic and pelagic habitat. Both Dr. Dill and Dr. Noakes agreed that the effects of waste from fish farms on both benthic and pelagic habitat is unlikely to be driving declines of Fraser River sockeye – that effects "do exist locally" but are probably not sufficient in geographic scale or impact to have been drivers of the decline.[552] However, Dr. Dill explained, there have been no studies of cumulative impacts of the large number of farms the sockeye have to pass in the Discovery Islands.[553]

Chemical inputs. Similarly, chemical inputs including therapeutants such as SLICE (used to treat sea lice in farmed salmon) are unlikely to have anything but a local environmental effect.[554] (See Volume 1, Chapter 9, Fish health management, and Chapter 7, Enforcement.) Moreover, such effects are likely limited to invertebrate species.[555] They are unlikely to have any population-level effects on sockeye.[556]

Atlantic escapees. Both Dr. Dill and Dr. Noakes agreed that "it's very unlikely that escapees are implicated in the decline of Fraser River sockeye salmon."[557] Atlantic escapees are not spawning in streams occupied by Fraser River sockeye, and they are not competing with Fraser River sockeye for food.[558] Very few Atlantic salmon have been found in the lower Strait of Georgia and the Fraser River.[559] Dr. Dill acknowledged a "slight potential for disease to transfer to wild sockeye via escaped Atlantics."[560] Dr. Noakes said "it is far more likely that farms would be a more viable source of pathogens than chance encounters between Pacific salmon and escaped Atlantic salmon."[561]

Sea lice – direct effects. Both Dr. Dill and Dr. Noakes agreed that there was insufficient evidence to conclude that sea lice from salmon farms were linked to the overall decline of Fraser River sockeye.[562] Dr. Korman, who synthesized fish farm data related to sea lice, testified there was "nothing unusual about 2007" that would explain the poor run in 2009, and "pretty similar conditions in the fish farms" in 2008 – so nothing to explain the exceptional run in 2010.[563] With respect to *Leps*, Dr. Dill testified that "migratory allopatry" is interrupted by the existence of salmon farms on wild salmon migration routes. He said that migratory allopatry means that wild adult fish make their return migration at a different time from the smolts' outmigration, so the adult and juvenile fish are not passing one another in the water.[564] Fish farms "close the loop" by providing a reservoir of adult salmon hosts for *Leps* that can then infect juvenile salmon during their outmigration, before they would normally encounter adult salmon and their associated parasites.[565] (See the discussion above related to sea lice.)

Sea lice – as vectors for disease. Both Dr. Dill and Dr. Noakes agreed that sea lice could serve as vectors for other diseases and pathogens from fish farms, but that this may be an incidental vectoring; transmission of pathogens through water may be more effective.[566] (See the discussion above related to sea lice.)

Disease from fish farms. Dr. Dill said of all the fish farm stressors, "disease" was the most likely candidate to provide a mechanism for a negative impact of salmon farms on sockeye productivity.[567] (As discussed below, Dr. Dill concluded that salmon farms do have "some sort" of negative impact on Fraser River sockeye.) Dr. Dill testified that having open-net pens in the ocean is a "game changer" in terms of the disease environment.[568] He said that fish farms can result in biomagnifications of pathogens, and that the high densities of hosts on fish farms "are likely to select for fast-growing, early-transmitted and more virulent pathogens."[569] He also set out several possible routes of transmission from farmed to wild fish: direct horizontal transfer through the water, through fish feces in the benthos, transmission from escaped Atlantic salmon, transmission by sea lice, and discharges of bloodwater from processing plants.[570] Despite the potential for diseases to have a negative impact, Dr. Dill noted that incidents of three of the diseases that Dr. Kent rated as "high risk" to Fraser River sockeye – furunculosis, BKD, and IHN – are not associated with sockeye survival in Dr. Connors's analysis (discussed below).[571]

Dr. Noakes reported that "[a]ll of the diseases found on salmon farms are common in BC and there is no evidence that any exotic pathogens or diseases have been introduced by the salmon farming industry."[572] He also noted that, of the roughly 32 million fish on BC salmon farms, about 3 million die each year (less than 10 percent).[573] Only about 2 percent (about 600,000 fish per year) are "fresh silvers," of which some unknown percentage died of disease.[574] (Other causes of death include predators and environmental factors such as algae or low dissolved oxygen.)[575] In Dr. Noakes's view, the mortality rate for fresh silvers has been "pretty low" when compared with a mortality rate of about 3 percent per day for juvenile wild salmon.[576] Dr. Noakes identified a significant problem in trying to assess the impact of disease on fish farms on the survival of sockeye salmon: "[T]here is no ongoing monitoring of the diseases identified [in Technical Report 1, Infectious Diseases] for any species of wild or hatchery Pacific salmon in BC."[577] "[E]ven if a particular disease is an issue on salmon farms," he added, "there is no way of knowing whether the same disease is causing problems for Fraser River sockeye salmon and if so whether the source of the infection is from other wild or hatchery fish or from salmon farms."[578] Dr. Noakes also traced disease outbreaks on salmon farms for four diseases (IHN, vibrio, furunculosis, and BKD, all identified as high risk by Dr. Kent) back to their farm locations. He found that there have been no IHN outbreaks since 2003, and incidents of the other diseases were predominantly on the west coast of Vancouver Island and in Jervis Inlet and Sechelt, not along the Fraser River sockeye outmigration route.[579]* In testimony, he concluded that diseases from salmon farms are not having a major impact, nor are they likely to have a major impact on the survival of Fraser River sockeye.[580]

Both Dr. Dill and Dr. Noakes testified in August 2011, before the testing for ISAv in wild stocks in the fall of 2011.

Relationships between salmon farms and sockeye productivity / decline

Although there is no evidence proving causation between any stressor related to salmon farming and the decline of Fraser River sockeye, Dr. Noakes and Dr. Dill gave me their views on whether any relationship exists between salmon farming and sockeye decline. This evidence arose in the context of Dr. Connors's work in Technical Report 5B, Salmon Farms and Sockeye Relationships, which I discuss here.

In his report, Dr. Connors used modified Ricker and Larkin models to predict sockeye productivity or survival anomalies for a number of different sockeye populations along the coast from Washington to Alaska.[581] (For an explanation of these productivity models, see Pre-season escapement target planning in Volume 1, Chapter 5, Sockeye fishery management.) In the first part of his analysis, Dr. Connors looked for relationships between sockeye survival anomalies and (a) sea lice abundance on farms, (b) disease frequency on farms, (c) mortalities of farmed salmon, and (d) the number of farmed salmon. Then, in the second part of his analysis, he used a "multi-model inference approach" to quantitatively compare the strength of the relationship between sockeye productivity and salmon farm production, sea surface temperature (SST) (as a

* I note that in May and June 2012, the media reported new outbreaks of IHN on BC salmon farms.

proxy for environmental factors), and pink salmon abundance in the North Pacific Ocean (which other researchers had identified as affecting sockeye productivity).[582]

In the first part of his analysis, Dr. Connors found no statistically significant relationships between sockeye survival and any of the fish farm factors tested.[583] However, he noted that the short time series of aquaculture variables meant that "there is low statistical power to detect a relationship between the aquaculture variables and sockeye survival, should such a relationship actually exist."[584] (See the discussion below.) In the second part of his analysis, Dr. Connors found that "increases in aquaculture production, SST, and pink salmon abundance all increase sockeye salmon mortality" and that the effects of aquaculture production on Fraser River sockeye are likely influenced by the abundance of pink salmon in the open ocean and SST in the winter preceding marine entry.[585] However, he said there was "large uncertainty around these estimated effects," making any conclusions "tenuous."[586]

Dr. Dill relied on Dr. Connors's report to conclude that "farms are having some sort of negative impact on wild salmon productivity, most likely in concert with other factors in the marine environment."[587] Dr. Dill pointed out a study by Ford and Myers (2008), which conducted a "meta-analysis to show that wild salmon stocks have declined, often as much as 50%, wherever aquaculture production has increased."[588] He said that looking at aquaculture worldwide, "wherever there is aquaculture practice there is evidence from population records of declines in wild salmon."[589]

In contrast, Dr. Noakes was highly critical of the second part of Dr. Connors's analysis, stating in his report that there were "several significant problems with [Connors's] assumptions, methods, analyses, and conclusions," and then describing them in detail.[590] Dr. Noakes concluded that "[t]here is no significant correlation between farmed salmon production within the main migration path of Fraser River sockeye salmon, the waters between Vancouver Island and the mainland of British Columbia, and the returns of Fraser River sockeye salmon."[591]

Dr. Connors responded to Dr. Noakes's specific concerns about his analysis with a written reply, to which Dr. Noakes then wrote a further reply.[592] Some participant counsel spent considerable time in their cross-examinations of these witnesses to explore the differences of opinion between them. I do not think it necessary or helpful to understanding the evidence as a whole to repeat that debate here. I agree with Dr. Korman, another statistics expert testifying alongside Dr. Connors and Dr. Noakes, who gave his view that little turned on the disagreement between Dr. Noakes and Dr. Connors:

> Well, for one thing I think that when you look at the bottom-line conclusions of Dr. Connors's report, he's not making claims of very strong effects. So the argument between Noakes and Connors in the end as far as conclusions is Noakes saying, you know, no effects shown, Connors saying weak effects in the case of the longer-term dataset only. So from a decision point of view, you know, that the justice may have enough information right there without getting into all the minutiae about how they come to that argument.
>
> In terms of what Dr. Connors did, I think a lot of his rationale is well justified in terms of using variables that other researchers have suggested in terms of the modelling framework that he did, and in terms of his interpretation. And while there is some speculation or assumptions made in his modelling, that's a totally normal part of the scientific process to basically begin with a set of assumptions, evaluate the data. There are some limitations to that result, which he I think adequately stated in his report as being limited. But that's – it would be irresponsible of us not to do the analysis that Dr. Connors did, in my opinion. So I don't have a problem with it because he was quite cautious in his interpretation.[593]

Further, I note that Mr. Marmorek, lead author of Technical Report 6, Data Synthesis, who was qualified as an expert in experimental design and decision analysis and modelling (among other areas), said he did not think Dr. Noakes's criticisms of Dr. Connors's work were sustainable:

> So all in all, I didn't think the criticisms from Noakes about Connors' work, rather, were sustainable. I think there are certainly weaknesses in the historical dataset, and it would be much better if there had been per farm production data and actual disease data going all the way

> back to the 1980s, but it didn't exist ... I didn't see anything wrong. I thought [Connors] was quite careful in the way he went through his work.[594]

Assessing the risks of salmon farms to Fraser River sockeye

Dr. Noakes and Dr. Dill took different views of the risk posed by salmon farms to Fraser River sockeye based largely on their views of the adequacy of the data available for assessment. Overall, Dr. Noakes concluded that salmon farms "pose no significant threat" to Fraser River sockeye:

> While some improvements are certainly possible and desirable, the industry generally leads the world in with [*sic*] respect to the management and control of disease and waste at their farm sites both through proactive policies and practices. Overall, the evidence suggests that salmon farms pose no significant threat to Fraser River sockeye salmon and that salmon farming has not contributed to the recent decline in Fraser River sockeye salmon productivity.[595]

In testimony he said that, based on the evidence, "there's a fairly low risk" of a negative impact to Fraser River sockeye from salmon farms.[596] Dr. Dill explicitly disagreed with Dr. Noakes's statement that salmon farming poses no significant threat.[597] He was of the view that the state of information did not allow one to say there was no effect of fish farms in the long term, nor to dismiss the risk:

> Open net pen aquaculture, as currently practiced in British Columbia, has the potential to create problems for wild salmon populations because the pens are open to the environment, allowing wastes, chemicals and pathogens to move freely back and forth. Indeed, wild salmon populations have tended to decline wherever this form of aquaculture is practiced, although the reason for this is not always apparent.
>
> ...
>
> It must be understood that the short time series of data available for this investigation precluded identifying salmon farms as an important driver of the decline of Fraser sockeye. But it must be equally understood that at this stage of our knowledge it is not possible to say they are *not* implicated. [Emphasis in original.][598]

I heard much evidence on the state of knowledge and the adequacy of the information available.

Dr. Korman, the researcher responsible for synthesizing the fish health data for use by the other researchers, testified that the fish health records from fish farms were available only from 2003 or 2004 to 2010.[599] Considering that five years need to elapse from the time sockeye spawn until the oldest fish from that brood year return, this leaves only about four years of data that can be used "to correlate conditions on the farms with survival rates of Fraser River sockeye."[600] According to Dr. Korman, the short data record affects the ability to reach conclusions from that data:

> And in statistics, that's a very, very low sample, which means two things. For one, if there [is] a true relationship, it's going to [be] very difficult to see it with that small sample size because your statistical power will be very low.
>
> Conversely, it's also possible to, not from a statistical sense, but just by random chance, to see a relationship between those variables just because you've got such a very low sample, that random chance can actually make it such that you'll see a positive correlation when, in fact, none exists. So therefore, there's going to be limited ability to learn something from statistics given our current data availability. Ten years from now, very different story when we'll have 13, 14, 15 years of data.[601]

Dr. Dill suggested that "perhaps eight to 10 years [of data] might be sufficient" to show no correlation between fish farms and sockeye returns, certainly not four or five.[602] However, Dr. Noakes said that, while one of the problems with short-term data is that you often have values around the mean, in this situation we have "a bit more power in terms of an ability to look at the relationship" because we have extreme data in 2009 and 2010.[603] Both Dr. Noakes and Dr. Dill agreed that more research on wild sockeye would be needed to prove any sort of cause-and-effect relationships of fish farms on Fraser River sockeye.[604]

Mr. Marmorek similarly testified that there is a lack of empirical evidence about wild salmon catching disease and that this is a gap that needs to be filled. In the absence of research, scientists are left with plausible hypotheses and mechanisms whereby salmon farms might cause disease in wild fish.[605] He said it would be possible to design studies that would show these mechanisms if they exist, and that the "simple answer, is go out and get the data."[606] One example may be work proposed in a public submission to this Commission, dated December 15, 2011, by Dr. Welch, who testified before the Inquiry in the fall of 2010 and again in July 2011. In his public submission, Dr. Welch reported that since he last testified, he and colleagues reanalyzed data to directly compare survival rates of acoustically tagged sockeye smolts migrating in the Strait of Georgia and then in Discovery Passage / Queen Charlotte Strait. They found that smolt survival rates per week of migration were substantially lower in the Discovery Passage region than in the Strait of Georgia. They therefore proposed a study designed to test the effect of fish farm exposure in the Discovery Islands on the survival of Fraser River sockeye smolts.[607]

Dr. Dill identified the following knowledge gaps in his report:

- detailed information on sockeye smolt migration behaviour and pathways through the Discovery Islands;
- the attraction of sockeye juveniles to net pens;
- the cumulative impacts of swimming past multiple farms (including repeated exposure to poor water quality and pathogens);
- whether the virus identified by Dr. Miller (described above) is found in farmed Atlantic and chinook salmon;
- the infective state of apparently healthy salmon in net pens;
- the potential for lice to act as vectors of high-risk pathogens;
- the impact of both species of sea lice and of other pathogens on feeding and predator avoidance abilities and survival of sockeye smolts;
- the potential for blood water from processing plants to be a source of infection;
- the evolution of resistance and/or increased virulence in sea lice treated with SLICE;
- interactions of lice and other pathogens with other stressors in the marine environment, such as low food availability and pollutants;
- disease incidence and levels in wild sockeye; and
- the potential for biological control of pathogens on farms (e.g., by using mussels to remove *Renibacterium salmoninarum*, the infective agent for BKD, from seawater).[608]

In his report, Dr. Noakes took the view that there is adequate information about fish farms, recommending that "the scope and level of fish health and sea lice monitoring and reporting currently in place for the salmon aquaculture industry" be maintained.[609] He said the focus of future work should be on wild and hatchery fish, recommending long-term disease-monitoring programs for wild fish, and mandatory fish health–monitoring and reporting programs for all federal, provincial, and Community Economic Development Program hatcheries.[610] Dr. Noakes also recommended regular and routine monitoring and reporting of water quality and oceanographic data, and research into the lethal and sublethal effects of sea lice on juvenile sockeye salmon.[611]

Dr. Connors noted that none of the researchers looked specifically at the nine fish farms in the "Wild Salmon Narrows" (one passage through the Discovery Islands, so named by an environmental group: see Volume 1, Chapter 8, Salmon farm management) to see if any relationships exist with wild sockeye productivity.[612] He agreed with both Dr. Dill and Dr. Noakes that more work into monitoring disease in wild fish was needed.[613] He also recommended, along with Dr. Dill, that his analysis be repeated in the future as more productivity data become available.[614]

I heard testimony from other experts on the risks posed by salmon farms – fish health and disease experts such as the author of Technical Report 1, Infectious Diseases, Dr. Kent (see above), and government and salmon farm veterinarians whose evidence I described in Volume 1 of this Report. I also heard evidence from a variety of witnesses with insights and perspectives on the risks posed by salmon farms.

Dr. Kent said, with the exception of sea lice, he has not seen "dramatic evidence" that pathogens are being transmitted from fish farms to wild fish.[615] He did say that there is a potential for salmon farms to change the environment that wild fish swim through, and that two ways in which fish farms can affect wild fish are by introducing new diseases or by making endemic diseases worse.[616]

He also said it is a “reasonable assumption” to say that the numbers of pathogens in and around net pens would be greater during a disease outbreak on a farm; what scientists do not know for sure is the effect that this has on wild salmon.[617] He said there would be an increased potential for wild fish to catch pathogens when swimming past a net pen than swimming through open water.[618]

Dr. Stewart Johnson, head of Aquatic Animal Health, Salmon and Freshwater Ecosystems, DFO, testified that moving salmon farms off the migratory route would be one thing that people could do to prevent wild stocks from being exposed to disease, noting that “other sources of pathogens are pretty much out of our control.”[619]

Dr. Marty; Dr. Mark Sheppard, lead veterinarian, Aquaculture Environmental Operations, DFO; Dr. Peter McKenzie, veterinarian and fish health manager for Mainstream Canada; and Trevor Swerdfager, former national director general, Aquaculture Management Directorate, DFO, all agreed that “the risk of disease in salmon farms is manageable with appropriate care and attention.”[620] Dr. Sheppard said that “the risk can never be zero,” but that managers minimize as best they can the risks to wild fish.[621] He further said that measures taken within the net cages and through the Fish Health Management Plans (described in Volume 1, Chapter 9, Fish health management) serve to minimize health risks not only to farmed fish, but also to “the ecosystem outside those cages.”[622] Mr. Swerdfager said the regulatory framework in place does not reduce the risk to zero, but “it substantially reduces it.”[623]

Mia Parker, an industry representative formerly from Grieg Seafood BC, testified that she believed fish farms could coexist with wild stocks because of the precautionary and adaptive management framework in place.[624] Another industry representative, Clare Backman from Marine Harvest Canada, said simply that “aquaculture is coexisting with the wild fish without demonstrated significant risk of disease.”[625] In contrast, Catherine Stewart, salmon farming campaign manager for the Living Oceans Society and a representative of the Coastal Alliance for Aquaculture Reform, testified that she believed they cannot coexist unless “a serious limit” is placed on salmon farm production.[626] Alexandra Morton, executive director of Raincoast Research Society, said she believed the salmon farming industry “cannot survive biologically” and “there’s no place that open net pens can coexist with wild fish.”[627]

Marine anemia / plasmacytoid leukemia on chinook farms

In Technical Report 1, Infectious Diseases, Dr. Kent described plasmacytoid leukemia, which he testified is also called “marine anemia” though scientists try not to use that term to avoid confusion with ISA, which is also sometimes called marine anemia in other parts of the world.[628] (I use both terms below to reflect how the witnesses spoke of this condition.)

> **Salmon Leukemia Virus and Plasmacytoid Leukemia (PL).** The histological presentation of this disease is massive infiltration of visceral organs and retrobulbar tissue of the eye by immature lymphocytes or plasmablasts (white blood cells) (Kent et al. 1990). Fish have an enlarged spleen and kidney. The disease causes severe anemia and is usually lethal. It has been most often seen in pen-reared Chinook salmon in British Columbia (Stephen et al. 1996), but has been detected in wild-caught Chinook salmon in the Province and hatchery-reared Chinook salmon in Washington State (Harshbarger 1984; Morrison et al. 1990). Sockeye salmon can be experimentally infected (Newbound and Kent 1991). The cause of PL has been controversial. In the early [1990s] various lines of evidence pointed to a retrovirus as the cause (Kent and Daw 1993; Eaton et al. 1994a, b), which was named the Salmon Leukemia Virus (SLV). The virus was never isolated in culture. In later years, almost all cases that I reviewed from netpen-reared Chinook salmon were associated with infections of the proliferating cells by *Nucleospora salmonis* [a micro-organism that infects lymphocytes].[629]

Dr. Kent and Dr. Stephen, author of Technical Report 1A, Enhancement Facility Diseases, are two of the world’s leading experts on plasmacytoid leukemia / marine anemia.[630] They both described it as a “pathology” or “condition” that may have various potential causal pathways or causal agents.[631] Dr. Kent said there were outbreaks of marine anemia at chinook farms in British Columbia in

1988–91, causing "severe losses" at those farms.[632] He also said that, in a general disease survey of multiple species conducted in 1998 by DFO Science (where he was then working), none of the sockeye surveyed showed signs consistent with plasmacytoid leukemia.[633] Dr. Stephen said that, in the period of 1988–92, marine anemia was detected in chinook farms whenever the industry moved into a new area of the BC coast.[634] Both Dr. Kent and Dr. Stephen stopped working on plasmacytoid leukemia in the 1990s, when they moved on to other research.[635]

Dr. Sheppard – formerly the provincial, now the federal, veterinarian responsible for making farm-level diagnoses, and qualified at the hearings as an expert in veterinary medicine, with experience in fish health – explicitly disagreed with Dr. Kent's work from the early 1990s that showed severe losses of farmed chinook salmon due to marine anemia, and he disagreed with the statement that there was a major epidemic of plasmacytoid leukemia in the early 1990s. In his view, marine anemia was "a finding of a clinical syndrome" that "became a point of interest for some researchers."[636] He said those "very same animals" diagnosed with marine anemia were often also infected with BKD, so marine anemia was not the sole cause of mortality.[637] Further, he said that since the early 1990s "we see next to no signs of plasmacytoid leukemia in chinook or coho salmon."[638]

One theory related to the impact of salmon farms on Fraser River sockeye put into evidence by Ms. Morton concerned marine anemia. Her theory was that, in 2007, Fraser River sockeye smolts picked up marine anemia from chinook salmon farms during the smolts' outmigration through the Discovery Islands. This, she hypothesized, contributed to the poor return in 2009. The chinook were harvested from the farms before the 2008 outmigration, so the smolts responsible for the large 2010 run were not affected by the same disease exposure.[639] Ms. Morton based her concern on what she considered to be symptoms of marine anemia appearing in the provincial fish health databases, particularly in relation to chinook salmon farms.[640]

Ms. Morton testified that she looked at the data from the provincial fish health databases showing *symptoms* of different diseases (rather than *diagnoses*) in government audit fish, counted the number of individual fish showing symptoms in different years, and plotted those numbers on a graph.[641] Her counsel showed that graph to Dr. Korman, who agreed that, on the graph, marine anemia symptoms look "a little higher in 2007" than in other years.[642] Ms. Morton said that, despite these symptoms of marine anemia, the disease never gets diagnosed at the farm level because Dr. Sheppard "doesn't believe in marine anaemia."[643] Further, questions to Dr. Korman from Ms. Morton's counsel suggested that, beginning in the fourth quarter of 2006, the Conville Bay fish farm site (a chinook farm owned by Marine Harvest Canada in the Discovery Islands) had elevated symptoms of marine anemia.[644] Dr. Korman agreed that "[i]f there were chinook farms experiencing marine anemia in the Discovery Islands in the Wild Salmon Narrows in 2007 but none at all in 2008," that would be a matter worth investigating.[645] Ms. Morton acknowledged that this was not a matter for her to investigate – just a pattern she saw in documents that she felt should be looked at by someone, presumably with disease expertise.[646]

In a document she wrote and put into evidence during the hearings, Ms. Morton explains that the symptoms of marine anemia that she relied on are those described in the fish health databases disclosed by the province as ISH, or interstitial hyperplasia of the kidney.[647] However, Dr. Kent testified that, while interstitial hyperplasia of the kidney is not inconsistent with marine anemia (and is associated with marine anemia), it is not pathognomonic for (or specific only to) marine anemia – ISH may be caused by a number of pathogens.[648] Indeed, Dr. Marty,* who prepared the fish health databases used by Ms. Morton, said that marine anemia is a clinical syndrome, so it is not something that he would diagnose in the lab.[649] He testified further, in reference to part of the database used by Ms. Morton to extract ISH symptoms, that each of the fish showing symptoms of ISH also has a cause of death associated with it – either BKD or *Piscirickettsia salmonis*.[650] He said it would be inappropriate to diagnose these fish as having marine anemia because "we have another cause of death instead of marine anaemia."[651] However, Ms. Morton pointed out in her testimony that Dr. Marty showed me the diagnoses of BKD associated with ISH symptoms for *Atlantic* salmon farms, not *chinook* farms.[652] She said that when you

* Dr. Marty was qualified as an expert in fish toxicology and fish pathology, with a specialty in veterinary pathology (Transcript, August 31, 2011, p. 13).

look at the Pacific chinook salmon with ISH symptoms, the symptoms are more severe (in terms of a rating scale assigned by Dr. Marty), and many of the diagnoses are listed as "open" diagnoses, not BKD.[653] Therefore, in her interpretation, marine anemia was not ruled out by another cause of death.

In response to Ms. Morton's theory, Dr. McKenzie testified that he spoke to the veterinarian for the Conville Bay fish farm, who told him that marine anemia had never been diagnosed for that site.[654] Also, Dr. McKenzie said he reviewed documents for that site, which showed the harvest began in December 2006, was 75 percent complete by March 2007, and was completed in early May 2007 – before most of the 2007 smolts would have passed by the farm.[655] Mr. Backman testified that marine anemia has never been diagnosed on-site at Conville Bay.[656] However, he also said that the fish health vets working at that site may not have been aware of the information that was recorded in the fish health audit database:

> [H]istopathological reporting is only one element of discovering whether or not there is an issue on a farm site, and so that it's very common and, actually, in most cases, the veterinarian is not made aware of records made through the provincial audit, and the reason for that is that within the provincial team, and that consists of the provincial veterinarians, fish health experts, the decision is made that they're not seeing something of a reportable nature. Should they see something where there's a histopathological result that's confirmed with farm-based evidence, then they will quickly advise the farm site.[657]

Further, Mr. Backman said the pattern of mortality for Conville Bay in 2006 and 2007 was "not inconsistent with what we normally see year over year. There was nothing particularly unusual."[658] He said that, although chinook salmon have not been placed back into the Conville Bay site, two other chinook farms continue to operate in the Discovery Islands area.[659]

Migration to rearing area along the coast

On leaving the Strait of Georgia, juvenile sockeye (also called "postsmolts") continue their migration through Johnstone and Queen Charlotte straits (or Juan de Fuca Strait and the west coast of Vancouver Island) toward the North Pacific Ocean, where they enter south of Haida Gwaii (Queen Charlotte Islands). There is some evidence that the postsmolts, on entering the North Pacific Ocean, migrate north and westward in a band within 35 km off the coasts of British Columbia and Central Alaska until they reach the overwintering grounds south of Alaska during late autumn. Figure 2.4.2 depicts the postsmolt migration along the continental shelf.

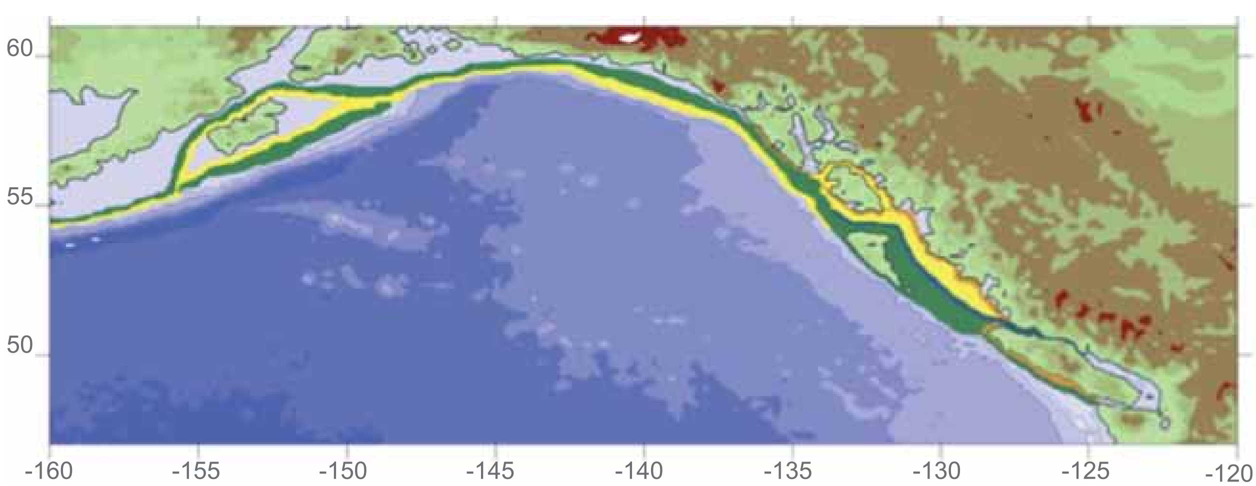

Figure 2.4.2 Seasonal migration of Fraser River sockeye salmon postsmolts after leaving the Strait of Georgia

Blue, May–June; green, July–August; yellow, October–November; orange, February–March.
Source: Technical Report 4, Marine Ecology, p. 17 (Exhibit 1291).

Several of the Commission's technical reports examined, and witnesses testified about, the stressors sockeye salmon encounter on the migration to the North Pacific Ocean, which may have caused or contributed to the recent decline. I summarize these discussions below.

Predation

The Humboldt squid lives in the eastern Pacific and, in recent years, has had a biomass of approximately 9 million tonnes. They were first observed in the waters of British Columbia in 2004, when the surface temperatures of the North Pacific were the highest on record. They move north from California in early summer and appear in British Columbia in late summer and early fall, before they return south. Dr. Christensen and Dr. Trites concluded that Humboldt squid eat prey the size of sockeye smolts, although there is no direct evidence that they prey on sockeye. It is not clear to what degree there was spatial and temporal overlap of Humboldt squid and Fraser River sockeye smolts leaving the Strait of Georgia in 2007. However, the researchers concluded that, if smolts had to pass through an accumulation of Humboldt squid, it is entirely possible they could have a strong predation impact on the sockeye.[660]

During the evidentiary hearings, Graham Gillespie, head of the Shellfish Section, Pacific Biological Station, Science Branch, testified that abundance of Humboldt squid increased between 2004 and 2009, although there are no quantitative estimates of abundance.[661] The squid feed mainly on small pelagic species, myctophids, small schooling rockfish, hake, and pelagic invertebrates, including other species of squid.[662] Mr. Gillespie testified that he did not think that Humboldt squid were responsible for eating a large number of Fraser River sockeye smolts in 2007, although he could not say definitively that they were not implicated.[663] In 2007, Humboldt squid were found off the west coast of Vancouver Island but not farther north, and there is no evidence that they were in the migratory pathways of outbound sockeye smolts.[664]

Climate change

The authors of Technical Report 9, Climate Change, Dr. Hinch and Dr. Martins, concluded that the recent warming of the waters along the British Columbia coast, as well as the negative relationship between early marine survival and coastal sea surface temperature, suggests that survival of juvenile Fraser River sockeye during their coastal migration has likely decreased in the past two decades.[665]

Marine ecology

In Technical Report 4, Marine Ecology, McKinnell and others considered whether marine conditions during the coastal migration facing postmolts in 2007 and 2008 could explain both the poor Fraser River sockeye returns in 2009 (owing to poor food availability) and the historically strong returns in 2010.

The researchers reported that, during the summer of 2007, the surface layer of eastern Queen Charlotte Sound had the lowest average salinity on record since 1970. It was caused by extremely high river discharge volumes from snowmelt combined with high precipitation in early June. Extreme wind anomalies can be added to the list of oceanic extremes that occurred in Queen Charlotte Sound that summer – the highest average southeasterly flow in the 63-year time series. These winds caused ocean surface waters to be pushed into Queen Charlotte Sound, raising coastal sea levels and generally enhancing the poleward flow of surface water along the BC coast. This wind regime caused the warm, less salty surface layer to be retained in the sound. It may also explain why the coast of southeastern Alaska was warmer than average during the summer of 2007.[666]

July 2007 began with neutral to positive sea surface temperature anomalies along the entire coast, strengthening in August and persisting at slightly lower levels through September. The summer temperatures in 2007 were the largest outlier from what May sea surface temperatures might have predicted. Queen Charlotte Sound was the only area along the coast that had extreme absolute sea surface temperature. Warmer-than-average coastal temperatures are normally a sign of lower-than-average survival of Fraser River sockeye.[667]

In years when the spring chlorophyll bloom, which is used as a proxy for phytoplankton presence, is delayed, marine survival of some sockeye populations (e.g., Chilko Lake) is low. The bloom in 2007 was the latest on record since 1998.[668] Although there are likely no Fraser River sockeye postsmolts in Queen Charlotte Sound during April, the lag until their arrival in June and July can allow time for their prey base, which depends on phytoplankton, to develop.[669]

The summer of 2008 was the opposite. Sea surface temperatures along the North American coast were cool following what was the coldest year in the Gulf of Alaska since 1972, and these cool anomalies persisted along the coast through September. Migrating sockeye in 2008, once leaving the coastal straits, would have had a very different thermal experience during their outmigration compared with 2007. This evidence is consistent with the expert report by Thomson and others.[670]

During the evidentiary hearings, Dr. McKinnell testified that there is a correlation between these unusual sea surface temperatures and wind patterns on the one hand, and Fraser River sockeye survival on the other, though that does not establish causation.[671] Further work would be required to understand causation.[672] He said that, in attempting to explain the poor 2009 return, it is also necessary to explain other observations from that time period, including double-the-average returns of sockeye to the Columbia River, better-than-expected returns of sockeye to Barclay Sound, and record high returns to the Harrison River.[673] One needs to develop a model that somehow satisfies all these concurrent observations – and placing the mortality of the 2007 age-one smolts in Queen Charlotte Strait / Sound has the possibility of doing so.[674]

Dr. McKinnell also testified that there is doubt whether a 2009–10 El Niño / La Niña event influenced the 2010 return, as postulated by Exhibit 1303 (Thomson and others, 2011) and testimony of Dr. Beamish.[675] This is because the high return for 2010 was evident in the 2009 test fisheries where the jacks (three-year-olds) were seen in unusually high abundance.[676] He explained that "[w]e interpret this to be an indication that the high abundance of the return in 2010 was established at least a year earlier than the time when the 2010 return occurred."[677]

Life stage 4: growth to adulthood

Growth in the North Pacific

The distribution and movement of immature Fraser River sockeye salmon at sea is the least understood of all life history phases. Stock-specific movements of Fraser River sockeye in the open ocean are unclear, but there is some evidence that different sockeye stocks are in different places in the offshore. Dr. Welch testified that sockeye from the Nass, Skeena, Fraser, and Columbia rivers and Rivers Inlet are spatially separated during the month of June.[678] Dr. Timothy Parsons, professor emeritus at the University of British Columbia and honorary research scientist with DFO, testified that radio isotope testing has shown that different stocks of salmon go to very specific locations in the Gulf of Alaska, and that populations of Atlantic salmon are distributed in different geographic locations in the North Atlantic.[679]

Dr. McKinnell testified that the period between when Fraser River sockeye are migrating northward along the continental shelf and when they appear in deep water is one of the least-understood periods for these animals, in part because logistically difficult winter sampling is involved.[680]

Several of the Commission's technical reports examined, and witnesses testified about, the stressors during the sockeye's residence in the North Pacific Ocean that may have caused or contributed to the recent decline. I summarize these discussions below.

Predation

In Technical Report 8, Predation, authors Dr. Christensen and Dr. Trites stated that salmon sharks migrate between Hawaii and Alaska. They are reported to feed primarily on Pacific salmon in spring and summer. However, Mr. McFarlane testified that salmon sharks are opportunistic and episodic feeders, and a lack of diet information makes it difficult to link salmon shark predation and changes in sockeye population.[681] Although abundance trends are very limited, there is an indication that abundance has increased in recent decades, an occurrence that means the predation impact on Fraser River sockeye salmon may have increased as well. The researchers concluded that the only way to reliably evaluate if salmon sharks have had an increasing impact would be to gather more information about their open-ocean abundance and abundance trends.[682] During the evidentiary hearings, Dr. Christensen testified that the salmon shark was at the top of their list of Fraser River sockeye predators.[683]

Blue sharks are much more abundant than salmon sharks, but since their population has not increased in recent decades, it is unlikely that abundance trends can explain the Fraser River sockeye decline, even if it may have contributed to it. Dr. Christensen and Dr. Trites also discounted the role of the Pacific sleeper shark in the decline.[684]

Sablefish are opportunistic feeders known to consume sockeye salmon, but sablefish in British Columbia and in the Gulf of Alaska have been in decline since the late 1980s. For that reason, the researchers concluded that it is not likely that sablefish would be a major factor in the decline of Fraser River sockeye salmon.[685]

The researchers also considered daggertooth, walleye pollock, and arrowtooth flounder, all of which could exert predation pressure on sockeye salmon. However, owing to inadequate information about abundance and abundance trends, they could not conclude that any of them has been a factor in the decline of Fraser River sockeye salmon.[686]

Climate change

In Technical Report 9, Climate Change, authors Dr. Hinch and Dr. Martins stated that the abundance of sockeye salmon has closely tracked decadal-scale fluctuations in sea surface temperature over most of the past 300 years. These fluctuations have been well documented during the past century and linked to major climate-driven changes in the marine environment occurring every 20–30 years. The abundance of Fraser River sockeye began to increase dramatically at the end of the 1970s, reaching historic high abundance in the early 1990s. Subsequently, abundance and productivity began to decline to recent low levels, in coincidence with the exacerbation of the long-term warming trend of the global climate.[687]

In the North Pacific marine environment, long-term climate change trends are difficult to detect because conditions are strongly related to both inter-annual and inter-decadal modes of climate variability:

- Inter-annual variability is related to El Niño Southern Oscillation events, which occur every two to seven years and persist for up to 1.5 years. Typically, El Niño events lead to warm sea surface temperature in the waters of the west coast of North America, and since the 1970s, El Niño events have become more frequent. By contrast, La Niña events result in cooler waters, and such events have become less frequent.[688]
- Inter-decadal variability in the climate of the North Pacific Ocean has been described by indices such as the Pacific Decadal Oscillation (PDO), which typically persists for 20 to 30 years. Warm sea surface temperatures over the eastern North Pacific Ocean characterize the warm or positive phase of the PDO, whereas opposite sea surface temperature patterns characterize the cool or negative phase. The PDO was predominantly in the positive phase between 1977 and 1997 and, since 1998, has exhibited more frequent alternations, lasting three to four years. Sea surface temperature in the Gulf of Alaska has increased by about 0.25°C per decade since the 1950s. It is now 1.5°C warmer than 60 years ago and 0.5°C warmer than 20 years ago. However, such observed warming has been attributed mostly to the positive phase of the PDO. In contrast to warming trends, both salinity and pH of the North Pacific Ocean has been decreasing in recent decades.[689]

Only a few studies have explored the relationship between temperature and survival of immature sockeye salmon in the open ocean. A 2009 study found that survival of Alaskan sockeye was positively correlated to sea surface temperature during all the years of ocean residence, while a 1991 study found that the survival of Fraser River sockeye was negatively correlated to sea surface temperature in their last few months in the open ocean.[690] The authors of Technical

Report 10, Production Dynamics, reported that increased sea surface temperature in the location of early ocean residence for smolts of a given stock is associated with increased productivity in Alaska.[691] The researchers concluded that increased temperatures in the Gulf of Alaska over the past two decades have possibly resulted in lower survival of Fraser River sockeye during open-ocean residence.[692]

During the evidentiary hearings, Dr. McKinnell testified that Intergovernmental Panel on Climate Change projections for future climate are difficult to represent in terms of the finer-scale climate, such as climate changes that will occur in British Columbia and what the response of the marine ecosystem will be in the northeastern Pacific Ocean.[693]

Marine ecology

Technical Report 4: Marine Ecology

The authors of Technical Report 4, Marine Ecology, considered the hypothesis that a volcanic eruption in the Aleutian Islands in August 2008 led to the widespread deposit of volcanic ash (including iron) in the northwestern Pacific. The theory is that this deposit enhanced productivity of chlorophyll in mid- to late August, benefiting the 2008 smolt year and explaining the strong 2010 Fraser River sockeye return. The authors doubted that this enhanced productivity of chlorophyll benefited the 2008 smolts because the fish would have been migrating along the continental shelf, where iron is not normally the limiting nutrient – so its addition would not have produced much benefit to this cohort. To have directly benefited this cohort, the enhanced biological production would need to be entrained in the pelagic food web and stored through, or made available to it over, the winter. However, zooplankton biomass in 2009 was near the average.[694]

Dr. Parsons testified about the possible impact of the 2008 volcanic eruption on Kasatochi Island, Alaska, on Fraser River sockeye.[695] There is no doubt that an enormous diatom bloom was generated in the Gulf of Alaska and that zooplankton increased by a factor of three, which he said benefited sockeye in the Gulf of Alaska.[696] A similar event occurred in 1956, when a volcano erupted in Kamchatka in eastern Russia, resulting in a return of 20 million fish in 1958. He explained why this affected only the younger 2008 fish, not the 2009 fish:

> Our take on that is that what you have taking place is a massive bloom of diatoms which are absorbed very quickly by the zooplankton. The zooplankton will be rather small zooplankton and they will be consumed much more easily by the young adolescent salmon than the larger 2009 salmon which are still waiting for something big to come along. They've already gone through the stage where they were eating small prey. They're a year older.[697]

Dr. Parsons acknowledged that these are really quite hypothetical answers to a question which is somewhat speculative, but worth recording as a possible mechanism for the 29 million Fraser River sockeye that returned in 2010.[698] Dr. Irvine raised several questions about Dr. Parsons's hypothesis. It was not clear to him why the older salmon (2009 return year) would not have consumed this additional prey, as nutrient sources were extremely important to them because they would be accumulating a lot of mass before beginning their return migration.[699] Also, it was to be expected that the 2010 return would be strong, since the 2008 smolts were migrating northward during a very strong La Niña, with cold waters.[700] Dr. Parsons responded that the impact of the volcanic ash may have been different for different sockeye stocks, since radio isotope testing has shown that different stocks of salmon go to very specific locations in the Gulf of Alaska.[701] Although satellite imagery seems to indicate that the chlorophyll (representing presence of phytoplankton) was distributed throughout the Gulf of Alaska, that does not mean that it was necessarily evenly distributed.[702]

McKinnell and others concluded that there was a strong, abrupt, and generally persistent shift to warm sea surface temperature anomalies along the continental shelf in late June of 2007. However, since most southern stocks were likely using this migratory route during 2007, and non-Fraser stocks did not suffer incremental mortality, it seems unlikely that this coast-wide phenomenon was the cause of incremental mortality of the Fraser River stocks.[703]

The report noted that lack of observations of salmon at sea, at relevant time and space scales, severely limits the ability to draw firm conclusions about their fate.[704] It concluded that, for most Fraser River sockeye populations (where smolt abundance is not observed), the relative contributions of freshwater and marine effects on survival can only be assumed. Fry-to-adult survival trends tend to favour a marine origin for the decline, because they share a common ocean but not a common lake. These populations also share a common means of egress to the sea, although this life history stage is rarely examined in detail.[705]

According to the researchers, the sea provides only limited amounts of food for growing sockeye salmon. Fraser River sockeye were smaller when the total abundance of sockeye in the Gulf of Alaska was greater, but this is not a universal truth.[706] During the habitat enhancement and restoration hearings, Dr. Peterman testified that the body size of a given age of adult sockeye salmon decreases as abundance of competitors increases.[707]

McKinnell and others said that there is some evidence that the mean fork length* of Fraser River sockeye was significantly smaller in brood years that matured in odd years. Because the odd / even cycle of abundance of pink salmon in the Fraser is potentially a source of competition for Fraser River sockeye returning the same year, it is reasonable to postulate that a reduction in mean size in odd years is a consequence of competition for food with pink salmon during the period of overlap in the Gulf of Alaska.[708]

Interaction between wild and enhanced salmon

Dr. Randall Peterman, professor at the School of Resource and Environmental Management, Simon Fraser University, and Canada Research Chair in Fisheries Risk Assessment and Management, testified about potential interactions between wild and enhanced fish.† In his view, increasing fish densities in the North Pacific may have negative impacts on wild stocks, including Fraser River sockeye.[709] None of Dr. Peterman's evidence was contested by DFO.

I also heard in hearings on the marine environment that the interactions between hatchery and wild salmon is a substantial issue in fishery science, and that there is extensive literature on the potential interactions for pink, chum, chinook, and coho.[710] Dr. Beamish stated that there is evidence of hatchery–wild interactions among various salmon species, although whether there could be a long-term substantial reduction in production is less clear among the scientific community.[711]

Dr. Peterman explained that in the North Pacific Ocean there is considerable potential for indirect interactions between wild and enhanced salmon.[712] Across pink, chum, and sockeye salmon, 22 percent of adults in the North Pacific Ocean are of hatchery origin, and plans exist to further increase annual hatchery releases, particularly in Russia and Alaska.[713] Although relatively few sockeye are produced by hatcheries, wild sockeye appear to interact with pink salmon.[714] Dr. Beamish testified that there is the potential, particularly for chum salmon that are enhanced in Asia and pink salmon enhanced in Alaska, to have a density-dependent effect on Fraser River sockeye in the Gulf of Alaska as a result of the large numbers of enhanced fish released into the same area.[715]

Dr. Peterman described several mechanisms for interactions between wild and enhanced salmon. Competition for food can occur between wild and enhanced salmon because their diets overlap and they are thought to generally pass through feeding areas at similar times and places.[716] Food supply in the open North Pacific Ocean has diminished as a result of feeding largely by pink salmon.[717] Also, predation-induced mortality on wild juvenile salmon can be increased because of the attraction of predators to high abundances of juvenile salmon driven by large hatchery releases.[718] Dr. Peterman noted, however, that high total abundance of hatchery plus wild juveniles could also increase the survival rate of wild juvenile salmon co-migrating

* Fork length is measured from the tip of the snout to the end of the middle caudal fin rays.

† Dr. Peterman was qualified as an expert in density-dependent effects on wild and enhanced fish populations for the purpose of this hearing topic (May 2, 2011, pp. 10–11). He was also previously qualified as an expert in fisheries biology with expertise in fish population dynamics and ecology and risk assessment during hearings on Exhibit 748 (Technical Report 10), which he co-authored for the Commission (May 2, 2011, pp. 4–5). His curriculum vitae is Exhibit 749.

with hatchery fish because the abundance of prey may satiate predators.[719]

Where adults of wild and enhanced salmon co-migrate through fishing areas, pressure is intense on managers to allow high harvest rates.[720] However, wild stocks generally have lower productivity (adults per spawner) than enhanced fish, so high percentage harvest rates targeted on enhanced fish are known to eventually lead to overharvesting and depletion of abundance of wild co-migrating stocks that are subject to those same harvest rates.[721] Finally, after adults leave the ocean, large numbers of hatchery fish straying into spawning areas for wild fish can decrease biological diversity and fitness of the wild stocks.[722]

Dr. Peterman provided evidence that the body size at a given age of adult sockeye salmon decreases as abundance of competitors increases.[723] He also explained how the survival rate of sockeye salmon can decrease as the abundance of pink salmon competitors increases, although he said that there are only a few documented examples of this reduction in survival rate compared with examples of reduced growth rate (reflected by adult body size).[724] Dr. Peterman noted that the concern about competition among wild and enhanced salmon for limited resources may become considerably more acute if the North Pacific Ocean becomes less productive again (as it was before the mid-1970s).[725] (For a discussion on DFO's management response to interaction between wild and enhanced salmon, see Volume 1, Chapter 6, Habitat management.)[726]

Return to the Fraser River

In their fourth (or in some cases, fifth) year of life, and after spending one-and-a-half years (or in some cases, two-and-a-half years) in the Gulf of Alaska, Fraser River sockeye leave the Gulf of Alaska and return to the Fraser River to spawn. During the evidentiary hearings, Dr. McKinnell testified that the decision when to return appears to be made in the winter preceding the year that the fish will mature; the decision has genetic and growth components.[727] From that time on, the maturing sockeye needs to find enough food to double its body weight in that last spring at sea. He stated:

> So there's a huge energetic demand on the maturing fish that does not exist for the immature fish, because they have to be able to have enough resources to get from the Gulf of Alaska to fresh water, to swim up the river, to mate and produce gametes and everything that goes along with maturation. That's an energy intensive process.[728]

Fraser River sockeye are captured in fisheries between Alaska and Washington State; their availability depends on their migration route. There are two migratory return routes – down the west coast of Vancouver Island and through Juan de Fuca Strait, or through Johnstone Strait and the Strait of Georgia (the northern diversion route).[729] (See also the discussion of pre-season forecasting in Volume 1, Chapter 5, Sockeye fishery management.)

The percentage that follows the northern diversion route varies from year to year. Dr. Welch testified that, when the ocean temperature is at 10°C, the migration is almost entirely through Juan de Fuca Strait, but when the temperature increases to 12–13°C, 80 to 90 percent of returning sockeye come through the northern diversion route.[730]

Several of the Commission's technical reports examined, and witnesses testified on, the stressors that may have caused or contributed to the recent decline during the return of adult sockeye to the Fraser River. I summarize these discussions below.

Predation

In Technical Report 8, Predation, authors Dr. Christensen and Dr. Trites reported that, after harbour seals received protection under the*Fisheries Act* in 1970, their numbers increased from approximately 9,000 to 108,000, with about 40,000 of these in the Strait of Georgia. An analysis of 3,000 fecal samples collected from 58 Strait of Georgia sites during the 1980s indicated that harbour seals primarily ate Pacific hake (42 percent) and herring (32 percent), while salmonids comprised only 4 percent of the overall diet. Harbour seals appear to prefer chum and coho salmon over sockeye or pink salmon.[731] The researchers concluded that the numbers of seals have been relatively stable in British Columbia for the past decade and showed no changes that might indicate a disproportionate level of predation on the Fraser River sockeye salmon run.[732]

Steller sea lions have also increased in abundance since being protected under the *Fisheries Act* in 1970. They stabilized at about 10,000 until the mid-1980s, then grew at about 4 percent per year, now totalling approximately 30,000.[733] Daily consumption ranges from 15–20 kg (females) to 30–35 kg (males). Salmon accounts for about 17 percent of their diet in British Columbia, although sockeye salmon appears to be the least favourite salmonid prey. The researchers concluded that Steller sea lions could exert some impact on returning numbers given their large body sizes and relatively high food requirements.[734]

During the evidentiary hearings, Peter Olesiuk, head of the Pinniped Research Program, Pacific Biological Station, Science Branch, testified that the total Steller sea lion population for British Columbia and southeastern Alaska is estimated to be 60,000.[735] Mr. Olesiuk and Dr. John Ford, program head of the Cetacean Research Program, also at DFO's Pacific Biological Station, did not stray from the opinion they had expressed at the 2010 Pacific Salmon Commission conference that Steller sea lions (and Pacific white-sided dolphins) appear to be the only potentially significant marine mammal predators of sockeye salmon.[736] For other species of marine mammals, sockeye seems to be the least preferred of salmon species.[737] Mr. Olesiuk testified that he would add the Steller sea lion to the list of species that warrant more attention in relation to the decline of Fraser River sockeye.[738]

California sea lion males migrate from Mexico to British Columbia during the non-breeding season and are most prevalent in the Strait of Georgia between January and May. Since 1984, the population has stabilized at about 3,000, and there is no diet information available. Because their residency in the Strait of Georgia does not overlap with returning Fraser River sockeye adults, they are unlikely to have contributed to the decline.[739]

Of the three forms of killer whales in British Columbia (resident, offshore, and transient), only the resident ecotype are known to feed on salmon. Northern resident killer whales (244 individuals) forage selectively on chinook and chum. For southern resident killer whales (85 individuals), a 2010 study of partial remains from kills in Juan de Fuca Strait and the San Juan Islands indicates that 82 percent of the diet was chinook. Smaller-bodied sockeye and pink were not significant prey despite their far greater seasonal abundance.[740]

During the evidentiary hearings, Dr. Ford testified that, in the analysis of 800 samples of salmonid kills conducted over 20 years, only four were sockeye salmon.[741] Three-quarters were chinook, because they are resident in the killer whale range throughout the year, they are much larger than sockeye, and they have the highest fat content of all salmonids.[742] Sockeye appears to be insignificant in killer whale diets.[743]

Approximately 100 Pacific white-sided dolphins took up year-round residency in the Strait of Georgia during the past decade, but little is known about their movement or diets.*

Climate change

The authors of Technical Report 9, Climate Change, Dr. Hinch and Dr. Martins, did not specifically discuss this phase of the Fraser River sockeye salmon life cycle. However, their discussion of changing climatic conditions, especially the trend toward warmer sea surface temperature, summarized earlier, could apply to this life stage as well.

Marine ecology

In Technical Report 4, Marine Ecology, authors McKinnell and others reported that, between 1952 and 1993, there was a significant linear decrease in mean size at maturity of 10 Fraser River stocks. The researchers felt the trend was due to increasing surface temperatures affecting sockeye metabolism on the return migration. However, when a more comprehensive view of the time series is considered (1952–2009), there is significant low-frequency variability in mean size. This variability is not a simple linear trend and, in fact, indicates in recent years that the mean size is increasing.[744]

During the evidentiary hearings Dr. McKinnell testified that, when the sea surface temperatures

* See also Exhibit 788 and Transcript, May 4, 2011, pp. 50–53, to the effect that they could have played some role in the overall cumulative predation impacts on Fraser River sockeye.

in the Gulf of Alaska get warmer, the sockeye get smaller.[745] There is also another effect – when the abundance of fish is high, their mean size tends to be low.[746]

Mortality-related genomic signature

Earlier in this chapter (see Life stage 1), I summarized the research under way within DFO on a mortality-related genomic signature, led by Dr. Miller. Her research is relevant to this life history stage as well. In brief, in 2008, 60 percent of smolts left the river with the mortality-related signature in the brain, and 40 percent with the signature in the liver. Overall, 82 percent of fish were affected in at least one tissue. However, there were 2.4 times as many fish without the signature in the fall as in the summer.[747]

In the case of adults returning to spawn, 50 percent carried the mortality-related signature. In the marine environment, when fish carry this signature, they had a 13.5 times lower probability of making it to the spawning grounds and spawning.[748] Dr. Miller testified that this study showed unequivocally that Fraser River sockeye are entering the river in a compromised state, and the freshwater environment alone may not be the sole source of the highly fluctuating mortalities of salmon in the river.[749]

Dr. Miller told me that she and Dr. Garver are trying to establish whether this signature is a virus, and possibly a novel parvovirus.[750] It had not been determined, when these witnesses testified in August 2011, whether it is infectious or whether it causes disease.[751]

Sea lice

A study published by DFO scientists on Alberni River sockeye in the 1990s showed a high prevalence (100 percent) and intensity (49 to 1,372 lice per fish, with an average of 300 lice per fish) of *Leps* infection on Alberni sockeye in coastal waters before river entry on the return migration.[752] The sockeye suffered anything from minor skin discolorations to large open lesions that exposed the musculature (87 percent of the fish).[753] In 1990, when these fish were delayed in their escapement into the river system, they suffered high mortality thought to be associated with the large lesions caused by the salmon lice, though death might actually be caused by secondary bacterial or fungal infections, or, in severe cases, osmotic stress.[754] The prevalence and severity of the lice infections in this study were "greater than previously reported for sockeye."[755] Dr. Dill described the circumstances as "a very unusual event."[756] The authors of the study described how high river temperatures forced 60 percent of the sockeye to remain in the inlet much longer than normal, exposing them to "an extended period of crowding, high water temperatures and low dissolved oxygen levels."[757] They concluded it was "highly probable" that, as sockeye mature sexually or are exposed to stresses such as adverse environmental conditions, they may become more susceptible to *Leps* infection.[758] The researchers summarized their results as follows:

> In summary, we saw higher prevalence and intensities of *L. Salmonis* on mature sockeye in Alberni Inlet than reported for immature and maturing sockeye collected on the high seas. Sockeye may become infected with *L. salmonis* as they pass through coastal waters or hold in inlets prior to entering fresh water. It is also possible that immunosuppression concomitant with maturation makes sockeye more susceptible to infection with sea lice as they come ashore. Stocks of Alberni sockeye were shown to differ in the severity of lesions caused by sea lice. The reasons for this difference are unknown. In years such as 1990, when unfavourable river conditions force the fish to remain in the inlet longer than normal, serious disease and mortality induced by sea lice can occur. In extreme cases, disease induced by sea lice appears to cause pre-spawning mortality in freshwater environments, thereby further affecting the reproductive success of these stocks. This observation highlights the important role that diseases may play in the population dynamics of fish stocks.[759]

Although the study was not about Fraser River sockeye, I understand this to be one of the few published field studies looking at the effects of sea lice, particularly *Leps*, on sockeye in the wild. It indicates that *Leps* is capable of causing mortality in sockeye if present in large numbers and/or in combination with adverse environmental factors.[760]

Life stage 5: return migration

Entry into the Fraser River and upriver migration

As returning Fraser River sockeye approach the mouth of the Fraser River, either through Juan de Fuca Strait or through Johnstone Strait, there is some variation in how promptly they move into the river and begin their upstream migration.

This variation is based on the four timing groups – Early Stuart, Early Summer, Summer, and Late-run. The Early Stuarts (which return in June and July) and the Early Summers and Summers (which return in July and August) enter the Fraser River with little or no delay – perhaps one day. Thus, sockeye passing through Johnstone Strait will need six or seven days to move through the Strait of Georgia, enter the river, and reach Mission. Fish returning through Juan de Fuca Strait will need five or six days to reach Mission.

However, the Late-run timing group, which has historically returned in August–September, exhibits two distinctive types of behaviour. Since the 1990s, some parts of the run will enter the river with little or no delay, at the same time as the Summer timing group. The others will, as Late-runs have done historically, delay at the mouth of the Fraser for 20 to 30 days, or longer in some years.[761]

Factors that influence river-entry timing for all Fraser River sockeye stocks include fish maturity, tides, river flow, and water temperature.[762] Over time, it has been observed that there has been increasing overlap of the different run-timing groups.[763] Karl English, the author of Technical Report 7, Fisheries Management, described the earlier appearance of the Late-run timing group at Mission from late August during the 1990s to late July in the 2000s.[764] Research undertaken between 2002 and 2006 showed that Late-run sockeye entering the river before the middle of August experienced a very low probability of survival, whereas those beginning their upstream migration in mid- to late September had a much better likelihood of reaching the spawning ground and actually spawning.[765]

During the evidentiary hearings, Dr. Hinch testified that several explanations have been proposed for why some Late-run stocks begin their upriver migration without holding at the mouth of the Fraser River for several weeks:

- The fish are physiologically compromised in some fashion (e.g., accelerated maturation), so the system which regulates their capacity to live in the marine environment is altered, or some form of disease may be pushing them into freshwater.[766]
- The changing salinity concentrations in coastal areas make the fish perceive they are entering freshwater.[767]
- The “stay in the school” hypothesis – the high abundance of Summer-run stocks reaches the mouth of the Fraser River at the same time as the Late-runs, which may entice the Late-runs to migrate with the Summer-run stocks.[768]

Once Fraser River sockeye begin their upstream migration, their digestive tract shuts down. Dr. Welch described the transformation that occurs:

> Sockeye store fat as oil in the muscles and as they migrate up the river, for example, they burn that oil or fat to fuel the migration and they replace it with water. So as they progress up the river, their shape doesn’t change, but they replace fat, which is energy-rich, with water and they become softer.[769]

According to Dr. Hinch, warmer river temperature can affect fish entering the river for these reasons:

- The metabolic and cardiac systems can cease operation at certain critical temperatures, resulting in acute mortality.
- High flows or high temperatures can lead to more rapid metabolism of energy, using up energy resources.
- Diseases, many of which are temperature-dependent, can proliferate.
- The buildup of stress metabolites can create conditions for mortality.[770]

Mr. English testified that, through the use of radio transmitters in fish and monitoring locations along the Fraser River, it is possible to measure how quickly the returning salmon move upriver, and to assess their passage through challenging areas such as Hell's Gate and the Bridge River rapids. Early Stuarts move the fastest – one fish, tagged at Mission, swam 800 km to the Stuart system in 16 days, averaging 45–50 km per day. A Summer-run sockeye, moving more slowly, took 24 days of freshwater migration to reach the Chilko system. During 2009, migration speeds of Summer-run sockeye ranged between 32 and 40 km per day.[771]

Water temperature plays an important role in survival. In the ocean, sockeye prefer temperatures of 12–14°C, or even cooler. However, as the fish migrate upstream, river water temperature is frequently between 18 and 20°C. There is some evidence that sockeye will interrupt their migration by remaining in cooler lakes for a week or more in order to moderate high temperatures, before pressing upstream to their spawning area.[772]

Mr. Lapointe testified that Environment Canada records show that increasing Fraser River water temperature is a significant environmental change – eight of the 10 warmest summer river temperatures on record have occurred in the past 15 years.[773]

Freshwater contaminants

In Technical Report 2, Contaminants, authors MacDonald and others stated that exposure to endocrine-disrupting compounds may be an even greater concern for sockeye returning to the Fraser River than during the outmigration, for these reasons:

- The sockeye are exposed to persistent endocrine-disrupting compounds (e.g., PCBs) during outmigration and during their residence in the marine environment. Then, during upstream migration, they use lipid and protein stores to promote gonadal development and undergo morphological alterations. The rigours of upstream migration can result in a 50–90 percent depletion of somatic energy reserves, with the result that concentrations of those contaminants in somatic or gonadal tissues can increase dramatically between the time they enter the river and the time they arrive at their natal streams. Such contaminant magnification may result in concentrations in eggs that exceeded the toxicity threshold for salmonid fish of 3 ng/g lipid, a level associated with 30 percent mortality of eggs.[774]
- Exposure during upstream migration could also compromise immunocompetence. The result could make sockeye more susceptible to infection by disease agents, particularly during migration periods characterized by elevated water temperatures. This susceptibility could translate into increases in en route mortality and/or pre-spawn mortality.[775]

Infectious diseases

The author of Technical Report 1, Infectious Diseases, Dr. Kent, documented and evaluated the potential effects of diseases and parasites on Fraser River sockeye salmon, during both the freshwater and marine life stages. Dr. Kent identified the following pathogens as either high or moderate risk to Fraser River sockeye salmon during their upriver migration.

Fungi and related organisms. Species of the *Saprolegnia* genus infect the skin and gills, and almost always follow damage that has been caused to these tissues by other factors. With wild salmon, it is one of the most common infections of adult fish once they return to freshwater to spawn, and occurs in warmer waters or those with high organic load. The researcher rated the risk as moderate.[776]

Protozoa. Ichthyophthirius multifiliis parasites cause severe damage to the skin and gills, often killing the fish by asphyxiation due to the tissue reaction to the parasite in the gills. Whereas pre-spawn mortality as high as 70 percent has occurred in the Nadina River, it has not increased in severity since 1990. The researcher rated the risk as high, noting that severity would increase with increased water temperature and reduced water flows. *Cryptobia salmositica* is a blood flagellate common in salmonids from freshwater throughout the Pacific Northwest. In wild fish, the infection is usually seen in sexually mature salmon that have returned to freshwater to spawn. Infections are often lethal. In 2008, it was associated with pre-spawn mortality in sockeye at Weaver Creek. The researcher rated the risk as moderate.[777]

Myxozoa. The *Parvicapsula minibicornis* parasite targets the kidneys. The infection may be severe in sockeye adults suffering pre-spawn mortality compared with successful spawners. The researcher rated the risk as high. *Myxobolus arcticus* has been detected in some pre-spawn mortalities from Weaver Creek, along with a variety of other pathogens. The researcher rated the risk as low to moderate.[778]

Hydroelectric power projects

There are no hydroelectric power projects on the mainstem Fraser River.[779] Section 4 of the *Fish Protection Act* now prohibits bank-to-bank dams on a number of BC rivers, including the Fraser River.[780]

Hydroelectric power projects have the potential to change freshwater environments that support salmon populations. These changes may be grouped into two general categories:

- *barriers*, which are the infrastructure that prevent or affect upstream and downstream movement of fish; and
- *alteration of the flow regime*, which encompasses changes to stream characteristics related to the amount of water present in a stream and its movement.[781]

Three dams now operated by BC Hydro were built on migration routes for Fraser River sockeye: the Alouette, the Coquitlam, and the Seton.[782] There is also the Wilsey Dam at Shuswap Falls, but it is not located on a sockeye migration route, and although it has the potential to affect sockeye habitat below the dam, apparently it does not do so in a significant way.[783] The Alouette and Coquitlam dams, completed in the early 1900s, caused the extirpation of historic sockeye runs.[784] Fish passage structures were not provided at either facility.[785] The Seton Dam (part of the Bridge River-Seton power project near Lillooet), completed in 1956, is located on the migration route of two Fraser River sockeye runs (Gates Creek and Portage Creek); a fish ladder provides access to spawning grounds above the dam.[786]

The two large-scale hydroelectric projects in the Fraser River watershed with the potential to affect Fraser River sockeye during the return migration are BC Hydro's Bridge-Seton Power Project and Rio Tinto Alcan's Kemano Hydroelectric Project (which diverts water from the Nechako River).[787]

There are two issues associated with upstream passage at the Seton Dam that have the potential to affect the Gates Creek and Portage Creek sockeye populations:

- When returning to Seton Lake, sockeye must successfully pass the "tailrace" of the Seton powerhouse and enter the Seton River.* However, due to the strong smell of Seton River water pouring into the Fraser River from the turbine, sockeye tend to school in the tailrace, thus delaying their migration upstream. BC Hydro sought to mitigate this concern by diverting flow from Cayoosh Creek into Seton Lake, which reduced the ratio of Cayoosh Creek water in the Seton River. Studies indicated that sockeye would move past the tailrace if the concentration of Cayoosh Creek water in the Seton River was reduced to 20 percent for Gates Creek sockeye and less than 10 percent for Portage Creek sockeye. BC Hydro has, since 1979, attempted to meet dilution guidelines reflecting these numbers. But a recent study raised concerns that the tailrace may still attract and delay sockeye, even if guideline conditions are met for dilution.[788]
- If returning sockeye successfully pass the tailrace and enter the Seton River, they must then locate the entrance of the fish ladder and ascend it.[789] A recent study suggests that sockeye may have trouble locating the entrance of the fish ladder, possibly owing to high flow discharge from the Seton Dam into the Seton River.[790] When asked about this study, Dr. Bradford, research scientist with DFO and Simon Fraser University, cautioned that the results of the study should be interpreted carefully because some fish may have failed to traverse the dam on account of handling and other cumulative stresses.[791]

Under the St'át'imc (PC) Settlement Agreement, BC Hydro agreed to undertake a collaborative research program with the St'át'imc First Nation

* Water discharged from the powerhouse of a hydroelectric dam enters the river through a channel called the "tailrace." See PPR 21, p. 35.

to study factors that may impede the success of upstream migration of salmon at the Seton Dam, including potential impacts caused by tailrace delay and the fish ladder.[792] This research program has been incorporated as a term of the Bridge River Water Use Plan.[793] (For a discussion of the regulation of hydroelectric power projects of water plans and planning, see Volume 1, Chapter 6, Habitat management.)

Adult mortality during river migration and on spawning grounds

In Technical Report 9, Climate Change, authors Dr. Hinch and Dr. Martins stated that several behaviours and physiological systems which can facilitate energy conservation are critically important for successful migration. For example, sockeye populations with difficult river migrations depart the ocean with high reserve energy and morphologies – such as short and relatively round bodies – that favour energy conservation. Also, when river discharge is unusually high, migration rates of some stocks are slowed, extending migration duration by several weeks. This process can deplete energy reserves to levels below critical thresholds. In years of extremely high discharge, hundreds of thousands of Fraser River sockeye have died during their migration, and energy exhaustion is thought to be partly responsible.[794]

Temperature is the most important environmental factor governing fish during the return migration because of its underlying effect on physiological, ecological, and behavioural aspects of life history. En route mortality could occur in rivers as a result of several high-temperature-mediated factors, such as collapse of aerobic scope, poor recovery from stress and strenuous exercise, and increased susceptibility to disease and parasites. High river temperatures can also deplete energy resources by accelerating routine metabolism. This change is unlikely to cause mortality by itself, although energy depletion in combination with other thermal-mediated processes could play a significant role in en route mortality.[795]

The researchers stated that, because river discharge in most years since the early 1990s has not been exceedingly high, discharge alone is not believed to be the driving factor underlying recent years' trends in en route mortality.[796]

Regarding temperature, Early Stuart-run and Late-run stocks experience the coolest Lower Fraser River temperatures (approximately 12–16°C historical daily average), whereas Early Summer and Summer-run stocks experience the warmest temperatures (approximately 15–17.5°C). Long-distance migrating stocks and early entering Late-runs accumulate relatively high levels of "degree days," whereas stocks that migrate in early August encounter the peak Fraser River temperatures.[797]

Three broad changes to the thermal experience of Fraser River sockeye have occurred over the past several years:

- The Fraser River has experienced approximately 2°C warming in the summer compared with 60 years ago.
- There have been several recent years with record high temperatures during mid-summer.
- As described above, since 1996, segments of Late-run sockeye have been entering the river three to six weeks earlier than normal, and thus may encounter temperatures up to 5°C warmer than they normally would.[798]

Taken together, these facts indicate that thermal conditions have been one of the largest environmental challenges that migrating adult Fraser River sockeye have had to deal with over the past 20 years.[799]

Some Fraser River sockeye stocks (Adams, Stellako, Late Stuart, and Quesnel) are affected by warm river temperatures, while others, such as Chilko, are insensitive, at least up to 20°C. For those migrants that were affected by temperature, one study showed that 17–18°C was the tipping point, and at 19–20°C stocks were exhibiting 20–40 percent mortality. In a 2004 study of Weaver Creek sockeye, 100 percent of fish perished if they encountered river temperatures exceeding 20°C, with 90 percent mortality at 18–19°C, and 20–50 percent mortality at less than 17°C. The researchers concluded that patterns of en route mortality are stock-specific. Stocks appear to be physiologically fine-tuned to function best at the river migration temperatures they historically encountered.[800]

The researchers described how river temperature and disease can cumulatively affect survival. One parasite (*Parvicapsula minibicornis*) infects kidneys and gills of all adult Fraser River sockeye salmon as they migrate through the estuary.[801] In laboratory studies, kidney infection has been shown to start when accumulated degree days exceed 350, and to become full blown at approximately 500 degree days.*

They concluded that en route loss has occurred in all run-timing groups of Fraser River sockeye over the past 17 years, and there is ample evidence that adverse environmental conditions, in particular those related to thermal issues, are largely responsible for the patterns. En route loss has been least severe and least frequent in the Summer-runs and most severe and most frequent in the Early Stuart and Late-runs.[802]

The researchers concluded that recent trends in climate have very likely decreased Fraser River sockeye survival during this life stage over the past 20 years.[803] During the evidentiary hearings, Dr. Hinch addressed the possible explanations for a fish reaching the spawning ground but not depositing its eggs:

> So these fish, you have to remember from the moment they are entering freshwater they are on a trajectory to die. They are all senescing just like we all senesce as we get older, our bodies, our immune systems start to break down. Their immune systems are becoming dysfunctional during the freshwater migration, and when they get to the spawning grounds, their immune function is almost nil. They have no ability to fight off diseases by the time they get to spawning grounds.
>
> They are going through rapid, rapid changes in their physiological systems that are irreversible at that point, with reproductive hormones and stress hormones flying up the charts. So on top of the natural diseases that they may be encountering and incubating within them, they also have these rapid changes in their body physiology that's occurring naturally, and the rate at which that changes on spawning grounds not only is mediated by temperature, but also by the density of fish, as well as the amount of time they spend once they're on the spawning ground looking for a mate.[804]

In Technical Report 9, Climate Change, Dr. Hinch and Dr. Martins stated that levels of pre-spawn mortality (females that do not suffer en route mortality because they arrive at spawning grounds but that die with most of their eggs retained in their bodies) are highly variable among stocks, run-timing groups, and years.[805] The causes are complex and multi-factorial, and include disease, stress, and energy level in adults, and time alive on spawning grounds. Most of these factors are accentuated by increasing temperatures.[806]

Across all run-timing groups over the past 70 years, pre-spawn mortality averages approximately 10 percent. It has exceeded 30 percent in only 12 years, and has exceeded 40 percent in only four years. Unlike en route mortality, there is no clear indication that pre-spawn mortality has been increasing over the recent decades, with the possible exception of the past 25-year trend in Late-run pre-spawn mortality.[807]

The researchers also examined the different freshwater conditions experienced by returning adults in 2009 and 2010. Significant mortality can occur during the upstream migration. The researchers found:

- In 2009, river temperatures were well above the long-term average during much of the migratory period. Temperatures exceeded 18°C from the third week of July to the third week of August, and during that period temperatures rose above 20°C for nine days – levels of thermal stress which would be expected to cause significant levels of en route mortality. Also, at least 50–60 percent of the Late-runs migrated in-river earlier than historically normal, and it would be expected that a large portion of these early migrants would suffer either en route or pre-spawn mortality.[808]

* Accumulated degree days is calculated by multiplying the number of days (e.g., 20) that a fish is exposed to water of a certain temperature (e.g., 18°C), in this example to get 360 degree days.

- In 2010, river temperatures were generally above the long-term average, but not as high as 2009. Temperatures exceeded 18°C from the third week of July to the third week of August, but exceeded 19°C on only six days. Only approximately 40 percent of Late-runs migrated in-river earlier than historically normal.[809]

En route and pre-spawn mortality are significant factors that reduce the number of effective female spawners, and thus may pose a threat to the long-term viability of the populations that are particularly affected.

The researchers concluded that recent trends in climate have possibly decreased Fraser River sockeye survival during this life stage over the past 20 years.[810]

However, it is important to understand that, while stressors along the upstream migratory route affect harvest and escapement, they do not affect productivity measured as recruits per spawner, because salmon biologists estimate recruits by adding the adults that reach the spawning grounds to harvest and en route mortality. Thus, adult mortality during river migration does not explain the long-term decline in productivity discussed throughout this Report.[811]

High spawner abundance

There are several ways to illustrate the decline in Fraser River sockeye salmon. One way is through data relating to abundances or annual sockeye returns. The other is in reference to productivity – comparing the number of adults returning to spawn (recruits) with the number of spawning adults four years previously.

In Technical Report 10, Production Dynamics, authors Dr. Peterman and Dr. Dorner examined production dynamics. During the evidentiary hearings, Dr. Peterman said that productivity is simply a measure of how successful parents are at producing offspring that mature to come back to the coast.[812] One of the measures of productivity is "recruits per spawner," which is the number of adults that return to the coast before the onset of fishing, produced per spawner. He testified that, for most Fraser River sockeye stocks, there has been a declining number of recruits per spawner since the early 1980s.[813] This decline is shown graphically in Figure 2.4.3. As noted in my Interim Report, if the number of progeny is less than the parental numbers, the stock would appear to be in decline. Since the early 1990s, there was a steady decline until 2009, to the point where the ratio of returning progeny per spawner was well below the 1:1 replacement level.[814]

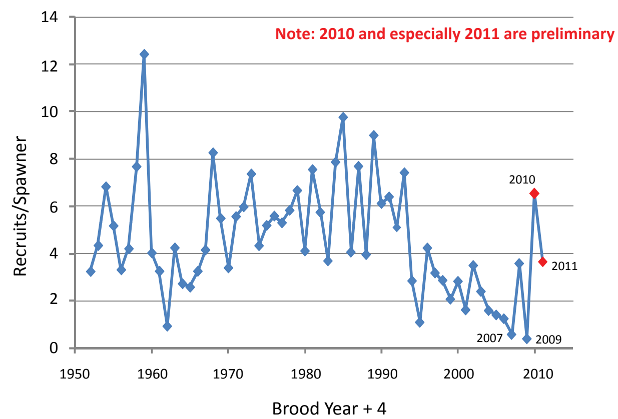

Figure 2.4.3 Annual variation in total Fraser River sockeye productivity, 1952–2011

Source: Exhibit 1851.

Dr. Peterman and Dr. Dorner examined the high spawner abundance, or "over-escapement," hypothesis. This hypothesis holds that a large number of spawners could be detrimental to productivity.[815] (For a discussion of the concept of over-escapement and its management implications, see Volume 1, Chapter 5, Sockeye fishery management.)

The researchers said that there are two ways in which increased escapement may have negative effects on productivity:

- *simple density dependence* – a large escapement (spawning population) in a given brood year (year of spawning) may cause the number of resulting adults to be low (less than the parental spawner abundance) owing to competition for limited resources (such as food for fry or oxygen for eggs in the gravel), and possibly mortality from the frequently observed diseases of sockeye salmon. The key issue in this context is the frequency and magnitude of their effect in years of high spawner abundance.[816]

- *delayed density dependence* – a large abundance of spawners in a given brood year would affect not only that brood year's productivity, but also productivity of the subsequent three brood years. The latter lag effect could occur through increased incidence of diseases on densely crowded spawning grounds, severe depletion of food supply in rearing lakes for juveniles across successive cohorts, increased reproduction and survival of long-lived predators of juvenile sockeye when their prey are plentiful, or any combination of the three. Delayed density dependence has been hypothesized to explain the phenomenon of cyclic dominance, but it appears other factors may be involved.[817]

The researchers stated that the literature offers some support that both simple and delayed density dependence occur for Fraser River stocks, but studies have so far failed to show conclusively that either form of density dependence has had a substantial influence on sockeye population dynamics in the Fraser River.[818] The effects can be examined most simply by plotting spawners and resulting recruits and looking for extremely low recruit numbers associated with extremely large previous spawning escapements.[819] Their analysis confirmed the findings of Dr. Carl Walters and others (2004): that there was no evidence of catastrophic decrease or collapse in recruitment per spawner following runs with very large numbers of spawners.* Witnesses who testified during the harvest management hearings generally agreed that the evidence did not support high spawner abundance leading to stock collapse.[820]

The researchers stated:

> For our 19 Fraser sockeye stocks across a total of 977 stock-years, there were only 70 instances (7.2 percent) in which the abundance of recruits was less than twice the number of effective female spawners (i.e., roughly below replacement). None of those cases followed an extremely large spawner abundance that subsequently led to chronic low abundance or stock collapse. The main reason for this result may be that there is only weak density-dependence in the spawner-to-recruit relationship for most of these Fraser sockeye populations.[821]

Looking for delayed density-dependent effects, which may be more diffuse over longer time periods, the researchers found that, for some Fraser River stocks (Quesnel, Chilko, and Fennell), recent declines in productivity were indeed associated with higher levels of spawner abundance.[822] Similarly, Dr. Walters, fisheries professor, University of British Columbia, and Dr. Brian Riddell, chief executive officer of the Pacific Salmon Foundation, formerly with DFO, cited the Chilko, Quesnel, and Adams as examples of stocks that have experienced a loss of productivity correlated with large escapements in prior years.[823] However, other stocks with declining productivity had normal or below-normal spawner abundances. This finding led Dr. Peterman and Dr. Dorner to conclude that "it appears that although density-dependence may have contributed to declines in productivity for some stocks such as Quesnel, it is *not* a sufficient explanation for the widespread regional pattern of decline in recruits per spawner apparent in the data[.]"[824]

There is some question over how many stocks show statistically significant delayed density-dependent effects. At the June 2010 PSC workshop, there was some support for the idea that delayed density dependence could have played a role in the long-term decline. At our hearings, Dr. Walters testified that, while in the early days none of the stocks showed such effects, new analyses "show it for most stocks."[825]

He expanded:

> It's possible that this is an artefact of confounding between the effects of population density and other things that are causing declining survival, coincident with high spawning stocks. But it's getting harder and harder to explain the patterns away as statistical artefacts of that kind.[826]

* The hypotheses being investigated in the Walters and others (2004) study (Exhibit 417) and in Technical Report 10, Production Dynamics, were somewhat different. Exhibit 417 looked only at whether there was stock collapse associated with higher spawner abundances, whereas Technical Report 10 examined whether high spawner abundances may be responsible for declines in Fraser River sockeye productivity, not necessarily extreme declines or collapse.

In contrast, Ken Wilson, a fisheries biologist, provided his view that over-escapement is a fisheries management construct better understood as "under-fishing," and should not be construed as biologically harmful.[827] He explained his view that historical Fraser River sockeye escapements may have been substantially larger than they are today – perhaps as high as 160 million – and that the ecosystems and the sockeye themselves have adapted to this natural periodic influx of nutrients.[828] Other witnesses also told me there were some ecosystem benefits to high levels of abundance, including more nutrients for other fish, for bears, and for eagles.[829]

Thomas Alexis from the Tl'azt'en Nation provided his perspective that there is no such thing as "over-escapement," nor has there ever been. He explained that fish are smart animals and they know where they are going. There are enough systems to accommodate these fish; if a stream is overfilled or overpopulated, the fish look to an empty or less full stream. For example, in Takla, there are over 100 natal streams that the salmon return to. If there was high abundance, then all these natal streams would be filled up and fully utilized, he testified.[830] Grand Chief Saul Terry of the St'át'imc Nation and a commissioner on the PSC spoke to over-escapement and dismissed this concern, saying that "nature looks after itself quite well," so long as we take care of habitats.[831]

Dr. Walters countered that allowing large escapements can ultimately create a strong cyclic dominant pattern wherein a number of large stocks are synchronized with respect to timing of a large year followed by three very small years.[832] This extreme synchronization, he explained, would not be good for a stable fishery or a stable ecosystem.[833]

Dr. Peterman and Dr. Dorner concluded that, although there is evidence of both simple and delayed density dependence for many Fraser River sockeye stocks, their results did not support the general hypothesis that efforts to rebuild Fraser River sockeye populations in recent years may have resulted in "over-spawning," thereby causing substantial declines in productivity for these stocks. They did, however, identify the Quesnel stock as an exception to this generalization.[834]

When Mr. Marmorek testified about his conclusions in Technical Report 6, Data Synthesis, there was some contradictory evidence on delayed density dependence in the form of an email exchange between Mr. Marmorek and Dr. Walters and a handout from the June 2010 PSC workshop, where Dr. Walters presented his hypothesis that several stocks exhibited delayed density dependence.[835] Mr. Marmorek explained the differences between Dr. Peterman and Dr. Dorner's methods, which were set out in detail in Technical Report 10, Production Dynamics, and Dr. Walters's methods.[836] Mr. Marmorek said that to compare the two, one would need to be able to examine Dr. Walters's methods.[837] He added that in Technical Report 6 he put "a lot more weight on the very thorough analysis by Peterman and Dorner in their Technical Report 10."[838]

Productivity comparisons across stocks

Across Fraser River sockeye populations

In Technical Report 10, Production Dynamics, authors Dr. Peterman and Dr. Dorner found clear shared trends across most Fraser River sockeye stocks, when clustered according to the four run-timing groups.* For the Early Stuart and most Early Summer stocks, productivity started a long downward trend in the 1960s and/or 1970s. Strong and persistent declines for the Summer and Late-run stocks occurred starting in the late 1980s.[839] The researchers concluded that most Fraser River sockeye stocks show declines in adult returns over recent years, and most of these declines have been associated with decreases in productivity (recruits per spawner).[840]

Between Fraser River stocks and non–Fraser River stocks

Dr. Peterman and Dr. Dorner also obtained data on abundance of spawners and their resulting

* Several stocks did not conform to the pattern of recent decline; most notably Harrison, Quesnel, and Pitt.

adult returns of all ages (recruits) for a total of 64 sockeye populations from Washington State, British Columbia, and Alaska.[841] They found that most Fraser River and many non–Fraser River sockeye stocks, both in Canada and the United States, show a decrease in productivity, especially over the past decade and often also over a period of decline starting in the late 1980s or early 1990s. In other words, declines since the late 1980s have occurred over a much larger area than just the Fraser River system and are not unique to it.[842] During the evidentiary hearings, Dr. Peterman noted that several stocks that migrate along the west coast of Vancouver Island, such as Washington State stocks and Great Central and Sproat Lake stocks, showed this decreasing productivity trend, although the Harrison River stock, which may follow a similar migratory route, showed increasing productivity.[843]

The researchers also found that *declines* in productivity of Fraser River and other BC stocks have generally coincided with *increases* in productivity of stocks in western Alaska, most notably Bristol Bay. The converse is also true. They reported that sea surface temperatures may be partially responsible for these patterns.[844] In testimony, Dr. Peterman said that the Bristol Bay anomaly may be explained by the regime shift in the 1970s.[845] Wind circulation patterns changed substantially. Those changes altered ocean currents, which led to an increase in productivity of the food supply – and the number of recruits per spawner increased dramatically.[846]

The researchers emphasized that their data analyses merely describe the extent to which time trends in productivity are similar across sockeye salmon stocks, but said that the causes for that similarity were not investigated in their study, except for delayed density dependence. However, they added that the large spatial extent of similarities in productivity patterns suggests that mechanisms which operate on larger, regional spatial scales, and/or in places where a large number of correlated sockeye stocks overlap, should be seriously examined in other studies.[847]

Moreover, the causes of declining trends in productivity may or may not have been the same across sockeye stocks from Lake Washington, British Columbia, southeastern Alaska, and the Yakutat region of Alaska. The prevalence of downward trends in productivity among these stocks might be coincidental. It is conceivable that, just by chance alone, processes that have operated independently in each sockeye population outside the Fraser River system (e.g., freshwater habitat degradation, contaminants, pathogens, predators) led to decreasing trends in productivity similar to the trends within the Fraser River.[848] However, in testimony Dr. Peterman gave his view that the coincidence was unlikely:

> To us it seems like there's a much greater chance that there's some shared trend across these populations to varying extents, than that there's some near coincidence of independently operating factor causing a downward trend in productivity of all these stocks simultaneously. It's possible that there's a coincidence. We can't deny that. But it seems unlikely over such a large spatial scale that that would happen. So it seems like there would be processes operating on a larger scale that would be affecting these populations simultaneously. And these could be things such as oceanographic patterns driven by climatic processes.[849]

Dr. Peterman added that it seems unlikely that these decreases in productivity are due to shared variation in freshwater processes, because those freshwater processes would have to occur in all those stocks.[850] He added that delayed density dependence can probably also be ruled out as the shared source of downward trend in productivity, since it appears to have played an important role only in the Quesnel stock.[851] Similarly, en route mortality can be ruled out, because estimates of adult recruits take en route loss into account.[852]

Dr. Peterman emphasized a need to know more about sockeye salmon in this environment:

> So if we really want to understand what is going on in the marine environment with these fish, I think we need to have a lot better coverage of where these fish are at various times, and that may require some tagging of a great extent, much larger than has been done for years, decades in fact, to find out where these fish are and what is happening to them.[853]

Data synthesis

The Inquiry commissioned ESSA Technologies Ltd. to synthesize the results of the Commission's other technical reports into an assessment of various factors potentially affecting the Fraser River sockeye fishery over the recent period of declining productivity.

The authors of Technical Report 6, Data Synthesis, organized their analysis of the other technical reports according to the Fraser River sockeye life stages, adopting a five-life-stages categorization. Their conclusions are summarized below.

Incubation, emergence, and freshwater rearing. The authors concluded that it is possible that climate change contributed to overall declines in productivity in recent decades. It is unlikely that most of the other stressors identified in the other technical reports did so.* No conclusion was possible respecting pathogens and hatchery-origin disease.[854]

Smolt outmigration. The authors' conclusions were the same for this life stage as above.[855]

Coastal migration and migration to rearing areas. The authors concluded that it was likely that climate change, and marine conditions along the nearshore migratory route, contributed to the overall declines in productivity. It was possible that predators contributed to the decline, and possible that disease originating at salmon farms contributed to the decline, if the conclusions of Technical Report 5D, Dill Salmon Farms Investigation, were accepted. The authors rated several other stressors as unlikely, or said that no conclusion was possible.[856]

Growth in the North Pacific and return to the Fraser River. The authors concluded that it was possible that predators, climate change, and marine conditions contributed to the long-term decline in productivity. They rated several other stressors as unlikely, or said that no conclusion was possible.[857]

Migration back to spawn. The authors concluded that climate change definitely affected harvest and escapement, but that en route mortality was unlikely to explain trends in recruits per spawner (productivity). They rated other stressors as unlikely, or said that no conclusion was possible.[858]

In their report, the authors of Technical Report 6, Data Synthesis, explained their usage of the terms "unlikely," "possible," and "likely":

> Even with considerable gaps in data and understanding, and mostly indirect evidence, contrasts over space and time in both salmon stock productivity and the potential stressors allow us to judge certain stressors to be unlikely to have been the *primary* factors causing declines in sockeye productivity or abundance. Other factors may be possible or even likely, provided that they fulfill most or all of various criteria (i.e., have a plausible mechanism by which survival could be affected; have generally exposed Fraser sockeye to increased stress over the period of productivity declines; correlate over space, time and stocks with variations in productivity; and (ideally) have other corroborating evidence from cause-effect studies).[859]

Having presented the conclusions of the authors of the technical reports, and having summarized the synthesis of those reports presented in Technical Report 6, I turn now to my own findings of fact respecting the causes of the decline of Fraser River sockeye salmon.

Notes

1 Exhibit 8, p. 10.
2 Exhibit 1454, pp. 23–24.
3 Mike Lapointe, Transcript, October 25, 2010, p. 24; Jim Irvine, Transcript, July 8, 2011, p. 91; Exhibit 1352.
4 Exhibit 1, p. 23.
5 Transcript, October 25, 2010, pp. 18–19.
6 Exhibit 783, p. 13.
7 Exhibit 783, p. 16.

* The researchers concluded that contaminants were unlikely to have contributed to the long-term decline in productivity. However, in reaching this conclusion, they considered only the impact of contaminants that were *measured* by Technical Report 2, Contaminants, and did not consider the potential impact of the large suite of chemicals (including endocrine-disrupting chemicals) that are not currently measured in the watershed (Transcript, David Marmorek, September 19, 2011, pp. 17, 22–24, 60).

8 Exhibit 783, p. 17.
9 Exhibit 783, pp. 17–18.
10 Exhibit 783, pp. 20–21.
11 Exhibit 783, p. 21.
12 Eduardo Martins, Transcript, May 8, 2011, p. 23.
13 Exhibit 553, pp. 21–22.
14 Exhibit 553, p. 22.
15 Exhibit 553, p. 25.
16 Exhibit 553, p. 26.
17 Exhibit 553, p. 26.
18 Exhibit 553, pp. 28–29.
19 Exhibit 553, p. 29.
20 Jason Hwang, Transcript, April 5, 2011, p. 1; Rebecca Reid, Transcript, April 5, 2011, p. 1; Patrice LeBlanc, Transcript, April 5, 2011, p. 2.
21 Randy Nelson, Transcript, April 8, 2011, p. 75.
22 Exhibit 562, p. 22.
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25 Exhibit 562, pp. 30–33.
26 Exhibit 562, p. 39.
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28 Exhibit 562, pp. 41–42.
29 Exhibit 562, pp. 44–45.
30 Exhibit 562, p. 46.
31 Exhibit 562, p. 57.
32 Transcript, March 10, 2011, p. 33.
33 Marc Nelitz, Transcript, March 10, 2011, p. 33.
34 Transcript, March 10, 2011, p. 29.
35 Transcript, June 17, 2011, p. 6.
36 Transcript, June 17, 2011, p. 12; see also Exhibit 1107.
37 Peter Tschaplinski, Transcript, June 17, 2011, p. 12; see also Exhibit 1107, p. 27.
38 Transcript, June 17, 2011, pp. 50–51.
39 Transcript, June 17, 2011, pp. 48–49, 57–58, 76.
40 PPR 17, p. 72.
41 PPR 17, p. 72.
42 PPR 17, p. 72.
43 PPR 17, pp. 74.
44 Transcript, June 17, 2011, p. 38.
45 Peter Tschaplinski, Transcript, June 17, 2011, p. 10; Ian Miller, Transcript, June 17, 2011, p. 47.
46 Peter Tschaplinski, Transcript, June 17, 2011, p. 10.
47 Peter Tschaplinski, Transcript, June 17, 2011, pp. 10–11.
48 Exhibit 1124, p. 5.
49 Transcript, June 17, 2011, p. 11.
50 Transcript, June 17, 2011, p. 73.
51 Exhibit 1125.
52 Peter Tschaplinski, Transcript, June 17, 2011, pp. 45–46; see also Exhibit 1125, p. 32.
53 Transcript, June 17, 2011, pp. 75–76.
54 Transcript, September 15, 2011, pp. 1–3; see also Exhibit 912.
55 Transcript, September 15, 2011, p. 5.
56 Michael Bradford, Transcript, September 15, 2011, pp. 4–5.
57 Michael Bradford, Transcript, September 15, 2011, p. 5.
58 Michael Bradford, Transcript, September 15, 2011, p. 5.
59 Michael Bradford, Transcript, September 15, 2011, pp. 5–6.
60 Transcript, September 16, 2011, p. 8.
61 Transcript, September 16, 2011, p. 8.
62 Transcript, September 15, 2011, p. 6.
63 Steve MacDonald, Transcript, September 15, 2011, p. 8.
64 Transcript, September 15, 2011, p. 6.
65 Transcript, September 15, 2011, pp. 2–3, 9; see also Exhibit 1760.
66 Transcript, September 15, 2011, p. 57.
67 Exhibit 1879, p. 1.
68 Transcript, September 16, 2011, p. 45.
69 Jason Hwang, Transcript, September 16, 2011, p. 45.
70 Michael Bradford, Transcript, September 15, 2011, p. 37; Jason Hwang, Transcript, September 16, 2011, p. 36.
71 Transcript, September 15, 2011, p. 38.
72 Transcript, September 15, 2011, p. 38.
73 Transcript, June 7, 2011, pp. 94–95; see also PPR 14, p. 45.
74 Exhibit 833, p. 26.
75 Exhibit 833, p. 26.
76 PPR 14, p. 68.
77 Exhibit 826, p. 108; see also Exhibit 833, pp. 15–16.
78 Exhibit 826, p. 40.
79 PPR 14, p. 40.
80 PPR 14, p. 40.
81 PPR 14, p. 40.
82 PPR 14, p. 40.
83 PPR 14, p. 41.
84 PPR 14, pp. 41–42.
85 PPR 14, p. 42.
86 Mike Lapointe, Transcript, October 25, 2010, pp. 17–18.
87 Mike Lapointe, Transcript, October 25, 2010, p. 19.
88 Mike Lapointe, Transcript, October 25, 2010, p. 19.
89 Mike Lapointe, Transcript, October 25, 2010, p. 16.
90 Transcript, October 25, 2010, p. 23.
91 Transcript, October 25, 2010, p. 20.
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95 Exhibit 783, p. 19.
96 Exhibit 783, p. 20.
97 Transcript, May 5, 2011, p. 31.
98 Exhibit 783, p. 21.
99 Exhibit 783, p. 22.
100 Scott Hinch, Transcript, March 8, 2011, p. 10.
101 Scott Hinch, Transcript, March 8, 2011, p. 10.
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108 PPR 15, pp. 7–8.
109 PPR 15, p. 68.
110 Janice Boyd, Transcript, June 13, 2011, p. 5.
111 Janice Boyd, Transcript, June 13, 2011, p. 5.
112 Transcript, June 13, 2011, p. 33; see also Exhibit 826, p. 97.
113 Robert Grace, Transcript, June 13, 2011, p. 79.
114 Exhibit 826, p. 21; see also Exhibit 562 and PPR 15, pp. 72-112.
115 Exhibit 826, p. 22.
116 Michael Hagen, Transcript, June 13, 2011, p. 34; see also Exhibit 826, pp. T-23-T-26 (Table 3.7); PPR 15, pp. 75–76.
117 Michael Hagen, Transcript, June 13, 2011, p. 35.
118 Michael Hagen, Transcript, June 13, 2011, pp. 35–36.
119 Michael Hagen, Transcript, June 13, 2011, p. 67.
120 Michael Hagen, Transcript, June 13, 2011, p. 36; see also Robert Grace, Transcript, June 13, 2011, p. 51.
121 Transcript, June 13, 2011, pp. 69–70.
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126 Exhibit 1449, p. 9.
127 Exhibit 1449, p. 10.
128 Exhibit 1449, pp. 12–13.
129 Transcript, August 24, 2011, p. 1
130 Exhibit 558.
131 Kristina Miller, Transcript, August 24, 2011, pp. 4–5.
132 Kristina Miller, Transcript, August 24, 2011, p. 5.
133 Transcript, August 24, 2011, p. 92.
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142 Transcript, August 24, 2011, p. 12.
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146 Kristina Miller, Transcript, August 24, 2011, p. 15.
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148 Transcript, August 24, 2011, p. 16.
149 Kristina Miller, Transcript, August 24, 2011, pp. 16–17.
150 Kristina Miller, Transcript, August 24, 2011, p. 17.
151 Kristina Miller, Transcript, August 24, 2011, p. 17.
152 Transcript, August 24, 2011, pp. 2–3; see also Exhibit 1511.
153 Transcript, August 24, 2011, p. 18.
154 Kyle Garver, Transcript, August 24, 2011, p. 18.
155 Kyle Garver, Transcript, August 24, 2011, p. 18.
156 Kyle Garver, Transcript, August 24, 2011, p. 18.
157 Transcript, August 24, 2011, pp. 18–20.
158 Transcript, August 24, 2011, pp. 22–23; see also Exhibit 1512, p. 2.
159 Transcript, August 24, 2011, p. 25; see also Exhibit 613G.
160 Kristina Miller, Transcript, August 24, 2011, pp. 26–27.
161 Transcript, August 24, 2011, pp. 30–31.
162 Transcript, August 24, 2011, p. 31.
163 Transcript, August 24, 2011, p. 33.
164 Transcript, August 24, 2011, p. 59.
165 Transcript, August 24, 2011, p. 59.
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167 Transcript, August 24, 2011, p. 95.
168 Transcript, August 24, 2011, p. 95.
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170 Transcript, August 24, 2011, pp. 98–99.
171 Transcript, August 24, 2011, p. 99.
172 Transcript, August 24, 2011, p. 13.
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174 Transcript, December 15, 2011, pp. 102–3.
175 Transcript, December 15, pp. 102–3; see also Exhibit 2084.
176 Transcript, December 15, p. 103.
177 Mike Lapointe, Transcript, October 25, 2010, pp. 19–20; see also Exhibit 1, pp. 14–15.
178 Exhibit 1, p. 15.
179 Mike Lapointe, Transcript, October 25, 2010, pp. 24–25.
180 Transcript, July 6, 2011, pp. 49–50; see also Exhibit 1293.
181 Exhibit 783, pp. 21–22.
182 Exhibit 783, p. 23.
183 Exhibit 783, pp. 24–25.
184 Exhibit 783, pp. 24–27.
185 Exhibit 783, p. 28.
186 Exhibit 783, p. 29.
187 Exhibit 553, p. 26.
188 Exhibit 1449, pp. 14–15.
189 Exhibit 562, p. 28; see also PPR 19, pp. 42–43.
190 Exhibit 562, p. 34.
191 Exhibit 562, p. 35.
192 Exhibit 562, p. 41.
193 Jason Hwang, Transcript, September 16, 2011, p. 36; Glen Davidson, Transcript, September 16, 2011, p. 36.
194 Craig Orr, Transcript, September 15, 2011, p. 38; see also PPR 21, pp. 52–53.
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196 Exhibit 562, pp. 41–42.
197 Exhibit 735, p. 2.
198 Exhibit 735, p. 26.
199 Exhibit 735, p. 27
200 Transcript, May 9, 2011, p. 55.
201 Exhibit 735, pp. 27–28.
202 Exhibit 735, p. 29.
203 Exhibit 735, p. 35.
204 Exhibit 735, p. 36.
205 Exhibit 735, p. 37.
206 Exhibit 767 at pdf p. 4; Testimony of Carol Cross, May 4, 2011, p. 11.
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212 Transcript, August 22, 2011, p. 46.
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215 Exhibit 1454, p. 83.
216 Exhibit 1454, pp. 83–84.
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218 Transcript, August 22, 2011, p. 43; Exhibit 1454, p. 4.
219 PPR 14, pp. 46–47.
220 PPR 14, p. 47.
221 PPR 14, p. 52.
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223 Exhibit 573, p. 30.
224 Exhibit 73, pp. 75, 77; Don MacDonald, Transcript, May 10, 2011, pp. 32, 59.
225 PPR 14, p. 50.
226 Exhibits 73, p. 76.
227 Transcript, June 14, 2011, p. 2; see also Exhibit 1043.
228 Peter Ross, Transcript, August 17, 2011, pp. 92–94; see also PPR 19, pp. 13–14.
229 Exhibit 573, pp. 31–32.
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231 Exhibit 73, p. 9.
232 Exhibit 73, p. 77; see also Exhibit 826.
233 Exhibit 73, p. 77; see also Don MacDonald, Transcript, May 10, 2011, p. 70.
234 Exhibit 73, p. 76.
235 Peter Ross, Transcript, August 17, 2011, pp. 94–96, 101–2; Peter Ross, Transcript, August 18, 2011, pp. 1–3; Peter Ross, Transcript, June 14, 2011, pp. 21–22.
236 Peter Ross, Transcript, August 18, 2011, p. 2.
237 Transcript, May 9, 2011, p. 28.
238 Don MacDonald, Transcript, May 9, 2011, p. 28.
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261 Don MacDonald, Transcript, May 9, 2011, pp. 55–56.
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268 Exhibit 826, p. 59.
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271 Exhibit 826, pp. 70–72.
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274 Exhibit 826, p. 96.
275 Ken Ashley, Transcript, June 14, 2011, pp. 62–63; Peter Ross, Transcript, June 14, 2011, p. 62; Graham van Aggelen, Transcript, June 14, 2011, p. 63.
276 Transcript, June 14, 2011, pp. 12, 25.
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279 Exhibit 826, p. 103
280 Don MacDonald, Transcript, May 9, 2011, pp. 47–48; see also Exhibit 826, p. 103.
281 Transcript, May 9, 2011, p. 55.
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286 Transcript, June 6, 2011, pp. 4–5.
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288 Exhibit 826, p. 112.
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291 Transcript, May 10, 2011, pp. 32–33, 41.
292 Transcript, June 6, 2011, p. 16. See also Peter Ross, Transcript, June 14, 2011, p. 87.
293 Don MacDonald, Transcript, May 9, 2011, pp. 13–14; see also John Carey, Transcript, June 7, 2011, p. 14.
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300 Ken Ashley, Transcript, June 14, 2011, p. 18.
301 Ken Ashley, Transcript, June 14, 2011, p. 19.
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307 Ken Ashley, Transcript, June 14, 2011, p. 36; see also Exhibit 1052, p. 1.
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386 Exhibit 1303, p. 2.
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474 Are Nylund, Transcript, December 15, 2011, p. 116.
475 Frederick Kibenge, Transcript, December 15, 2011, p. 119; Kristina Miller, Transcript, December 15, 2011, p. 128.
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483 Simon Jones, Transcript, September 6, 2011, p. 6.
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485 Simon Jones, Transcript, September 6, 2011, p. 4.
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496 Transcript, September 6, 2011, pp. 7, 15; see also Exhibit 1476; Exhibit 1771; Exhibit 1764, p. 4.
497 Lawrence Dill, Transcript, August 26, 2011, p. 25; Brendan Connors, Transcript, August 26, 2011, p. 25.
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531 Transcript, September 6, 2011, pp. 18, 26.
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533 Transcript, August 25, 2011, pp. 100–1; see also Exhibit 1536, p. 20.
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536 Transcript, September 6, 2011, p. 24; see also Exhibits 1764 and 1780.
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552 Transcript, August 25, 2011, p. 76; Lawrence Dill, Transcript, August 25, 2011, pp. 97–98; Donald Noakes, Transcript, August 25, 2011, p. 98; see also Exhibit 1540, pp. 17–19; Exhibit 1536, pp. 14–15.
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