Impacts of a Freshwater or Marine Oil Spill on Aquatic Resources from the Fraser River or its Tributaries

Kinder Morgan Trans Mountain Expansion Project

Prepared for

Shxw'ōwhámel First Nation and Peters Band

by

Tracy K. Collier, PhD

Consultant in Environmental Toxicology

9390 Miller Rd NE, Bainbridge Island, WA, USA, 98110

April 23, 2015

Table of Contents

Introduction

Trans Mountain Pipeline ULC (hereafter Trans Mountain), a subsidiary of Kinder Morgan, is proposing to greatly expand the capacity of an existing pipeline that runs from Alberta to British Columbia, through the territories of the Shxw'ōwhámel First Nation (SFN) and Peters Band (PB). Pipeline capacity will increase from approximately 300,000 barrels per day to 890,000 barrels per day. This expansion will involve construction of almost 1000 kilometers of new buried pipeline as well as re-activation of existing pipelines that are currently not in operation. In addition, the Westridge Marine Terminal located in Burnaby BC will be expanded to include three additional berths, with a concomitant increase in tanker traffic bearing pipeline products from Burnaby out through the Salish Sea.

The products that are proposed for transportation via pipeline and tanker ships are stated to be a variety of crude oils, including diluted bitumen, synthetic crude oils, light and sweet crude oils, synthetic bitumen, and diluted synthetic bitumen. Up to 630,000 barrels per day of products will be shipped on tankers from the Westridge Marine Terminal, and a large proportion of the products to be transported via pipeline and tanker traffic will be diluted bitumen. All crude oils are known to be toxic to a wide range of animal species, including many species of fish that are of high importance to the people of the SFN and PB. Crude oils are toxic to humans as well.

The SFN and PB have commissioned this technical report in order to better understand the risks to fisheries resources that would be posed by a) oil spills into or near the Fraser River and its tributaries, resulting from pipeline ruptures or other accidents; or b) oil spills into marine waters adjacent to the Westridge Marine Terminal, or in the Salish Sea as tankers make their way out to the open ocean. Their concerns are well-founded, because previous spills of crude oils, including diluted bitumen, have impacted a wide range of fisheries, both though direct impacts of the oil and its constituents on fish, as well as the fish being rendered unfit for consumption because of contamination with those same substances.

This report focuses primarily on the potential impacts of spilled oil on Pacific salmon (sockeye, coho, Chinook, chum, and pink salmon), steelhead and rainbow trout, as well as white sturgeon. Pacific salmon and trout are of high importance as a food supply in addition to having great cultural significance. White sturgeon were historically of high significance as a food supply for indigenous people living along the Fraser River, but now are present in such low numbers that this species is subject to very limited harvest. Nonetheless, the sturgeon of the Fraser River are of extremely high cultural significance to the people of the SFN and PB.

This technical report describes the properties of crude oils, focusing on diluted bitumen and synthetic crude oil, and what constituents are believed to be most toxic to fish. The life histories of important fish species are also described, including at what stages these fish are likely to come into contact with spilled oil. The effects of oil exposures on fish are characterized, paying attention to both embryonic and juvenile/adult life history stages, as these have very different sensitivities to crude oils. This is followed by a discussion of how oil spills can render many species of fish unfit for consumption for extended periods of time. Finally, there is a section that summarizes the likely impacts of both a freshwater as well as a marine spill on the ability of the SFN and PB to utilize fishery resources.

Characteristics of products that might be spilled

In their application materials, Trans Mountain states that there are a variety of crude oils that will be transported via the expanded pipeline and increased tanker traffic. These include diluted bitumen, synthetic crude oils, light and sweet crude oils, synthetic bitumen, and diluted synthetic bitumen (see Volume 8A, table 5.4.2, page 8A-539 of project application materials). Diluted bitumen is expected to be a 'large proportion' of the products to be transported, and synthetic crude oil is also a substantial product derived from Alberta oil sands. The composition of these products is not fixed, i.e. the constituents and their concentrations will likely vary from batch to batch. To place these products in perspective, they can be compared to crude oils, which are well known and would share many of their characteristics, and to other synthetic crude oils derived from oil sands mining.

Crude oil

Crude oil is a mixture primarily of hydrocarbons, i.e., compounds composed only of carbon and hydrogen. However, most crude oils include small amounts (<1%) of compounds containing sulphur, nitrogen, or oxygen, and traces of elements, including metals of concern such as cadmium and mercury. As shown in Figure 1, the hydrocarbons in oil vary widely in their molecular size, shape and properties, but can be classified into *saturates*, i.e., compounds without double bonds, including aliphatics (linear or branched chains of carbon atoms, including waxes) and cyclic compounds (rings of carbon atoms); *aromatics* (one or more benzene rings, with or without alkyl side chains); and *resins* and *asphaltenes* (very large and complex molecules that combine aromatic rings, aliphatics, and unsaturated rings). The majority of compounds in oil are non-polar, i.e., they do not have chemical structures or characteristics that allow them to readily dissolve in water. Solubilities of oil-derived compounds range from less than 1 ng/L (essentially insoluble) to 2000 mg/L (low solubility).

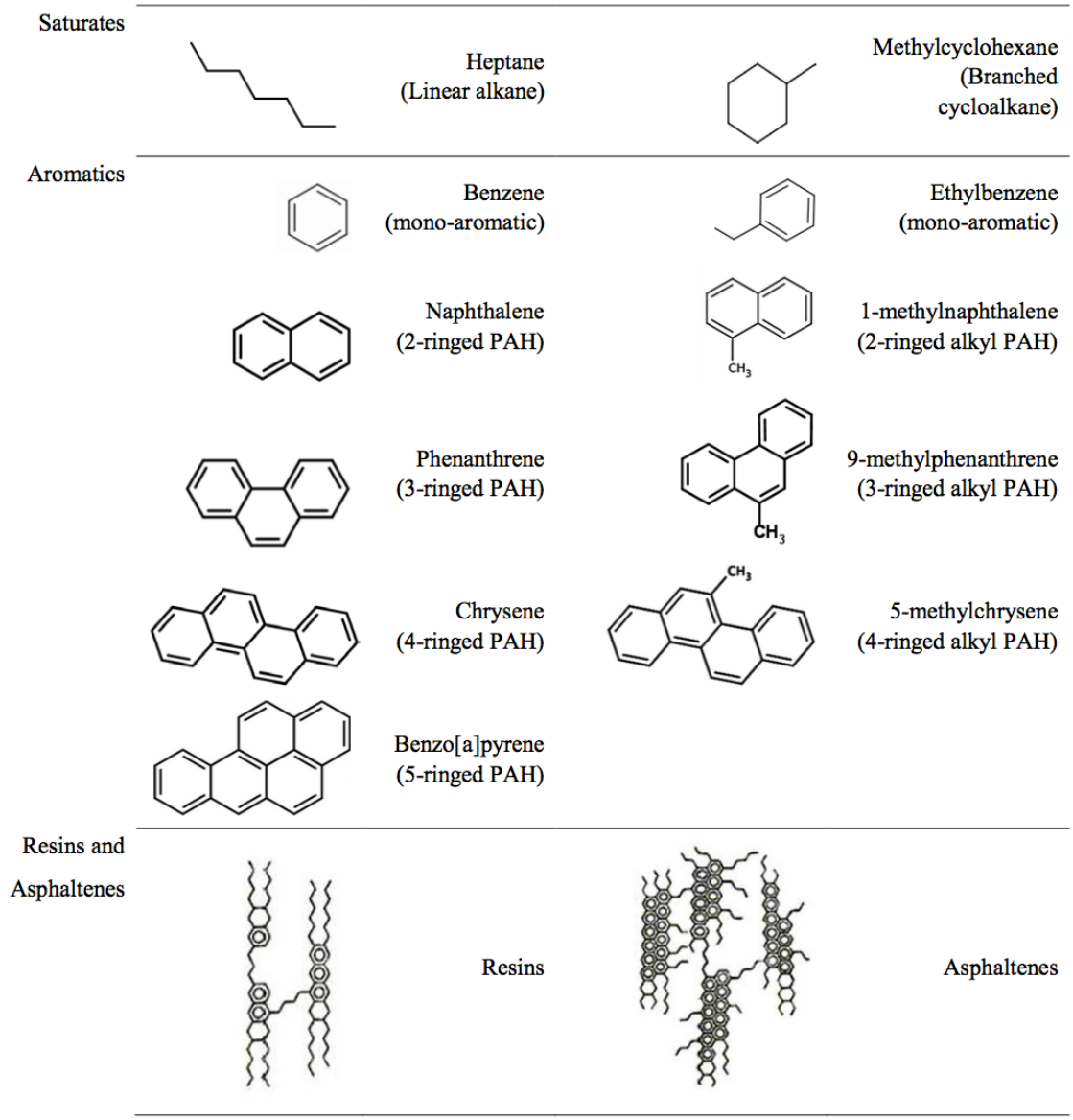

Figure 1. Typical structures of hydrocarbons found in crude oils. The structures shown are a tiny fraction of the total.

Aromatics include *mono--aromatics* (single benzene rings) with and without alkyl substituents, *polycyclic aromatic hydrocarbons* (PAHs; two or more benzene rings fused together in flat plates, with and without alkyl substituents), and *heterocycles* (PAHs containing one or more atoms of nitrogen, sulphur, or oxygen). The most abundant aromatics are the monoaromatics (1.62% by weight in Alaska North Slope Crude (ANSC) oil, and the most abundant of these are benzene, toluene, ethylbenzenes, and xylene (*BTEX*); oils can be classified by their BTEX content. The mono-aromatics are highly volatile, relatively water soluble, and relatively easily

biodegraded, so they are generally lost quickly (hours to days) after an oil spill as the oil weathers when exposed to air, water washing, and bacteria.

The most abundant and the smallest PAHs in crude oil are the two ringed naphthalenes, which are also lost quickly following a spill. Those with alkyl substituents are heavier, less volatile, and less water soluble, and the rate at which they are lost during weathering decreases with increasing molecular size or weight. Similarly, other PAH with 3 to 8 rings and varying numbers of alkyl substituents will persist longer in spilled oil in proportion to their increasing molecular size and weight. Alkyl-substituted PAH generally comprise more than 90% of all PAH in oil, in contrast to combustion-derived PAH, which are predominantly un-substituted. In crude and refined petroleum products, total PAH (sum of alkyl substituted and unsubstituted PAH) vary from almost absent in gasoline to 0.1 to 1.5% by weight in light to heavy crude oils, 3 to 6% by weight in heavy fuel oils (Martin 2011), and 0.01 to 1.1% in various samples of oil sand products, including diluted bitumen and synthetic crude oils.

Linear and branch chained aliphatics exhibit similar behaviour, with short-chained low molecular weight compounds lost quickly from oil after a spill and persistence in oil increasing in proportion to chain length and molecular size. Very large aliphatics are classified as waxes, with extremely low water solubilities. Similarly, the resins and asphaltenes are generally so large that they are not volatile, they are insoluble in water, and thus are persistent, comprising the majority of tarry materials that remain following weathering of spilled oil. If the smaller components of oil are mostly removed during weathering, the residue of waxes, resins, and asphaltenes may no longer be liquid. These components determine much of the oily or tarry nature of fresh and weathered oil, and they provide a reservoir and transport medium for the lower molecular weight compounds.

Crude oils can be classified according to their relative proportions of these major constituents. A light crude contains a higher proportion of low molecular weight aliphatics and aromatics than a medium or heavy crude, and a lower proportion of waxes, resins and asphaltenes. As a consequence it will have a lower viscosity (flows more easily, even at low temperatures) and a lower density or specific gravity. All except the heaviest crude oils have a specific gravity less than 1.0, and will float on the surface of the water. When fresh, films of lighter oils have a greater propensity to be broken up into small droplets than heavy oils, so they are more easily dispersed by mixing energy or by chemical dispersion.

As will be described later, the toxicity of oil to aquatic organisms is a function of chemical composition. The low molecular weight aliphatics and aromatics are acutely toxic to fish within their solubility limits, and can cause fish kills within the first 24 hours following a spill. The higher molecular

weight 3- to 5-ringed PAH are toxic to fish embryos, causing developmental defects measurable at hatch, and often death of embryos because they do not develop sufficiently to begin feeding. Developmental toxicity to newly fertilized fish embryos can be caused by exposures to oil as brief as one hour at concentrations typical of oil spills, or at concentrations 100-fold lower if the exposure is prolonged throughout development (McIntosh et al. 2010).

'Sweet' oils are those low in sulphur, which may be present as a component of polycyclic compounds known as thiophenes. Oils that contain more sulphur are termed 'sour' and have a noticeable sulphur odour. The sulphur-containing PAH include dibenzothiophenes, naphthobenzothiophenes, and their alkyl congeners, and they may be as toxic, or more toxic to fish embryos as alkyl PAH (Rhodes et al. 2005).

Diluted bitumen

Bitumen is diluted in Alberta with naphtha, condensate, or synthetic oil at a typical ratio of 25:75 because it is too viscous to flow through a pipeline in its natural state (Bakker 2011). For purposes of conducting risk assessments and modeling spill scenarios, Trans Mountain has assumed that Cold Lake
Winter Blend diluted bitumen will be the product being transported. Diluted bitumen resembles a heavy crude oil, in that it may contain higher proportions of high molecular weight compounds compared to ANSC; it also contains 3.0% sulphur by weight (Bakker 2011), and would be classified as 'sour' due to its high sulphur content. When weathered, it will lose a smaller overall proportion of hydrocarbons and there will be larger amounts of residual oil stranded on surface structures or entrained into sediments. Due to its high viscosity, diluted bitumen will be less easily dispersed than many crude oils.

Synthetic crude oil

Synthetic crude is upgraded bitumen, refined sufficiently to flow through a pipeline, with the addition of naphtha, condensate or other gas oil materials (Bakker 2011). Synthetic crude oil shares many characteristics with ANSC oil and should behave in a similar manner to the general pattern described above when first spilled. However, its characteristics may change considerably with weathering as the more volatile diluents are lost with weathering. If so, the residual would be more like bitumen than oil, with an increased viscosity, density and tendency to sink. Synthetic crude also contains sulphur and metals. The concentrations of metals in most crude oils (and in diluted bitumen) are sufficiently low that toxicity to fish species would not be expected unless all are released from the oil at once, although metals might be a food web issue for benthic organisms. The sulphur suggests the presence of dibenzothiophenes, which are sulphur-containing

heterocycles highly toxic to fish embryos (Rhodes et al. 2005; Incardona et al. 2005).

Major fisheries potentially affected by a spill

Pacific salmon and trout

The life histories of Pacific salmon, as well as steelhead and rainbow trout, are fairly well described elsewhere (Hart 1973; Quinn 2005), so they are only briefly summarized here.

The 5 species of Pacific salmon native to the Fraser River are Chinook salmon (*Oncorhynchus tshawytscha*), sockeye salmon (*Oncorhynchus nerka*), coho salmon (*Oncorhynchus kisutch*), chum salmon (*Oncorhynchus keta*), and pink salmon (*Oncorhynchus gorbuscha*). All of these fish are anadromous, meaning that they spawn in freshwater, spend some portion of their lifecycle in freshwater before migrating to the ocean for growth to adult size, and then return to freshwater to spawn. Pacific salmon all deposit their eggs in the gravel and cobble of streams and rivers, generally in areas with hyporheic flow. Adults are semelparous, meaning that they always die after spawning, although spawning can consist of more than one spawning event over a period of several days.

Steelhead trout and rainbow trout are members of the same species, *Oncorhynchus mykiss*. Steelhead trout are also anadromous, but are iteroparous, meaning that they can spawn multiple times and do not necessarily die after spawning. Rainbow trout do not migrate to the ocean for growth to adult size, instead they are resident in freshwater streams, rivers, and lakes. Similar to Pacific salmon, both steelhead and rainbow trout spawn in gravel and cobble bottoms of streams and rivers. Because of similarities in life history, and especially because all of these species (Pacific salmon and trout) deposit their fertilized eggs in bottom substrates, the effects of a major oil spill will be broadly similar across species. The primary factors that will affect these species are their presence or absence following a spill, and where their fertilized eggs are deposited. As described below, entrainment of diluted bitumen into river sediments and gravels is likely to occur after a spill, and poses a significant risk to all fish that utilize bottom substrates for spawning.

Many distinct population segments of Pacific salmon and steelhead are protected by legislation aimed at maintaining species from becoming extinct. In the United States this law is the Endangered Species Act, and in Canada the law is the Species at Risk Act. Populations of many of these species are maintained or augmented by culture of fish in hatcheries and release of juveniles into freshwater, and there is considerable complexity

involved in managing these fisheries, both in marine waters as well as in freshwater systems.

White sturgeon

> *So the people here at Shxw'ōwhámel, we have a very close relationship with the sturgeon. We know that now, today, the sturgeon -- there's a conservation concern for the sturgeon, so the only time that we have sturgeon is when we have to share a meal. Every spring and every fall we have a ceremony where we share food and clothing with our ancestor spirits, it's a way that we take care of them. And so that's the only time we pretty well now have sturgeon because of the restrictions on the sturgeon. And we maintain that connection to the sturgeon through, in our language, what we refer to the as the Soowahlie. Soowahlie is like the life force or the spirit.*[1]

The white sturgeon (*Acipenser transmontanus*) is the largest freshwater fish in North America, and the third largest of all sturgeon species. These impressive fish grow to almost 6 m in length and can weigh over 500 kg (Hart 1973), and are very long lived, reaching ages of over 100 years (Rien and Beamesderfer, 1994). The white sturgeon of the Fraser River are very important in the culture of indigenous people living in the Fraser River valley, including the Shxw'ōwhámel First Nation and Peters Band, with strong ties between the people and the sturgeon (see above). White sturgeon was historically a very important species in the food supply of the Shxw'ōwhámel First Nation and Peters Band, because they are available at all times of the year, even in winter. In fact the white sturgeon was created from people by Chíchelh Siyá:m, the Creator, to provide a winter food supply. However, with the arrival of Europeans the Fraser River sturgeon were dramatically overfished, such that by 1901 it was noted by the fisheries inspector that the fishery for these sturgeon was "practically extinct commercially" (Semakula and Larkin, 1968). Today members of the SFN and PB are rarely allowed to harvest sturgeon.

White sturgeon are benthic (i.e. bottom-dwelling) fish, inhabiting large river systems on the west coast of North America from Alaska and British Columbia south to central California. In the Fraser River, white sturgeon spend most if not all of their lives in fresh water, although they are capable of migrating into estuaries and marine waters. Females reach maturity at 11-34 years of age, and spawn once every 3-4 years. The sequence of spawning and early development is as follows (modified from Perrin et al. 2000)

---

[1] From Paragraph 2307 of Sonnie McHalsie's oral history of the Shxw'ōwhámel (and Sto:lo), listed under NEB Hearing Order OH-001-2014, Volume 6:

- Eggs and sperm are broadcast. Fast water prevents clumping and smothering. Eggs are adhesive. Suspended sediment can adhere to the egg surface and inhibit clumping.
- The adhesive eggs sink (Wang et al. 1985) and because of asymmetrical density, eggs settle micropyle side up. Opportunity for fertilisation is high because each egg has 5-40 micropyles and the sperm remains motile for 3-5 minutes.
- The embryonic development period is about 7 days. In culture, broods hatch at the same time. Hatching is complete in 20-48 hrs. It occurs in darkness in the lab, which may be an adaptation to avoid predators. The optimum temperature for larval viability is 11-17°C (Wang et al. 1985). Higher temperatures result in higher mortality and hatching at earlier developmental stages.
- Larvae are black and about 10 mm in length. They are thought to be planktonic, drifting in ambient current. In the laboratory, larvae frequently swim up and sink down in the first day after hatching. At water velocities near 8 cm/s in the lab, larvae enter a hiding phase faster and earlier than when velocities are near 2 cm/s.
- Dispersal lasts about 6 days in the lab (Brannon et al. 1986a). During dispersal, Conte et al. (1988) found that larvae become photophobic in less than 4 days post- hatch and will hide in response to light at that point. Within the first three days after hatching, larvae may actively select habitats of certain velocities and in so doing may move several times a day in response to variation in velocities. This behaviour may be particularly prevalent downstream of dams from which diurnal variation in water releases may occur. Because of the dispersal behaviour, larvae can be found in wide ranging water depths; PSMFC (1992) report observations of larvae captured at 9-19 m in the Columbia River.
- Larvae progress to a hiding phase in which they sink to the bottom and aggregate, sometimes burying themselves in gravel or other substratum materials. This period lasts up to another 6 six days. After yolk sac absorption (about 12 days post-hatch) larvae emerge and begin active feeding. Within 20-30 days, metamorphosis is complete.

Once the larvae have metamorphosed into juveniles, they feed on insects, small fish, and small crustaceans such as mysid shrimp. The adult diet can include fish such as eulachon, sculpins, young sturgeon, sticklebacks, and lamprey, as well as clams, mussels, and crayfish. Adults feed opportunistically, by extending their mouth near the bottom of the river and 'sucking up' their food.

Because white sturgeon are so long-lived and can feed at a fairly high trophic level, they often bioaccumulate substantial body burdens of

persistent organic pollutants, such as polychlorinated biphenyls, DDTs, and methylmercury. Their benthic lifestyle, especially for eggs and larval fish, make them potentially very susceptible to spills of oil that sinks and becomes entrained in river sediments (see below).

Exposure scenarios

The fate and distribution of spilled oil in a freshwater environment, and the resulting exposure of fish to oil or its constituents, will depend on the interactions among the physical and chemical characteristics of the oil, the physical and hydrogeological characteristics of the Fraser River and its tributaries, the weather during the spill, and the weathering of the oil. In the marine environment going the Westridge Marine Terminal and out through the Salish Sea to the open ocean, additional considerations include tidal action as well as wave heights and directions.

Source of petroleum spill

Spill scenarios include pipeline breaks that would contaminate the Fraser River or its tributaries through direct discharge to the watercourse (e.g., breaks at tributary crossing) or overland transport from a break away from the river. In the nearshore marine environment and out through the Salish Sea, spills could result both from shipping accidents as well as from terminal operations. In addition to spills of diluted bitumen or synthetic crude oil from shipping accidents in the marine environment, there is a risk of spills of heavy fuel oils, diesel, and other refined products used by vessels or shore facilities as part of terminal operations.

Duration of the spill

The duration of a pipeline spill will be a function of the size of the break and the time needed to detect a break and close adjacent valves. For a complete rupture of the pipe, the time for the pipeline section between valves to empty will depend on the upstream grade. Because the pipeline is under pressure, there is a potential for a slow leak without a significant detectable pressure drop that would activate alarm systems and close pipeline valves. The result could be a prolonged discharged detected only by the presence of oil on the ground or in the river. For a marine spill associated with shipping, the duration of a spill would be highly variable, depending on the nature of the accident and the amount of oil being carried.

Volume of oil spilled

For spills into the Fraser River or its tributaries, a maximum spill of 1300 m^3, the volume between adjacent valves, is a worst case scenario in the region near Hope BC, assuming a full bore pipeline rupture, according to the Trans

Mountain application materials. This is equivalent to about 8,200 barrels of oil, or almost 350,000 gallons. However, this assumes that leak detection systems function perfectly, and that the cause of a break is not the destruction of the valve itself, e.g., by a land slide. Additionally, human error can contribute substantially to the amount of oil released from a pipeline break. In the case of the Kalamazoo River spill of diluted bitumen, in 2010, a pipeline rupture went undetected for over 17 hours, despite numerous alarms and reports of foul odors in the affected communities—the pipeline operators assumed the alarms were due to a large air bubble between batches of crude oil (http://crrc.unh.edu/sites/crrc.unh.edu/files/media/docs/Workshops/oil_sands/The%20Dilbit%20Disaster-%20Inside%20the%20Biggest%20Oil%20Spill%20You've%20Never%20Heard%20Of%20(3%20Parts).pdf). Where a break occurs at a river or stream crossing, it is likely that the majority of oil would be discharged directly to the watercourse. For breaks between crossings, the oil would spread across land before encountering a stream, so only a portion would enter a watercourse. If the projected maximum spill was discharged directly to a water course, a layer of oil 1 mm thick and 200 m wide would be 6.5 km long. However the application materials submitted by Trans Mountain (7.1.4.2.1) state that oil would be visible as far as 100 km downstream from such a spill in this area. It is inarguable that major reaches of the Fraser River would be subject to extensive oil contamination.

Time of exposure to spilled oil

The influence of time on exposure of aquatic organisms to the toxic components of oil is important. Time affects the risk of spills to aquatic environments in relation to rates of weathering, duration of the spill event (short-term spill compared to a prolonged leak), duration of exposure of organisms, and time of year (or season), as it affects when organisms are present and the interaction of the environment with rates of weathering. In particular, salmon and trout that spawn in the Fraser River deposit fertilized eggs in buried gravel shoals where embryos develop and hatch. White sturgeon deposit adhesive eggs that attach to the surface of the gravel. For the most part, hatched embryos of these fish species remain sessile in or on the gravel until the yolk sac is absorbed and the larval fish commence swimming and feeding. The time from fertilization and incubation to emergence of swimming fry is crucial since the young fish are immobile and would be vulnerable to a spill event occurring in these spawning habitats. As well, sensitivity to oil toxicity will vary with the age of the embryo, with highly sensitive stages in development including gastrulation, organogenesis, and the development of the capacity to metabolize and excrete PAH, which can modify the response of fish to oil exposure (Brinkworth et al. 2002).

Weathering

When oil is spilled, the process of weathering begins immediately, and significant amounts of the low molecular weight aliphatic hydrocarbons (< C12) and aromatic hydrocarbons (e.g., BTEX, naphthalenes) will be lost within the first 24 hours due to evaporation, dissolution into water, biodegradation, and photo-oxidation (McAuliffe 1977; George et al. 1995). The effects of weathering and mixing would be greatest for light oils, but synthetic crude oils and diluted bitumen will weather at a slower rate. The components that can be lost from these oils due to weathering are a much smaller percentage of the total mass of oil, and the chemical composition of synthetic oil and diluted bitumen will not differ markedly from the composition where it was spilled, if there is a rapid transit downstream to marine waters (1 to 2 days). However, if the oil pools in backwaters or eddies, or low energy tidal marshes, and there is a substantial loss of the diluent from diluted bitumen, the residual material may resemble bitumen more than oil, and a substantial portion would likely sink and accumulate in bed sediments. As oil weathers and mixes with water, there is a greater potential to form water-in-oil emulsions, which may sink or be washed ashore as sticky masses of oil. For all oils, biodegradation will not likely play an important role in weathering during the first few days, because this is a much slower process than evaporation or dissolution in water.

In general, weathering processes will occur most rapidly in the summer during warm, dry weather, and will slow significantly in the winter, with cloud cover, lower temperatures, shorter days, and ice or snow cover.

With weathering, the residual oil contains a higher proportion of high molecular weight components (e.g., PAH with greater than 3 rings, waxes, resins, and asphaltenes) so that the viscosity and specific gravity of the residual oil would increase, with a greater potential to sink or be entrained in the water column. With high energy mixing (e.g. due to wave action, or turbulence), fresh, unweathered oil may be dispersed as droplets. Oil emulsions, oil slicks, and droplets can also mix with particles from the water or bed sediments, thus increasing the propensity to sink with increasing specific gravity or density, if the particles are sand or gravel. The interaction with particles would be least during low summer flow when the water is clear, and greatest during snowmelt and during rainstorms when soil erosion creates high levels of turbidity due to suspended solids. If the suspended solids are finely ground, the flocculent oil-mineral aggregates may be sufficiently light to remain in suspension (Sun and Zheng 2009), in which case the particles may facilitate the contamination of sediments by hyporheic flow.

Highly weathered heavy oil may develop a thick shell of waxy or asphalt-like material (i.e. tar balls) that would hinder the transfer of PAH to water, even

though the interior of the tar ball can remain liquid (Goodman et al. 2003). The formation of tar balls is critically important because they deliver oil directly to sediments, where liquid oil and PAH may be released to habitats essential for benthic species and fish reproduction. For example, Bunker C oil was stranded on a shoreline in Nova Scotia following the 1970 sinking of the *Arrow* in Chedabucto Bay. Within five years, the oil had weathered to tar balls and hard pavements, but the undersides of tar deposits at the upper high tide mark were still liquid, resembling freshly-spilled oil (Vandermeulen and Gordon 1976). The liquid oil remained as a reservoir to periodically re-oil the beach when storms disturbed the beach. In 1997, 27 years after the spill, sufficient oil was still present in the beach to release oily sheens when sediments were disturbed, and concentrations were still acutely toxic to marine amphipods, although other test species were unaffected (Lee et al. 2003). Oil in contaminated sediments persists longer than in the water column, because it is not mobile and not readily subject to dispersion and dilution (Fingas et al. 2005; Hollebone et al. 2011).

Oil spilled on land may weather significantly before overland flows reach the Fraser River or its tributaries. During overland flow, the oil will accumulate soil particles, vegetation and other debris that will raise the overall specific gravity. The experience at Wabamun Lake, AB in 2005, demonstrated that a large portion of spilled heavy fuel oil, which had a nominal specific gravity of less than 1.0 and thus should have floated, instead sank due its contact with soil during a short (<100 m) overland flow (Parker-Hall an Owens, 2006). As a consequence, a large part of clean-up costs and environmental damage was associated with the removal of oil and contaminated vegetation from shoreline reed beds where pike and other fish species spawn (Evans 2008). The oil was present as small (<5 mL) and large (>1 L) masses of liquid oil contained by a waxy exterior caused by weathering, and significant amounts were also found offshore at whitefish spawning shoals (Evans 2008).

Stranding of oil

The effects of oil spills in water are often evident in the extent to which oil slicks strand on surfaces such as shorelines to create sticky coatings that ultimately weather to tar. Much of the oil spill response described in the Trans Mountain application concerns the recovery of oil from surfaces, and the success of remediation is judged by visual surveys of affected shorelines by a Shoreline Cleanup Assessment Technique (SCAT). The amount of shoreline contaminated in a river will depend on the sinuosity of channels, channel braiding, emergent bars, the amount of debris, and the nature of the surfaces coated. The Fraser River and its tributaries have frequent tangles of woody debris from toppled trees, and river banks and numerous gravel bars characterized by grain sizes ranging from sand particles to stones that are 1 to 20 cm in diameter or larger. The extent of

oiling will also be determined by whether a spill occurs on a rising or falling stage of river height. In both cases, the length of shoreline will change, and particularly on a falling stage, much larger areas will be contaminated. In effect, the 'bathtub ring' will broaden as water levels fall. For a rising stage, increasing turbulence due to increasing river flow rates may re-mobilize oil that was stranded at a low stage, limiting the bathtub ring to the high water mark. Similar effects would be seen in marine waters with rising and falling tides.

Dispersion of oil

Some of the impacts of spilled oil are associated with the extent to which it strands on surfaces, including on wildlife. Therefore, to protect some wildlife, spilled oil is often chemically dispersed to enhance the loss of hydrocarbons by volatilization, dilution in water, and biodegradation. Dispersion aims to dilute surface oil into a much larger water mass by allowing downward mixing into near-surface waters (McAuliffe 1977). The extent of dilution will be a function of mixing energy, and the net effect is to increase the volume of water containing measurable (and likely toxic) concentrations of oil, while at the same time enhancing the rate of photooxidation and biodegradation (McAuliffe 1977). Compared to surface slicks, droplet formation accelerates all of these processes due to increased surface-to-volume ratios and increased rates of partitioning of hydrocarbons from oil to water. Because many of the impacts of oil on aquatic species are associated with the toxicity of petroleum compounds dissolved in water, the exposure of fish to hydrocarbons, and the risks of toxicity, will increase when oil is chemically dispersed.

The extent to which oil is dispersed into the water column as fine droplets is a major factor controlling exposure of aquatic species to the constituents of oil. When oil is dispersed into fine droplets (1 – 100 μm in diameter), the surface-to-volume ratio increases, and the rate of partitioning of hydrocarbons from oil-to-water is enhanced. The droplets, or particles of oil, are colloids that may be stabilized in water by surface interactions (Shaw 1977). The extent of droplet formation will be reduced if oil forms stable emulsions with water, which is likely for diluted bitumen because of its high viscosity and density. In contrast, synthetic crude oils are more likely to form fine droplets (Belore 2010), increasing the exposure of aquatic species to their constituents.

Each droplet is a separate phase from water, and the rate of partitioning of hydrocarbons from oil to the water phase is enhanced in smaller droplets by a shorter diffusion distance from the center of a droplet to its surface. The effect of droplet dispersion is not to make the hydrocarbons more water soluble, but rather to transport oil into the water column where the droplets act as small reservoirs that release the constituents of oil to the water

phase. Laboratory tests of oil dispersed in fresh water have demonstrated that the exposure of fish to PAH, and hence the risk of toxicity, was increased by 6 to 1100-fold, depending on the nature of the oil being tested (Ramachandran et al. 2004; Schein et al. 2009). During the process of dispersion, the concentrations of soluble components may exceed the threshold for toxicity to aquatic organisms for hours or days (McIntosh et al. 2010). For this reason, chemical dispersants are not usually considered for spill management in small or confined water bodies where the opportunities for dilution are limited, and where sensitive organisms may be abundant.

Nevertheless, oil dispersion is an important risk factor in the Fraser River and its tributaries because oil can also be physically dispersed by mechanical energy associated with standing waves and water turbulence in rivers and streams, particularly during flood conditions. The entrainment of droplets and water-in-oil emulsions into the water column increases the potential for transferring oil to sediments where it may continue to release components to water at concentrations toxic to aquatic organisms that live on or in sediments, or that feed on sediment-dwelling species. Filter-feeding organisms can accumulate particulate oil, making it bioavailable to the benthic food web or to fish that feed on drifting invertebrates.

Entrainment of oil into sediments

The entrainment of oil within bed sediments of the river is of more concern to fish than contamination of the shoreline. In virtually all cases of oil spills to rivers, there has been significant oil contamination of sediments, with measurable amounts of residual oil for months and years following the spill (e.g., Pine River, B.C. (Goldberg, 2006); Kalamazoo R., MI http://epa.gov/enbridgespill/pdfs/enbridge_fs_20110811.pdf). In addition to significant fish kills that occurred immediately following these spills (e.g., Baccante 2000), the ecological impacts of spilled oil were associated primarily with sediment contamination and oil toxicity to benthic invertebrates, a major source of fish food, and oil toxicity to fish species that deposit eggs in nests created in the gravel of bed sediments. The Pine River spill on August 1, 2000 provided an excellent example. After a spill of about 475,000 L of heavy sour crude to the river, about 29% was mechanically removed. However, sediments were significantly contaminated in first months following the spill (AMEC 2001), and oily sediments were still evident after 5 years, although much reduced in frequency and concentration (Goldberg 2006). Similarly, following marine oil spills, such as the *Amoco Cadiz, Exxon Valdez, Braer, and Erika*, the mixing of spilled oil into sediments resulted in prolonged contamination. Such sediment-entrained oil can then be remobilized by wave action associated with storm event, or burrowing organisms, leading to the potential for prolonged exposure to marine species (Law et al, 2002).

There are several possible mechanisms by which bed sediments could be contaminated with oil. If an oil spill is followed by a severe flood, contaminated surface sediments on shorelines and gravel bars can be eroded and mixed with clean sediments, carrying oil-coated sediments deep within new bars formed downstream as the river channel is re-structured. The residual hydrocarbons in the unweathered or partially weathered oil coatings would be free to partition into interstitial water that flows through the bed sediments.

If oil is mixed into the water column as small droplets or particulates mixed with sediments, it is likely that oil will also be entrained into sediments where salmonids spawn and their eggs incubate. As river currents encounter gravel bars or the downstream side of pools, they exert hydrostatic pressure that drives water into the bar. At the downstream edge of gravel bars, and below pools, there is a negative pressure. The result is a variable but predictable induced flow of water through the gravel substrate, called hyporheic flows, with measurable currents as deep as several meters below the bed of the river (Tonina and Buffington 2007, 2009a, 2009b; Buffington and Tonina 2009). Hyporheic flows transport dissolved oxygen into gravel bed sediments, and remove metabolic waste products such as ammonia from eggs that have been buried by spawning salmon and trout. However, hyporheic flows may also entrain pollutants into bed sediments (Tonina and Buffington 2009b), such as dissolved and particulate oil, and the particulate oil would be intercepted and retained by the smaller size fractions of gravel. As water flows over and through this stranded oil, the constituents of oil that are toxic to the early developmental stages of fish (eggs, post-hatch embryos, larvae) will partition from the oil to water to the limits of their water solubility, creating conditions that may reduce embryo survival and fisheries productivity in areas that have been oiled.

These exposure conditions have been modelled in laboratory tests, using containers of oil-contaminated gravel as hydrocarbon desorption columns to supply flows of hydrocarbon-contaminated water to containers of trout or salmon embryos (e.g., Carls et al. 1999; Heintz et al. 1999; Martin 2011). Depending on the oil tested, the concentrations of oil on gravel that can cause elevated rates of developmental anomalies and mortality ranged from 2 to 8000 mg oil/kg gravel, corresponding to concentrations of TPAH between 1 and 100 µg/L in interstitial waters (Marty et al. 1997b; Carls et al. 1999; Heintz et al. 1999; Martin 2011). While high rates of hyporheic flow may dilute the constituents of oil dissolved into interstitial waters, the flow rates will vary widely, and there will be areas in most gravel beds with flows sufficiently low that hydrocarbon concentrations would approach saturation. With many salmonid species as well as white sturgeon spawning in the Fraser River and its tributaries, it is likely that the full range of habitat

available is used, and that an oil spill may render much of it unsuitable for embryo survival.

The persistence of oil in sediment could threaten the continued production of species that are particularly vulnerable to oil spills by virtue of a long residence time in river sediments (increases the likelihood and severity of oil exposure) or a short life cycle (vulnerable to repeated weak year classes due to on-going recruitment failure). Pacific lamprey (*Lampetra tridentate*) would be the species most vulnerable to prolonged exposure to oil in sediments, since embryos and ammocoetes reside in sandy and silty sediments for 3 to 7 years (Beamish 1980; Beamish and Levins 1991). Pink salmon would be most vulnerable to repeated recruitment failure because they have a two-year spawning cycle, and there are no reserve year classes that would spend a prolonged time at sea while the river recovers from oil pollution.

Summary of exposure scenarios for the Fraser River

Section 7.1.4, of *Volume 7 Risk Assessments and Management of Pipeline and Facilities Spills – Scenario 3: Fraser River near Hope, British Columbia, RK 1,072.8* of the Trans Mountain application, states that oil spilled to surface waters of the Fraser River. or of a tributary will flow quite quickly, reaching 60 to 100 km downstream. Fast-flowing rivers typically do not mix laterally unless there are rapids, tributaries inflows, or, in the case of surface oil, lateral winds. Thus, the side of the river where the oil entered will likely be most affected by stranding, as will gravel bars that cause the river to divide or divert from its channel. Effects on fish resident in the river would likely be immediate and catastrophic, with extensive fish kills caused by the low molecular weight components of oil that readily dissolve into water to the limits of their solubility. Surviving fish would be those in un-exposed backwaters or tributaries. The Trans Mountain application materials acknowledge this acute impact as a certainty because of past experience in other rivers affected by oil spills.

The fate of oil spilled to the river will depend to a great extent on flow and flood state of the river. At low flows, much of the oil will be transported directly downstream because there will be insufficient energy to mix it into the water column as oil droplets, and there will be only low concentrations of suspended solids to accelerate sinking and transfer of oil to sediments. Some will wash up on shore, stranding on gravel bars, river banks, drift wood, and vegetation. This oil will not likely have an effect on fish unless a severe flood mixes the contaminated material into new gravel bars before clean-up can remove it from the river bank. Nevertheless, there will be transfer of oil to sediments. Where the water flow is in a shallow sheet over gravel substrates, some oil may contact and strand on bottom sediments, and in all reports of oil spills to streams, oil has been found in bottom sediments where it can be difficult to remove. Thus the extent of chronic

impacts on benthos and fish under low flow conditions will likely be site specific, but more limited than if a large proportion of the oil is entrained in sediments.

If oil spills occurred at higher flow rates, there will be increased mixing energy and turbulence, and standing waves will be evident in riffles and rapids. As flow rates increase with rainfall or snowmelt, rising water would carry oil higher onto river banks, progressively stranding more oil on river banks, gravel bars, dead trees etc. Oil slicks would be broken up by turbulent mixing in rapids, so that oil droplets would be entrained into the water column, and under these conditions weathering would occur faster because of the greater surface-to-volume ratio of droplets compared to surface slicks. With flood conditions, the turbidity of the river will increase as soil erodes into the streams and as bottom sediments are moved and mixed by stronger currents. The result will be greater interactions of soil particles with oil droplets and a larger proportion of oil will interact with sediments, mixing with the bed load of sediment mobilized by the flood, and being entrained into gravel substrate by hyporheic flow. The results of this scenario will be long term (years) impacts on benthic invertebrate productivity and on survival and recruitment of fish larvae. It is quite possible for large portions of the Fraser River to become effectively sterile, with fish populations reduced considerably.

Toxicity of oil and oil components to fish

Oil can harm aquatic biota by coating the organism (e.g. birds and fur bearing mammals), by coating the substrates on which they live (e.g. benthic species), by contaminating their food supply, by the toxicity of oil dissolved in water, by being ingested, or by being inhaled (e.g. marine mammals, birds, and terrestrial mammals). Fish could be exposed to dietary oil if they consume filter feeders, because filter feeders can accumulate droplets of oil (Neff and Burns 1996). Benthic fish species (those that live in association with sediments, such as sturgeon and sculpins) or species with a benthic life stage (e.g., eggs and embryos that are deposited in sediments or attached to rocks or vegetation), would also be exposed to oil that coats substrates. Because of gill respiration, all fish would be exposed to oil droplets in water, and to oil-derived compounds dissolved in water.

Oil toxicity to fish arises from the direct action of hydrocarbons on tissues, or the indirect effects of hydrocarbon derivatives created by metabolism in fish tissues or by photoactivation of light sensitive PAHs in fish tissues. Indirect effects also could include suffocation if there is sufficient microbial

activity to rapidly biodegrade oil and reduce water levels of dissolved oxygen. This chapter describes the broad array of direct and indirect effects on fish of exposure to petroleum products and their toxic constituents that have been observed in laboratory and field studies of oil toxicity.

Acute toxicity of oil

Acute toxicity refers to any effect observed after a ***short*** exposure period, and/or an effect that becomes apparent within a short period of time following exposure. Acute is a relative term. What is acute for an adult salmon (e.g., 1 or 2 days in a 6 year life span) would be chronic for a bacterium whose life span is 1 or 2 days. This report will use 'acute' to refer to exposures or toxic effects that occur within 1 to 4 days, and 'chronic' to refer to exposures or responses that may take weeks, months, or years.

These categories can overlap. For example, the concentrations of oil that cause toxicity decrease as exposure duration increases. Acute exposures (1 to 2 hours) of Atlantic herring (*Clupea harengus*) embryos to dispersed oil immediately after egg fertilization caused toxicity that was evident in embryos when they hatched 12 days later, thus it was a chronic or delayed effect (McIntosh et al. 2010). Because increasing exposure time creates an increase in overall exposure to hydrocarbons in water, the median effective concentration (EC_{50}) that was lethal to herring embryos decreased with increasing exposure time. For 24 hour exposures, the concentrations causing long-term embryo toxicity had decreased by 3-fold compared to the 1-hour exposure, and by 40-fold if the exposure was prolonged throughout the entire 12 day period of embryonic development (McIntosh et al. 2010).

While McIntosh et al. (2010) measured the chronic toxicity of acute exposures to oil, most acute toxicity refers to acute or short-term mortality tests with juvenile fish. Typical examples of acute toxicity of oil to fish were observed after oil spills in the Pine River in British Columbia. In 1994, a spill of 30 m^3 (30,000 L) of gasoline and 24 m^3 of diesel fuel caused a fish kill of 150 mature and 1000 juvenile individuals (Goldberg 2011). On August 1, 2000, a pipeline rupture (in about the same area as the 1994 spill) leaked 985 m^3 of crude oil, half of which (475 m^3) entered the river. About 1600 dead fish were collected downstream of the rupture (Goldberg 2011), but the river was difficult to survey. Overall estimates of total fish mortalities ranged from 25,000 to 250,000.

As reviewed earlier, the components of oil considered to be acutely lethal are low molecular weight (LMW) alkanes, monoaromatics (benzene, toluene, ethylbenzene, xylenes; BTEX), and PAHs (2-ringed naphthalenes). These compounds are sufficiently water soluble to quickly reach lethal concentrations in water in contact with fresh oil. While their concentrations may diminish rapidly due to dilution, volatilization, and biodegradation, lethal

concentrations may persist if conditions do not favor weathering (e.g., cold, cloudy conditions, with little wind) or if there is a continuous input of fresh oil.

Hydrocarbons have long been thought to cause acute toxicity by narcosis, a reversible, non-specific effect akin to over-anaesthesia. While narcosis implies one mode of action, a review by Campagna et al. (2003) indicated a variety of different molecular interactions between lipid-soluble organic compounds and different membrane receptors. Despite multiple modes of action, the acute toxicity of lipid-soluble petroleum hydrocarbons is correlated to their octanol-water partition coefficients (Kow), calculated as the ratio of their solubilities in n-octanol, a surrogate for lipid, and water. The larger the molecular size of a hydrocarbon, the more lipid soluble and the less water soluble it is, so that more is taken up by fish through their gills. The result is a linear relationship between the log Kow and log bioconcentration factors (log BCF), i.e., the ratio of chemical concentrations in fish tissues and concentrations in water. Ultimately, the uptake of hydrocarbons, and hence toxicity, is limited by molecular size – for hydrocarbons with a log Kow greater than 6.0, the molecular size is too great to allow rapid passage through complex cell membranes.

For the Pine River spills, only small amounts of gasoline and diesel fuel were required to cause a fish kill comparable to the larger spill of crude oil, likely because these products contained a much higher proportion of the LMW components associated with acute lethality. However, in-depth studies on the potential for chronic exposure and effects after the Pine River oil spill are unavailable. Based on experiences in the Pine River, spills of light petroleum products (e.g. gasoline or condensate) in the Fraser River would cause much larger fish kills than would a comparably sized spill of diluted bitumen. However, as will be reviewed below, the heavier oil may cause a much greater chronic toxicity to fish embryos.

An oil spill to the Fraser River or one of its tributaries will likely cause an immediate fish kill in any area covered with an oil slick. The fish kill will last about 24-48 h, or until the oil has weathered significantly or cleared from the surface. An on-going input of fresh oil will prolong the conditions lethal to fish beyond 24-48 hours.

The extent of fish kills will vary with weather and flow conditions as they affect transit times to the estuary, the extent of weathering following the spill, and the presence and abundance of each species

Because the river turbulence would mix the contaminants dissolved from oil throughout the water column, a fish kill would affect all species and life stages present in the river at the time of the spill.

Chronic toxicity of oil

Chronic toxicity refers to effects on exposed individuals that develop over a long period of time, either from a brief exposure (i.e., delayed effects), or from a prolonged or chronic exposure. Compounds associated with chronic toxicity will be those that persist after a spill and that are small enough to be bioavailable, i.e., they can cross biological membranes and reach toxic concentrations in fish tissues. These would include intermediate-sized linear or branch-chained alkanes with 12 to 24 carbon atoms and 3- to 6-ringed PAH and heterocycles. Of these, PAH are considered to be the most chronically toxic (Tuvikene 1995). The 3- to 5-ringed alkyl PAH are the most likely PAH causing chronic toxicity to fish embryos, based on studies of single compounds [e.g., alkyl phenanthrenes,(Turcotte et al. 2011)], on similarities between the toxic effects of whole oil and of reference PAH such as retene [e.g., diesel oil (Schein et al. 2009)], the correlation between concentrations of specific families of PAH and chronic toxicity in studies of weathered oil (e.g., Carls et al. 1999), and effects-driven chemical fractionation of crude oil (Hodson et al. 2007a).

In fractionation studies with Alaska North Slope Crude (ANSC) and Scotian Light (SCOT) oils, fractions rich in alkyl phenanthrenes, fluorenes, chrysenes, pyrenes, dibenzothiophenes, and naphthobenzothiophenes were toxic to Japanese medaka embryos and caused CYP1A induction in rainbow trout (Hodson et al. 2007a). Less toxic fractions contained low molecular weight aliphatics and aromatics (including alkyl naphthalenes) and the very high molecular weight waxes, resins, asphaltenes and PAH. Alkyl PAH concentrations were much higher in ANSC than in SCOT fractions, and ANSC fractions containing alkyl PAH were more toxic than equivalent SCOT fractions.

Although the chronic toxicity of complex mixtures of hydrocarbons such as crude oil is associated with exposure to PAH, predicting the toxicity of crude oil is difficult because there are too few data on the toxicity of the thousands of individual PAH present in oil. The acute toxic potentials of compounds found in oil can be calculated by comparing the concentration of compounds in water to critical concentrations that causes toxicity (Di Toro et al. 2000). While this method was first developed to estimate acute lethality (usually 96-h LC50s), McGrath and Di Toro (2009) applied the model to published data on chronic toxicity of PAH using an average acute to chronic ratio, and concluded that it could also predict concentrations chronically toxic to fish embryos. However, there were too few chronic toxicity data published to truly validate the model with statistical strength, and the model includes a wide array of assumptions.

The need for validation reflects different toxic effects of different PAHs. Phenanthrene, fluorene, and dibenzothiophene exert direct toxic effects on

the development of the heart, whereas chrysene was comparatively non-toxic, and the cardiac toxicity of pyrene was aryl hydrocarbon receptor (AhR)-dependent, meaning that enzymatic transformation by cytochrome P4501A (CYP1A) was a necessary step in activation to toxic forms. Moreover, cardiac toxicity was responsible for edema and other deformities seen in fish embryos (Incardona et al. 2004, 2005). For some alkyl PAH as well, metabolism by CYP1A enzymes plays a role. These enzymes add oxygen to the double bonds of the aromatic rings of PAH, as the first step in their degradation or excretion. Experiments with compounds that inhibit CYP1A metabolism of PAH suggested that specific metabolites might be more toxic to fish embryos than the parent compounds (Hodson et al. 2007b; Scott and Hodson 2008; Scott et al. 2009). Subsequent tests of hydroxylated derivatives of 1-methylphananthrene demonstrated that some were four times more toxic than the parent compound, and that toxicity was not due to narcosis (Fallahtafti et al. 2011).

This next section reviews the potential effects of oil on fish to indicate how different life stages might be affected by oil toxicity.

Embryotoxicity

Embryo toxicity can be caused by light and medium crude oils (McIntosh et al. 2010; Wu et al. 2012), crude and refined oils such as diesel (Schein et al. 2009), and by heavy fuel oils (Martin 2011; Incardona et al., 2012a,b), bituminous sediments (Colavecchia et al. 2004), and extracts of solids from oil sands tailings ponds (Farwell et al. 2006). There is a large database of literature that describes how the chronic exposure of fish embryos to PAH causes embryonic malformations, by altering or stopping embryonic development, with effects on population level recruitment. The association of embryonic developmental toxicity with PAH in oil has been shown by tests of single PAH found in crude and refined oils (Incardona et al., 2004, 2005, 2006, 2011), effects-driven fractionation of crude oil (Hodson et al. 2007a), and statistical correlations between toxicity and the concentrations of PAH in test solutions (Carls et al. 1999; 2005).

The most embryotoxic PAHs are those with 3 to 5 rings, with or without alkyl side groups, and including the sulphur-containing dibenzothiophenes. They include PAHs that appear to act directly on cardiac development [e.g., phenanthrene, fluorene (Incardona et al. 2004, 2005)], by indirect effects due to enzymatic metabolism in fish tissues to reactive intermediates [e.g., pyrene (Incardona et al 2005), retene (Hodson et al. 2007b; Scott and Hodson 2008; Scott et al. 2009)], or indirectly by photo-activation of PAH to reactive intermediates in tissues [e.g., anthracene (Oris and Giesy 1987); retene (Vehniainen et al. 2003); complex mixtures of PAHs (Farwell et al. 2006, Incardona et al 2012a,b)]. As indicated above, PAHs comprise up to 6% of oil by weight, with alkyl PAH the predominant form (85 to 95% of

total PAHs). Therefore, embryonic development of fish is an important target of oil toxicity. Some of the products of PAH metabolism are phenol derivatives which are embryotoxic, but also potential reproductive endocrine disruptors. Thus, the reproductive capacity of sexually maturing fish could also be affected by oil.

Embryonic malformations may occur directly as a result of PAH exposure, or may be caused by a process including induction of CYP1A enzymes, CYP1A metabolism of PAHs to toxic metabolites, and disruption of normal development. Malformations include hemorrhaging, edemas, cardiac dysfunction, craniofacial, spinal, and ocular deformities, fin erosion, reduced growth, and increased mortality rates (Birtwell and McAllister 2002; Brinkworth et al. 2003; Carls et al. 1999; Carls and Rice 2007; Colavecchia et al. 2004, 2006; Incardona et al. 2004, 2009; Fallahtafti et al. 2011; Marty et al. 1997a,b; Turcotte et al. 2011). Most if not all of these deformities result from cardiac toxicity, leading to reduced circulation and subsequent effects (Incardona et al., 2011). The relative extent of each response varies among different species of fish and the specific PAH tested. Where there is photo-modification of the PAHs accumulated in embryo tissues, acute embryo mortality is associated with tissue necrosis rather than cardiac toxicity, implying a different mode of action (Incardona et al., 2012a,b). The effects of PAHs may range in severity from a complete cessation of embryo development and death before feeding begins, to minor reductions in growth. Embryos affected by petroleum-induced deformities die because of circulatory failure or they fail to develop the structures needed for swimming and feeding and ultimately starve to death. Mortality rates increase with increasing frequency and severity of deformities, and after an oil spill, any impaired embryos would likely be consumed by predators (Heintz et al. 2000).

The overall consequence of chronic oil toxicity is a reduction in recruitment, i.e., a reduction in the numbers of young that grow to commercially valuable sizes or that survive to reproduce. However, even fish that survive exposure and appear completely normal may be adversely affected. Pink salmon that survived exposure as embryos to ANSC were tagged and released along with control salmon. Although the oil-exposed fish appeared normal, they returned to spawn in far fewer numbers than control fish, suggesting a sublethal effect on capacity to swim, feed, escape predation or migrate back to their release point (Heintz et al. 2000). A substantial reduction in swimming capacity and altered heart morphology of adult zebrafish that were exposed to sublethal levels of PAHs as embryos provides strong evidence for that hypothesis (Hicken et al. 2011).

The effects of an oil spill on fish reproduction will be severe due to contamination of the sediments used for spawning and incubation of eggs, embryos, and larvae. Oil entrained and stranded in bed

sediments will render spawning shoals toxic to fish embryos, and potentially reduce oxygen concentrations to harmful levels if there is extensive biodegradation of oil in sediments.

The effects on fish reproduction of sediment contamination will last for months to years, reflecting the residence time of oil in spawning shoals. Because most fish species in the Fraser River are sediment spawners (surface or sub-surface), all of those species may show multiple weak or missing year classes following an oil spill, depending on the extent to which their primary spawning areas are affected by the spill.

There is a potential for reductions in returns of migrating adult salmon because of the delayed effects of exposure to oil as eggs and embryos that would reduce survival during growth and maturation at sea.

Effects on sexual maturation and reproduction

PAHs can affect reproduction in aquatic organisms (Hall and Oris 1981; Casillas et al. 1991; Johnson et al. 1998; Thomas and Budiantara 1995), specifically by effects on ovarian growth and spawning success, and by mimicking or altering the concentrations of hormones such as estradiol. The exposure of sexually maturing fish to crude oil can impair physiological function, e.g., by disrupting the regulation of reproductive hormones (Truscott et al. 1983). Sol et al. (2000) estimated the extent of reproductive impacts (hormone concentrations, fecundity, spawning behavior, gamete production, fertilization success) in three fish species exposed to oil from the Exxon Valdez oil spill. Because no measurements were made in the year of the spill, they projected back from the results of sampling in subsequent years, and predicted that the spill had caused a significant decline in the estradiol concentrations of mature Dolly Varden trout. If reproductive impairment of sexually maturing or mature fish only followed chronic exposure, it would be of concern primarily for resident species such as white sturgeon and rainbow trout that might be exposed to an on-going release of oil from sediments contaminated by a spill.

The impacts of oil exposure on the sexual maturation of fish and on their ability to reproduce are not well characterized. If impacts do occur, they would be most severe in fish species that are year-round residents of the Fraser River, including rainbow trout and white sturgeon.

Effects on immune function and disease

PAHs, including many PAHs found in petroleum, are well known immunosuppressants in fish and other vertebrates (Khan 1991, 1995; Faisal and Huggett 1993; Payne and Fancey 1989; Tuvikene 1995; Kiceniuk and

Khan 1987; Moles and Norcross 1998; Carls et al. 1999; Reynaud and Deschaux 2006; Kennedy and Farrell 2008; Bado-Nilles et al. 2009; Hogan et al. 2010). Visible signs of disease in fish, such as skin ulceration, fin rot, and tumors can be direct consequences of exposure to PAHs. In some studies, immunosuppression by PAHs has been linked to increased mortality following a subsequent exposure to pathogens (Khan 1991; Arkoosh et al. 2000, 2001; Bravo et al. 2011). Thus, while exposure to PAHs may not always directly result in measurably increased mortality, the combination of PAH exposure and the presence of naturally occurring pathogens have the potential to reduce survival of a wide range of fish species.

While the potential may be real, it is often difficult to link cause-and-effect with certainty in studies of wild fish populations. For example, Pacific herring (*Clupea pallasi*) populations crashed in 1993, more than 3 years after the 1989 Exxon Valdez oil spill (EVOS). Some authors attributed the crash to the combined effects of oil toxicity to herring embryos hatched in 1989 with the natural occurrence of viral hemorrhagic septicemia virus (VHS; Hose and Brown 1998). The opposite case was made that the problem was poor fish condition because of over-population, which would also interact with diseases such as VHS, unconnected to oil exposure (Pearson et al. 1999). Proof-of-concept laboratory tests were provided by Carls et al. (1998), who demonstrated an interactive effect of oil exposure with VHS in lab tests, when wild-caught adult herring developed acute signs of VHS during exposure to low concentrations of oil in water (4-13 µg/L of total PAHs). Control fish from the same wild stock and held under the same conditions were virtually free of signs of disease. Their hypothesis was that herring were carriers of the virus but asymptomatic until the stress of oil exposure reduced their immunity. While this is not proof that the EVOS spill directly caused a disease outbreak in herring, these studies point to the likelihood that the EVOS was a contributing factor, without which the outbreak of disease and population crash would not have occurred. Overall, these studies underline the risk of adding oil exposure on top of natural environmental stressors.

Exposure of fish to oil will reduce their immunity to natural pathogens. Combined with the stress of exposure to oil and stress due to natural factors such as food limitation, spawning migrations, saltwater and freshwater adaptation, and temperature change, impaired immunity may trigger outbreaks of disease that could decimate fish stocks.

Genotoxicity

While embryo toxicity is associated with 3-5-ringed PAHs, some high molecular weight PAHs, such as benzo(*a*)pyrene (BaP), have additional toxic effects. Metabolism (oxygenation) of these PAHs by CYP1A enzymes

creates highly reactive metabolites, some of which can react to form covalent bonds with fatty acids, proteins, DNA, and other macromolecules. These 'adducts' to macromolecules can produce cellular damage (e.g., loss of membrane integrity) and genotoxicity, leading to mutagenesis, teratogenesis, and carcinogenesis (Tuvikene 1995), so that mutations, deformities, and cancers might be expected in fish exposed to oil.

There are a variety of PAHs identified as potentially carcinogenic by the USEPA (1993), including: benz[a]anthracene; benzo[a]pyrenes; benzo[b]fluoranthene; benzo[j]fluoranthene; benzo[k]fluoranthene; chrysene; 7, 12-dimethylbenz[a]anthracene, indeno[1,2,3-cd]pyrenes, and 5-methylchrysene; many of these are found in oil. However, the majority of carcinogenic PAHs are most commonly associated with combustion (pyrogenic) PAHs, and together, constitute only a few percent of the total PAHs from oil. Hence, liver, skin, and bile duct tumors associated with PAH carcinogenicity are most often found in benthic fish species such as brown bullheads (*Ictalurus nebulosus*) and English sole (*Parophrys vetulus*) that are in continuous contact with sediments contaminated by pyrogenic PAHs from heavy industry or urban runoff (e.g., Baumann et al. 1996, Johnson et al. 2008). It is also of interest to note that PAHs derived from aluminum smelter operations are linked to increases in prevalence of liver cancer in English sole in Kitimat Arm, BC (Johnson et al. 2015). While there are reports of genotoxicity in fish after oil spills (e.g., Hose et al. 1996; Hose and Brown 1998; Aas et al. 2000), no reports were found that linked cancer in fish to an oil spill, perhaps because periods for these delayed effects are in months to years.

There is a risk of an elevated prevalence of cancer in fish following exposure to some PAH found in oil. The size of this risk is difficult to judge because of the long latency period between exposure and the appearance of tumors.

Physiological biomarkers

Physiological biomarkers are useful indicators of exposure and effect resulting from exposure to petroleum products and its toxic constituents, namely PAHs and alkyl PAHs. While there are many different definitions of biomarkers, a biomarker of exposure is usefully defined as "the detection and measurement of an exogenous substance or its metabolite or the product of an interaction between a xenobiotic agent and some target molecule or cell that is measured in a compartment within an organism" (Douben 2003). Similarly, a useful definition of a biomarker of effect is "a measurable biochemical, physiological, or other alterations within tissues or body fluids of an organism that can be recognized as associated with an established or possible health impairment or disease" (Douben 2003).

There are many biomarkers that can be used in relation to oil exposure and effects, including but not limited to:

• Increased synthesis (induction) of cytochrome P4501A (CYP1A) proteins as indicated by *cyp1a* mRNA concentration, CYP1A protein concentration, and ethoxyresorufin-*O*-deethylase (EROD) or aryl hydrocarbon hydroxylase (AHH) activity (e.g., CYP1A catalytic activity; Collier et al. 1993, 1995, 1996; Ramachandran et al. 2004).

• Oxidative stress as indicated by increased activity of antioxidant enzymes (Balk et al. 2011), decreased concentrations of antioxidants (e.g., Vitamin E; Bauder et al. 2005), or increased concentrations of the products of oxidative stress (fatty acid methyl esters; Balk et al. 2011).

• PAH metabolites in bile and/or urine (glucuronic acid conjugates of PAHs; Aas et al. 2000; Collier and Varanasi 1991; Collier et al. 1996; Balk et al. 2011).

• Genotoxicity (assays of DNA fragmentation and adducts; Hose and Brown 1998; Aas et al. 2000; Collier et al. 1993; Amat et al. 2006).

In natural fish populations located near known sources of oil, biomarker responses have been measured that indicate exposure to oil has occurred and that there is an increased likelihood of adverse effects. For example, there is a significant background contamination of the North Sea by produced water, drill cuttings, muds, and accidental spills from offshore oil production (Balk et al. 2011). In haddock (*Melanogrammus aeglefinus*) and Atlantic cod (*Gadus morhua*) exposure to oil drilling muds (including oil), PAH exposure and potential effects were assessed using the induction of biotransformation enzymes, oxidative stress, altered fatty acid composition, and genotoxicity (Balk et al. 2011). Chronic exposure to environmental pollution has also been demonstrated in Atlantic cod using tissue concentrations of PAHs, metabolites of PAH in bile, and DNA adducts (Aas et al. 2000).

Because these biomarkers respond rapidly to oil exposure, and because they can indicate both exposure and effects, they are very useful indicators of contamination of fish by petroleum products, the potential ecological effects of spills, and cause-effect relationships that facilitate assessments of injuries to natural resources following oil spills. They are also valuable tools to assess the extent, degree, and duration of exposure to spilled oil. Biomarkers can also provide insight into sources of spilled oil (e.g., new spills versus residual oil; location of oil), and the route of exposure (e.g., sediments versus water column).

Within the context of the proposed Trans Mountain pipeline and terminal expansion, the ability to use and interpret biomarker responses would be

greatest if there were pre-construction surveys to establish baselines. If this were the case, biomarkers would be one of the most valuable tools available for assessing the impacts of future oil spills on fisheries.

There are many sensitive biochemical and physiological responses to oil of fish that survive the initial spill, or are subject to periodic or continuous exposure to oil following a spill. These responses are useful indicators of oil exposure and toxicity for monitoring and assessing the impacts of an oil spill. Baseline surveys of biomarker responses in fish along the pipeline and marine transportation route would be invaluable in assessing the impacts from any resulting oil spills.

Effects on behavior

Fish behavior can make individuals more or less susceptible to contaminants, and disruption of behavioral processes can be detrimental to population survival. Avoidance is a well-documented response to chemicals in water, but there are only preliminary indications that fish can avoid oil (Maynard and Weber 1981). In the Fraser River, there are a large number of tributaries with suitable habitat for fish reproduction and development. This habitat may provide fish some refuge in uncontaminated habitat, should a spill occur in one tributary or in the main stem. However, given the rate at which spilled oil would spread across water and downstream with the flow of the river, and the propensity of low molecular weight compounds that cause acute lethality to partition from oil to water, fish in the spill zone might not have sufficient time to avoid the spill, even if they could.

The early developmental stages of fish are also highly susceptible to exposure because eggs deposited in or on bottom substrates and newly hatched embryos are incapable of avoiding contaminated water. As well, benthic species (sculpins, Pacific lamprey, white sturgeon) that live in contact with the sediments or feed directly on benthic invertebrates would be exposed to oil through preferential contact with substrate and through ingestion of contaminated prey. This is particularly true if they are particulate feeders like Pacific lamprey ammocoetes, which are the larval forms that live buried in mud and feed on microoganisms—these animals would have a high propensity to ingest fine droplets of oil.

The potential for avoidance of spilled oil (Weber et al. 1981) can also be negative if it deters salmonids, lamprey, sturgeon, and eulachon from completing their spawning migrations. While adult Chinook salmon that survived an oil exposure were able to migrate to their natal stream (Brannon et al. 1986b), they may avoid oil spills that block access to spawning habitat. If they spawn only once in their lifetime, their reproductive potential could be lost due to avoidance, and this could be a potential issue even with repeat spawners. For example, fish are known to avoid elevated concentrations of copper and zinc. In the Miramichi River (New Brunswick)

watershed, sexually mature Atlantic salmon (*Salmo salar*) were tagged as they migrated upstream through a counting fence over a four-year period. However, each year these same fish were counted moving downstream through the fence without spawning when concentrations of copper and zinc increased due to an upstream mine (Saunders and Sprague 1967). In each year's run, only a small percentage of salmon re-ascended the river when concentrations declined, and an estimated 62% failed to complete their spawning migration.

Other behavioral impacts could include a reduction in feeding, particularly if the food source is contaminated with oil, and capacity to capture prey, avoid predators, or complete pre-spawning behaviors, particularly for fish showing deformities or fin erosion.

The behavioral responses of fish to oil exposure, and the impacts of oil on normal behaviors such as migration, feeding, nest building and mating, are not well characterized. Evidence that fish can avoid oil is positive in that fish could escape areas rendered acutely toxic and find refuge in clean tributaries, but negative if reproductive potential is lost when fish do not have access to uncontaminated spawning shoals because they avoid intervening areas of contamination. Avoidance is not an option for early life stages of fish.

Factors that affect toxicity

Oil type

Concentrations of total PAHs (the sum of all un-substituted and alkyl-substituted homologs) vary considerably among crude oils, with concentrations up to 6.0% in heavier oils (Hollebone et al. 2011). Thus, if PAHs are the cause of chronic toxicity, concentrations of whole oil that cause embryo toxicity should vary in proportion to their total PAH content, assuming that exposure methods are consistent. Wu et al (2012) found that the toxicity to trout embryos of four chemically dispersed oils varied only two-fold when expressed as the dilution of the chemically enhanced water accommodated fraction (% v/v). However, the concentrations of PAHs in whole oil varied 5-fold, indicating that the lightest oil (Scotian Light) was more toxic than predicted. When toxicity was compared among the four oils using water borne concentrations of PAHs estimated from measured PAH concentrations in 0.1% (v/v) CEWAF, and the EC50s expressed as dilutions (% v/v), there was little difference in toxicity among the oils. In other words, no matter what the type of oil, the concentrations of PAHs that were toxic were virtually constant. The higher than expected toxicity of Scotian Light when expressed on a dilution basis may have been due to a more efficient chemical dispersion due to its lower viscosity and density compared to the heavier oils. In toxicity tests of heavy fuel oils coated on gravel in simulated

spawning shoals, HFO 6303 was more nearly 2.5-fold more toxic than MESA crude oil. In this case, the HFO 6303 contained nearly 2.5-fold more PAHs than MESA (Martin 2011). In this case, toxicity was predicted by total PAH concentration on gravel because the concentration gradient from oil to gravel would control the PAH concentrations in test solutions.

Compared to the crude oils tested by Wu et al (2012) and Martin (2011), the diluted bitumen and synthetic crude oils that will be transported through an expanded pipeline contain about 2-fold lower concentrations of total PAHs compared to medium crudes (Yang et al. 2011). However, if the primary factor that determines toxicity is the rate at which PAHs are released from oil rather than their absolute concentrations, toxicity will be governed primarily by the issues of environmental fate and distribution discussed earlier.

The type of oil spilled will determine the amount and type of PAHs that can diffuse into water. While toxicity should increase with PAH concentrations in spilled oil, the amount that dissolves into water may be determined more by the physical characteristics of oil and how easily it forms droplets.

Ultimately, it is the concentration of total PAHs in water that determines toxicity, and existing data do not demonstrate wide differences among oil in the toxicity of their PAHs.

Weathering

Weathered oil retains the less soluble but more persistent PAHs that exert their effects in the parts per billion concentration range (Carls et al. 1999, 2000; Heintz et al. 2000; Middaugh et al. 2002; Albers and Loughlin 2003; Payne et al. 2003; Heintz 2007; Logan 2007). The LMW components are lost due to evaporation, partitioning into water, biodegradation, and photodegradation, processes that quickly reduce the concentrations of LMW compounds in spilled oil; these loss rates decrease with an increasing degree of weathering and with increasing molecular weight of the residual compounds (McAuliffe 1977). As a consequence, the acute lethality of oil also decreases with weathering, so that fish kills occur less frequently under conditions that accelerate weathering. Thus, if weathering of spilled oil occurs without acute lethality to fish in the first 24 hours, the overall impact of the oil will be greatly reduced on juveniles and adult fish.

With weathering, the residual heavy components, such as 3- to 6-ringed PAHs, waxes, resins, and asphaltenes become progressively enriched as the total volume of oil decreases. This causes an apparent increase in chronic toxicity to early life stages of fish, expressed as the µg/L of residual oil (Carls et al. 1999). For chronic embryo toxicity, Heintz et al. (1999) observed that highly weathered ANSC oil coated on gravel in desorption

columns was more toxic to pink salmon embryos than fresh oil due to a higher proportion of HMW PAHs that are slower to degrade. The increased toxicity of the residue was likely due to the increased concentration gradient between water and weathered oil, compared to the gradient with unweathered oil.

The extent and nature of weathering will determine which constituents remain in residual oil in slicks and stranded on shorelines or in bed sediments.

The apparent toxicity of residual oil increases with weathering because the residual toxic components (e.g., PAHs) increase in concentration as the low molecular weight components are removed by weathering. However, the toxicity of the residual PAHs does not change until they, too, are degraded by weathering, a process that may take many years.

Photomodification of PAH

The UV wavelengths of sunlight can photomodify many of the components of oil, especially those with double bonds (photolysis). While double bonds can absorb energy, some are broken by the increased energy and the derivatives formed are more polar, more water soluble, and less bioavailable and toxic to aquatic species. However, for some hydrocarbons, toxicity may be enhanced by exposure to UV-A and UV-B radiation. For the PAHs anthracene, fluoranthene, and pyrene, UV photomodification creates oxygenated derivatives, such as quinones and anthroquinones, which are more water soluble but also more reactive and much more toxic than the parent compound. The products of these reactions will depend on the structure of each PAH and on its specific light absorption characteristics (Diamond et al 2000).

In nearly transparent fish embryos, photomodification of PAHs accumulated by the embryos occurs in tissues, where the photo-products and associated reactive oxygen species cause oxidative stress, cell death, and acute mortality (Arfsten et al. 1996; Duesterloh et al. 2002). The toxic photoproducts formed *in situ* can react directly with cellular components (Hatlen et al. 2010) causing rapid tissue necrosis. In Pacific herring embryos exposed to a water-accommodated fraction (WAF) of ANSC oil for 96 h and then to weak sunlight, toxicity increased by up to 50-fold in comparison to embryos exposed to WAF only (Barron et al. 2003). Similarly, juvenile tidewater silversides (*Menidia beryllina*) were unaffected by exposure to UV alone, but the acute lethality of the WAF of a weathered Californian oil was enhanced five times by UV irradiance during the test (Little et al. 2000). The same result was found for larval whitefish (*C. lavaretus*) exposed to UV-B and to retene; neither alone was lethal, but the two together caused acute lethality (Vehnianen et al. 2003). Photosensitive PAHs exhibit enhanced toxicity to fish at concentrations as low as 1.0 µg/L (Dong et al. 2000; Little

et al. 2000; Barron and Ka'aihue 2001). Recently, Pacific herring were demonstrated to be exquisitely sensitive to spilled bunker oil when exposed to sunlight, after the Cosco Busan oil spill San Francisco Bay (Incardona et al. 2012a,b).

Overall, the risk of increased toxicity of PAHs due to photomodification will be site-specific and a function of turbidity, dissolved organic carbon (affecting water color and light quenching), the time of year (solar radiation intensity, shading), and the fish species present. For each species, the location of spawning shoals and the extent of protective pigmentation in developing embryos will determine the exposure to UV-A and UV-B (reviewed in Little et al. 2000).

The risk of toxicity of some PAHs (e.g. anthracene) accumulated by fish embryos will be greatly increased if they are exposed to light when they emerge from gravel as larvae. The photomodification of these PAHs in the tissues of larvae will cause acute lethality. Hence, exposure of embryos to oil in contaminated spawning shoals that appears non-toxic may in fact increase the risk of mortality at emergence.

Cumulative effects

The primary focus of this section on toxicity has been on the direct effects of an oil spill on different life stages of fish species resident in the Fraser River. However, the overall ecological impacts of an oil spill on the river more properly represent the 'effects of effects'. The ultimate issue is how toxicity to individuals of one or more species translates into subsequent effects on populations of fish (abundance, growth rate, size and age distribution, size and age at first maturity, fecundity), communities (fish species present or absent; diversity; indicator species), ecosystems (productivity, biomass, energy flow, resilience, interactions with terrestrial and marine systems), and human use of fish (subsistence, sports, and commercial fishery production; fisheries closures; tainting, quality of a fishing experience). Many of these responses can only be guessed at or modeled, and they will not be addressed here. Due to the site-specific nature of each river ecosystem, the nature and extent of those impacts on the Fraser River may be partially predicted by experience elsewhere (e.g., the Pine River, BC), but will only become evident after an actual oil spill.

The difficulty in predicting actual impacts is also a function of cumulative effects, i.e. the potential responses of the fish community of the Fraser River to the sum of all the different natural and anthropogenic stressors that act on the river as a whole and on each fish species. Cumulative effects can have unexpected and serious effects on aquatic ecosystems, a concern described by Arkoosh and Collier (2002) and Jacobson et al. (2003). For example, heavy fuel oil #6 (Bunker C) was spilled into Lake Winona, MN

over the winter, where it pooled on the bottom in depressions. The spill was undetected, and there were no observable effects until a massive spring die-off of bluegill sunfish (*Lepomis macrochirus*), associated with the combined stressors of oil exposure, rapid temperature shifts, spring spawning, and the loss of condition over the winter (Fremling 1981). Therefore, effects on fish following oil spills may not coincide exactly with the spill, and may vary in intensity according to each species' level of stress associated with annual cycles of migration, reproduction, feeding, and growth.

In the Fraser River system, cumulative effects could arise from deforestation and its impacts on the severity of flood events, on bank erosion, and on the suspended sediment load of the river, which would affect the suitability of river habitat for fish as well as the fate and distribution of oil during flood conditions. Deforestation and the removal of contaminated wood debris from the river during spill clean-up could also have major effects on river re-structuring during floods and the distribution or redistribution of contaminated sediments. Exploitation of fish stocks by sports and subsistence fisheries could interact with the population-level effects of oil toxicity to aggravate impacts on fish productivity.

While these ideas are extremely difficult to test in natural ecosystems, interactive stressors have been suggested as an underlying cause of the collapse of Pacific herring in Prince William Sound following the Exxon Valdez oil spill (Carls et al. 1998). The health and performance of each fish represents the integrated effects of all environmental factors, but the consequence of exposure of fish to oil has been predicted to be a reduction in survival (Heintz 2007), despite the difficulty of separating such effects in the wild (Thorne and Thomas 2008). Thus, the impacts of oil spills into the Fraser River, its estuary, or marine environments going out to the open ocean through the Salish Sea may not be easily understood or managed when they are combined with other environmental factors and stresses.

The toxicity of spilled oil to fish in the Fraser River may interact with other environmental or anthropogenic stressors to cause unexpected effects, or an unexpected severity of effects. These stressors could include seasonal changes in temperature and the physiology of migration (saltwater and freshwater adaptation) and reproduction (sexual maturation, energy depletion), and changes in habitat quality associated with deforestation, road construction or other development.

The impact of a significant fish kill on all species and life stages of fish present in the river, combined with outbreaks of disease facilitated by increased stress, and followed by a prolonged period of reduced recruitment due to fewer spawners and toxic spawning shoals would

create a "one-two-three punch" that would reduce overall fisheries productivity in the river for years to decades.

Due to the site-specific (and spill-specific) nature of the river and of a future oil spill, the overall impact of a spill on the fish communities of the Fraser River can only be guessed at. There are too many uncertainties and unmeasured conditions to provide hard estimates of risks or effects. Thus, it is difficult to predict exactly what effects will occur, when they will occur, and where they will occur, but there is no reason to believe that they will not occur.

If eulachon and pink salmon experience multiple weak year classes, there will be significant impacts on stocks of pink salmon and eulachon, with the potential for local extirpation.

To understand the significance of cancer risks, and to take advantage of sensitive biomarkers of oil exposure and toxicity, the prevalence and severity of tumors and of biochemical and physiological responses of fish should be assessed before pipeline or terminal expansion and for up to ten years following a spill.

Fishery resources rendered unfit for human consumption

Fish and other seafood are considered to be one of the healthiest sources of protein in the human diet. Fish, especially marine fish, contain high levels of healthy fatty acids, known as omega-3 fatty acids, which are believed to be part of the health-giving capacity of seafood. The consumption of seafood has been strongly linked to improved heart health, improved mental acuity, improved neonatal health (especially brain development and function), improved immune function (i.e. better resistance to disease), and numerous other health benefits in humans (Dickhoff et al. 2007; Raatz et al. 2013; Wheeler et al. 2014). The indigenous people of British Columbia and Washington State have historically had some of the highest rates of fish and other seafood consumption, with estimated heritage fish consumption (FCR) rates for some groups of several hundred grams to over a kilogram per person per day; highest rates were estimated for tribes living along the Columbia River (Harper et al. 2002; Harper and Walker 2015). This is in stark contrast to non-native people living in North America, where FCRs of between 6.5 and 17.5 grams per day are used by the USEPA to determine risks from chemical contaminants (USEPA 2000, Donatuto and Harper 2008). For the SFN and PB, much of their fish consumption has been and still is in the form of sockeye salmon, which is a species that is very highly

enriched in omega-3 fatty acids. For these reasons, oil spill impacts in the Fraser River valley that could harm fish populations, as described above, are extremely significant and problematic for the members of the SFN and PB. However, even if an oil spill occurred, but did not directly or substantially harm the fish themselves, impacts to members of the SFN and PB would likely still be significant. This is because of the contamination of fish by constituents of the oil, as well as the perception and concern that fish and other seafood may have been exposed to oil.

Following oil spills, it is well documented that fisheries in oiled areas are closed, sometimes for extended periods of time (Yender et al. 2002). A notable and recent such event occurred following the Deepwater Horizon oil spill that lasted from April 20 to July 15, 2010. This spill resulted from an explosion and fire on the BP drilling platform Deepwater Horizon, where 11 workers were killed in the initial incident, over 200 million gallons of oil entered the waters of the northern Gulf of Mexico, and over 2 million gallons of dispersants were applied to try and keep oil from reaching the shorelines. This oil spill led to a closure of fisheries within 2 weeks of the spill beginning, and as the magnitude of the spill increased, the federal fisheries closures expanded to over 200,000 square kilometers. At the same time states were closing their inshore waters, and multiple agencies began testing seafood to see if it was safe for consumption. Figure 2 (which is taken from Gohlke et al. 2011) shows a timeline of some of the closings, openings, re-closings, and re-openings of fisheries that happened in the several months following the DWH spill.

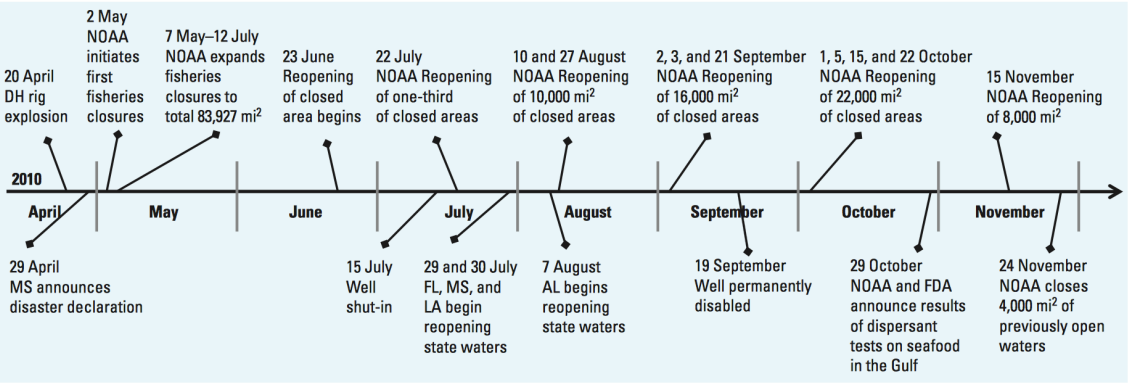

Figure 1. Timeline of fisheries closures and reopenings in the Gulf of Mexico due to the DH blowout in 2010. Abbreviations: AL, Alabama; MS, Mississippi; LA, Louisiana; FL, Florida. Data from NOAA (2010b) and FDA (2010). On 13 January 2011, 4,213 mi² of federal waters around the well and parts of area 12 in LA State coastal waters remained closed. On 1 February 2011, NOAA repoened the federal waters that had been reclosed to royal red shrimp fishing. On 19 April 2011, NOAA reopened all remaining federal waters. Approximately 1.5% of LA coastal waters remain closed.

Environmental Health Perspectives • VOLUME 119 | NUMBER 8 | August 2011

Figure 2. A depiction of fisheries closures and openings in the waters of the northern Gulf of Mexico following the Deepwater Horizon oil spill in 2010 (from Gohlke et al. 2011).

This serves to demonstrate that the issue of fisheries closures following an oil spill is not a simple one. The reasons for this are varied, and will differ

between spills. Following a major freshwater or marine spill associated with the Trans Mountain expansion project, fisheries closures are almost certain to occur. Factors that will affect the duration of any closures include the entrainment of spilled oil into river or marine sediments, or stranding along shorelines in tidally influenced areas. As described above, these are both highly likely following a release of diluted bitumen. The subsequent re-release of oil following storm events, or changes in river flows, or extreme tidal cycles, would add uncertainty as to whether exposure of aquatic animals might re-occur episodically. This will generally lead to increased duration of fisheries closures.

Another factor that would cause major uncertainty about seafood safety following a major spill is the lack of baseline data on chemicals that might be present in fish and shellfish from the Fraser River before a spill occurred. Without such information, it will not be possible to determine if and when levels of increased contamination have completely disappeared. The need for baseline monitoring of seafood safety was highlighted in Dickhoff et al (2007).

The chemical complexity of bitumen and the diluents that will be used to formulate the diluted bitumen products that will be transported also makes issues around seafood safety complex. Traditional risk assessments about seafood safety following an oil spill only focus on a small set of compounds, usually non-alkylated PAHs, and usually the 16 priority PAHs listed by the USEPA (Wickliffe et al. 2014). However as described above, crude oils, including diluted bitumen, contain thousands of complex compounds, including a wide array of alkylated compounds that are extremely difficult to analyze, and for which there is no toxicity data. Thus, risk assessments following an oil spill are generally not satisfactory to affected consumers or producers (fishers) of seafood. These problems were recently well-described (Wilson et al., 2015). Essentially, communities with high rates of fish consumption in areas affected by an oil spill are not included when risks to human health are assessed, because of the absence of regulatory information about relevant chemicals.

Summary of possible impacts to Shxw'ōwhámel First Nation and Peters Band utilization of fish

Effects of a pipeline rupture and spill into the Fraser River

A pipeline spill into the Fraser River or its tributaries could cause large scale injury to fisheries in the region. It is important to note that the worst case scenario outlined in the Trans Mountain application materials (1300 m^3) could be easily exceeded if leak detection systems are not operating correctly, if a landslide were to destroy one or more pipeline valves, and if

human error does not occur. One needs only to consider the 2010 Kalamazoo River spill to realize the potential for human error to greatly magnify the size and impact of an oil spill.

Once a major spill of diluted bitumen reaches the water, fish kills are likely to occur immediately. While the acutely toxic effects of spilled oil are expected to only last for s few days, persistent oil in the environment (such as from stranding on the shoreline or becoming entrained in sediments) will lead to continuing contamination of the environment. Continuing exposure of juvenile and adult fish to components of crude oils that are present at less than acutely lethal levels will still likely cause harm. Such exposures have been demonstrated to lead to a variety of effects, including reduced reproductive function, reduced disease resistance, increased damage to DNA, and potentially the development of cancer at some period of time after the spill. If eggs or larvae of bottom spawning fish such as salmon and sturgeon are present, the potential for catastrophic impacts to fisheries increases. Developing eggs and larvae are extremely sensitive to exposure to petroleum components, and can be readily killed in large numbers following a spill. Even if eggs and larvae survive a very low level of exposure, the juveniles and adults that grow from those exposed embryos may show reduced fitness, in terms of heart structure and ability to swim. Thus in the case of salmon, there is a potential for a failure of fish to return from the ocean, if they were exposed as eggs and larvae.

In addition to the direct impacts on fish, a major oil spill will also affect the ability and willingness of people to consume fish after an oil spill. Authorities generally close fisheries in the vicinity of any spill, and it is difficult to know when to re-open a closed fishery. Especially in the absence of pre-spill information on levels of contaminants in fish, it is hard to judge when contamination drops to pre-spill levels. Thus some people may never regain confidence in the safety of fish following an oil spill, especially an oil spill that persists in the environment. Because fish is a healthy component of the human food supply, these effects on seafood safety can directly affect the health of the members of SFN and PB.

Effects of a marine oil spill in waters near Westridge Marine Terminal or in the Salish Sea

A marine oil spill could affect marine species in all the same ways that a freshwater spill could affect freshwater species. Also, similar to the case for scenarios of freshwater spills, a marine spill could be significantly larger than the scenarios imagined by Trans Mountain in the application materials. For example, if a loaded tanker were to suffer a catastrophic failure while it was underway after leaving the Westridge Marine Terminal, the amounts of oil that might be released into the marine waters of the Salish Sea could be substantially higher than imagined by Trans Mountain. In their application

materials, Trans Mountain assumes a worst case spill scenario from a partially loaded Aframax tanker of 105,000 barrels of oil, whereas the capacity of an Aframax tanker can be over 800,000 barrels.

An added impact from a marine spill in the Salish Sea would be to potentially contaminate, or at least raise questions about contamination of, any fish migrating up the Fraser River. This could greatly diminish or eliminate the ability or the desire of members of the SFN and PB to harvest salmon, lamprey, and eulachon from the Fraser River, even though a spill might not have occurred in the Fraser River. As discussed above in the section on human consumption of contaminated seafood, merely the perception of contamination can greatly affect fish consumption, with resulting implications for human health.

References

Aas E, Baussant T, Balk L, Liewenborg B, Andersen OK. 2000. PAH metabolites in bile, cytochrome P4501A and DNA adducts as environmental risk parameters for chronic oil exposure: a laboratory experiment with Atlantic cod. Aquat Toxicol 51:241-258.

Albers PH, Loughlin TR. 2003. Effects of PAHs on marine birds, mammals and reptiles. In PAHs: An Ecotoxicological Perspective (ed PET Douben), John Wiley & Sons, Ltd, Chichester, UK.

Amat A, Burgeot T, Castegnaro M, Pfohl-Leszkowicz A. 2006. DNA adducts in fish following an oil spill exposure. Environ Chem Lett 4:93-99.

AMEC Earth & Environmental Limited. 2001. Environmental assessment of the Pembina pipeline oil spill to the pine river near Chetwnd, BC: Volume 1 - Sediment quality. Submitted to: District of Chetwynd, Chetwynd, BC, AMEC Earth & Environmental Limited, Bumaby, BC, 25 May 2001.

Arfsten, DP, Schaeffer DJ, Mulveny DC. 1996. The effects of near ultraviolet radiation on the toxic effects of Polycyclic Aromatic Hydrocarbons in animals and plants: A review. Ecotox Environ Safe 33: 1-24.

Arkoosh MR, Casillas E, Clemons E, Huffman P, Kagley N, Collier TK, Stein JE. 2001. Increased susceptibility of juvenile chinook salmon (*Oncorhynchus tshawytscha*) to vibriosis after exposure to chlorinated and aromatic compounds found in contaminated rban estuaries. J Aquat Anim Health 13:257-268.

Arkoosh MR, Casillas E, Clemons E, Huffman P, Kagley A, Collier TK, et al. 2000. Increased susceptibility of juvenile Chinook salon to infectious disease after exposure to chlorinated and aromatic compounds found in contaminated urban estuaries. Mar Environ Res 50: 470-471.

Arkoosh MR, Collier TK. 2002. Ecological risk assessment paradigm for salmon: Analyzing immune function to evaluate risk. Hum Ecol Risk Assess 8:265-276.

Bado-Nilles A, Quentel C, Thomas-Guyon H, Le Floch S. 2009. Effects of two oils and 16 pure polycyclic aromatic hydrocarbons on plasmatic immune parameters in the European sea bass, *Dicentrarchus labrax*. Toxicol in Vitro 23: 235-241.

Balk L, Hylland K, Hansson T, Berntssen MHG, Beyer J, Jonsson G, Melbye A, Grung M, Torstensen BE, Borseth JF, Skarphedinsdottir H, Klungsoyr J. 2011. Biomarkers in natural fish populations indicate adverse biological effects of offshore oil production. PLoS ONE 6:1-10.

Bakker G. 2011. Diluted Bitumen and Condensate Backgrounder. Report prepared for M.R. Gordon Associates, February 2011, 16 p.

Barron MG, Carls M, Short J, Rice S. 2003. Photoenhanced toxicity of aqueous phase and chemically dispersed weathered Alaska North Slope crude oil to Pacific herring eggs and larvae. Environ Toxicol Chem 22:650-660.

Barron MG, Ka'aihue L. 2001. Potential for photoenhanced toxicity of spilled oil in Prince William Sound and Gulf of Alaska waters. Mar Pollut Bull 43:86-92.

Bauder MB, Palace VP, Hodson PV. 2005. Is oxidative stress the mechanism of blue sac disease in retene-exposed trout larvae? Environ Toxicol Chem 24:694-702.

Baumann PC, Smith IR, Metcalfe CD. 1996. Linkages between chemical contaminants and tumors in benthic Great Lakes fish. J. Great Lakes Res 22:131-152.

Beamish RJ. 1980. Adult biology of the river lamprey (*Lampetra ayresi*) and the Pacific lamprey (*Lampetra tridentata*) from the Pacific coast of Canada. Can J Fish Aq Sci 37:1906-1923.

Beamish RJ, Levings CD. 1991. Abundance and freshwater migrations of the anadromous parasitic lamprey (*Lampetra tridentate*) in a tributary of the Fraser River, British Columbia. Can J Fish Aq Sci 48:1250-1263.

Belore R. 2010. Properties and fate of hydrocarbons associated with hypothetical spills at the Marine Terminal and in the Confined Channel Assessment Area Technical Data Report. Prepared for Northern Gateway Pipelines Inc. SL Ross Environmental Research Ltd, Ottawa, ON.

Birtwell IK and McAllister CD. 2002. Hydrocarbons and their effects on aquatic organisms in relation to offshore oil and gas exploration and oil well blowout scenarios in British Columbia, 1985. Can Tech Rep Fish Aquat Sci no. 2391, 60 p.

Brannon E, Setter A, Miller M, Brewer S, Winans G, Utter F, Carpenter L, Hershberger W. 1986a. Columbia River white sturgeon (*Acipenser transmontanus*) population genetics and early life history study. Final Report. January 1, 1986 -December 31, 1986. Project 83-316. Contract No. DE-AI79-84BP18952.

Brannon EL, Quinn TP, Whitman RP, Nevissi AE, Nakatani RE, McAuliffe CD. 1986b. Homing of adult chinook salmon after brief exposure to whole and dispersed crude oil. Trans Am Fish Soc 115:823-827.

Bravo CF, Curtis LR, Myers M, Meador JP, Johnson LL, Buzitis J, Collier TK, Morrow JD, Laetz CA, Arkoosh MR. 2011. Biomarker response and disease susceptibility in juvenile rainbow trout *Oncorhynchus mykiss* fed a high molecular weight PAH mixture. Environ Toxicol Chem 30: 1-11.

Brinkworth L, Hodson P, Tabash S, Lee P. 2003. CYP1A induction and blue sac disease in early developmental stages of rainbow trout (*Oncorhynchus mykiss*) exposed to retene. J Toxicol Environ Health 66:627-646.

Buffington JM, Tonina D. 2009. Hyporheic exchange in mountain rivers II: Effects of channel morphology on mechanics, scales, and rates of exchange. Geogr Comp 3:1038-1062.

Campagna JA, Miller KW, Forman SA. 2003. Mechanisms of actions of inhaled anesthetics. New Engl J Med 348:2110-2124.

Carls MG, Marty GD, Meyers TR, Thomas RE, Rice SD. 1998. Expression of viral hemorrhagic septicemia virus in pre-spawning pacific herring

(*Clupea pallasi*) exposed to weathered crude oil. Can J Fish Aquat Sci 55:2300-2309.

Carls MG, Rice S, Hose J. 1999. Sensitivity of fish embryos to weathered crude oil: Part I. low-level exposure during incubation causes malformations, genetic damage, and mortality in larval Pacific herring (*Clupea pallasi*). Environ Toxicol Chem 18:481-493.

Carls MG, Hose J, Thomas R, Rice S. 2000. Exposure of Pacific herring to weathered crude oil: Assessing effects on ova. Environ Toxicol Chem 19:1649-1659.

Carls MG, Heintz R, Marty G, Rice S. 2005. Cytochrome P4501A induction in oil-exposed pink salmon (*Oncorhynchus gorbuscha*) embryos predicts reduced survival potential. Mar Ecol-Prog Ser 301:253-265.

Carls MG, Rice SD. 2007. Fish embryo sensitivity and PAH toxicity. In Environmental Impact of Polynuclear Aromatic Hydrocarbons (ed C. Anyakora), Research Signpost, Kerala, India, pp. 159–190.

Casillas E, Misitano D, Johnson LL, Rhodes LD, Collier TK, Stein J, Christiansen T, Korsgaard B, Jespersen A. 1991. Inducibility of spawning and reproductive success of female English sole (*Parophrys vetulus*) from urban and nonurban areas of Puget Sound, Washington. Mar Environ Res 31:99-122.

Colavecchia MV, Backus SM, Hodson PV, Parrott JL. 2004. Toxicity of oil sands to early life stages of fathead minnows (*Pimephales promelas*). Environ Toxicol Chem 23:1709-1718.

Colavecchia MV, Hodson PV, Parrott JL. 2006. CYP1A induction and blue sac disease in early life stages of white suckers (*Catostomus commersoni*) exposed to oil sands. J Toxicol Env Health 69:967-994.

Collier TK, Varanasi U. 1991. Hepatic activities of xenobiotic metabolizing enzymes and biliary levels of xenobiotics in English sole *(Parophrys vetulus)* exposed to environmental contaminants. Arch Environ Contam Toxicol 20:462-473.

Collier TK, Stein JE, Goksøyr A, Myers MS, Gooch JW, Huggett RJ, and Varanasi U. 1993. Biomarkers of PAH exposure and effects in oyster toadfish (*Opsanis tau*) from the Elizabeth River, Virginia. *Environ Sci* 2:161-177.

Collier TK, Anulacion BF, Stein JE, Goksøyr A, Varanasi U. 1995. A field evaluation of cytochrome P4501A as a biomarker of contaminant exposure in three species of flatfish. *Environ Toxicol Chem* 14:154-162.

Collier TK, Krone CA, Krahn MM, Stein JE, Chan S-L, Varanasi U. 1996. Petroleum exposure and associated biochemical effects in subtidal fish after the *EXXON Valdez* oil spill. Am Fish Soc Symp 18:671-683.

Conte FS, Doroshov SI, Lutes PB, Strange EM. 1988. Hatchery manual for the white sturgeon *Acipenser transmontanus* Richardson with application to other North American Acipenseridae. University of California. Publication 3322.

Diamond SA, Mount DR, Burkhard LP, Ankley GT, Makynen EA, Leonard EN. 2000. Effect of irradiance spectra on the photoinduced toxicity of three polycyclic aromatic hydrocarbons. Environ Toxicol Chem 19:1389-1396.

Dickhoff WD, Collier TK, Varanasi U. 2007. The seafood dilemma: A way forward. *Guest Director's Line*. Fisheries 32:244-246.

Di Toro DM, McGrath JA, Hansen DJ. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. I. Water and tissue. Environ Sci Technol 19:1951-1970.

Di Toro DM, McGrath JA, Stubblefield WA. 2007. Predicting the toxicity of neat and weathered crude oil: toxic potential and the toxicity of saturated mixtures. Environ Toxicol Chem 26: 24-36.

Donatuto J, Harper BL. 2008. Issues in evaluating fish consumption rates for Native American tribes. Risk Anal 28:1497-1506.

Dong S, Hwang HM, Harrison C, Holloway L, Shi X, Yu H. 2000. UVA light-induced DNA cleavage by selected polycyclic aromatic hydrocarbons. Bull Environ Contam Toxicol 64:467-474.

Douben PET. 2003. PAHs: an ecotoxicological perspective. Volume 4 of Ecological & Environmental Toxicology series. John Wiley and Sons, Chichester, West Sussex, England.

Duesterloh S, Short JW, Barron MG. 2002. Photoenhanced toxicity of weathered Alaska North Slope crude oil to the calanoid copepods *Calanus marshallae* and *Metridia okhotensis*. Environ Sci Technol 36:3953-3959.

Evans D. 2008. An evaluation of the harmful alteration, disruption, and destruction (HADD) of fish habitat following the Bunker 'C' and pole treating oil release on Wabamun Lake, Alberta. Report submitted to: Public Prosecution Service of Canada, Alberta Regional Office, Alberta, Canada.

Fallahtafti S, Rantanen T, Brown RS, Snieckus V, Hodson PV. 2011. Toxicity of hydroxylated alkyl-phenanthrenes to the early life stages of Japanese medaka (*Oryzias latipes*). Aquat Toxicol 106-107:56-64.

Faisal M, Huggett RJ. 1993. Effects of polycyclic aromatic hydrocarbons on the lymphocyte mitogenic responses in spot, *Leiostomus xanthurus*. Mar Environ Res 35:121-124.

Farwell AJ, Nero V, Croft M, Rhodes S, Dixon DG. 2006. Phototoxicity of oil sands–derived polycyclic aromatic compounds to Japanese medaka (*Oryzias latipes*) embryos. Environ Toxicol Chem 25:3266-3274.

Fingas M, Hollebone B, Fieldhouse B. 2005. The density behaviour of heavy oils in freshwater: The example of the Lake Wabamun spill. Emergencies Science and Technology Division, Environment Canada Environmental Technology Centre Ottawa, Canada.

Fremling CR. 1981. Impacts of a spill of No. 6 fuel oil on Lake Winona. In Proceedings of the 1981 Oil Spill Conference, American Petroleum Institute, Washington, DC. pp. 419-421.

George SG, Wright J, Conroy J. 1995. Temporal studies of the impact of the Braer oil spill on inshore feral fish from Shetland, Scotland. Arch Environ Contam Toxicol 29:530-534.

Gohlke JM, Doke D, Tipre M, Leader M, Fitzgerald T. 2011. A review of seafood safety after the *Deepwater Horizon* blowout. Env Health Perspect 119:1062-1069.

Goldberg H. 2006. Pine River: 2005 Assessment –Residual Oil Survey and Snorkel Survey. Submitted to: District of Chetwynd, West Moberly First Nations, and Saulteau First Nation by ARC Environmental Ltd, Kamloops, B.C.

Goldberg H. 2011. Pine River 2011 Fisheries Update: Status of Recovery Post-2000 Pipeline Rupture. Report submitted to Enbridge Northern Gateway Pipelines Project.

Goodman R. 2003. Tar balls: The end state. Spill Sci Tech Bull 8:117-121.

Hall AT, Oris JT. 1991. Anthracene reduces reproductive potential and is maternally transferred during long-term exposure in fathead minnows. Aquat Toxicol 19:249–264.

Harper BL, Flett B, Harris S, Abeyta C, Kirschner F. 2002. The Spokane Tribe's multipathway subsistence exposure scenario and screening level RME. Risk Anal 22:513-526.

Harper BL, Walker DE. 2015. Columbia Basin heritage fish consumption rates. Human Ecology. DOI 10.1007/s10745-015-9735-3. Published online March 31, 2015.

Hart JL. 1973. Pacific Fishes of Canada. Fisheries Research Board of Canada, Bulletin 180, Ottawa Canada.

Hatlen K, Sloan CA, Burrows DG, Collier TK, Scholz NL, Incardona JP. 2010. Natural sunlight and residual fuel oils are an acutely lethal combination for fish embryos. Aquat Toxicol 99:56-64.

Heintz RA. 2007. Chronic exposure to polynuclear aromatic hydrocarbons in natal habitats leads to decreased equilibrium size, growth, and stability of pink salmon populations. Integr Environ Assess Manag 3:351-363.

Heintz RA, Short JW, Rice SD. 1999. Sensitivity of fish embryos to weathered crude oil: Part II. increased mortality of pink salmon (*Oncorhynchus gorbuscha*) embryos incubating downstream from weathered Exxon Valdez crude oil. Environ Toxicol Chem 18:494-503.

Heintz RA, Rice SD, Wertheimer AC, Bradshaw RF, Thrower FP, Joyce JE, Short JW. 2000. Delayed effects on growth and marine survival of pink salmon *Oncorhynchus gorbuscha* after exposure to crude oil during embryonic development. Mar Ecol-Prog Ser 208:205-216.

Hicken CE, Linbo TL, Baldwin DH, Willis ML, Myers MS, Holland L, Larsen M, Stekoll MS, Rice SD, Collier TK, et al. 2011. Sublethal exposure to crude oil during embryonic development alters cardiac morphology and reduces aerobic capacity in adult fish. Proc Natl Acad Sci USA 108:7086-7090.

Hodson PV, Khan CW, Saravanabhavan G, Clarke LMJ, Brown RS. 2007a. Alkyl PAH in crude oil cause chronic toxicity to early life stages of fish. In Proceedings of the 30th Arctic and Marine Oil Spill Program (AMOP)

Technical Seminar. Edmonton, Alberta: Emergencies Science and Technology Division, Environment Canada.

Hodson PV, Qureshi K, Noble CAJ, Akhtar P, Brown RS. 2007b. Inhibition of CYP1A enzymes by alpha-napthoflavone causes both synergism and antagonism of retene toxicity to rainbow trout (*Oncorhynchus mykiss*). Aquat Toxicol 81:275-285.

Hogan NS, Lee KS, Kollner B, van den Heuvel MR. 2010. The effects of the alkyl polycyclic aromatic hydrocarbon retene on rainbow trout (*Oncorhynchus mykiss*) immune response. Aquat Toxicol 100: 246-254.

Hollebone BP, Fieldhouse B, Sergey G, Lambert P, Wang Z, Yang C, Landirault M. 2011. The behaviour of heavy oil in fresh water lakes. In Proceedings of the 34th Arctic and Marine Oil Spill Program (AMOP) Technical Seminar, Banff, Alberta: Emergencies Science and Technology Division, Environment Canada.

Hose JE, Brown ED. 1998. Field applications of the piscine anaphase aberration test: lessons from the Exxon Valdez oil spill. Mutat Res 399:167-178.

Hose JE, McGurk D, Marty GD, Hinton DE, Brown ED, Baker TT. 1996. Sublethal effects of the Exxon Valdez oil spill on herring embryos and larvae: Morphological, cytogenetic, and histopathological assessments, 1989-1991. Can J Fish Aquat Sci 53:2355-2365.

Incardona JP, Collier TK, Scholz NL. 2004. Defects in cardiac function precede morphological abnormalities in fish embryos exposed to polycyclic aromatic hydrocarbons. Toxicol Appl Pharm 196:191-205.

Incardona JP, Carls MG, Teraoka H, Sloan C, Collier T, Scholz N. 2005. Aryl hydrocarbon receptor-independent toxicity of weathered crude oil during fish development. Environ Health Perspect 113:1755-1762.

Incardona JP, Day HL, Collier TK, Scholz NL. 2006. Developmental toxicity of 4-ring polycyclic aromatic hydrocarbons in zebrafish is differentially dependent on Ah receptor isoforms and hepatic cytochrome P450 1A metabolism. Toxicol App Pharmacol. 217:308-321.

Incardona JP, Carls MG, Day HL, Sloan CA, Bolton JL, Collier TK, Scholz NL. 2009. Cardiac arrhythmia is the primary response of embryonic pacific herring (*Clupea pallasi*) exposed to crude oil during weathering. Environ Sci Technol 43:201-207.

Incardona JP, Collier TK, Scholz NL. 2011. Oil spills and fish health: exposing the heart of the matter. J Exposure Sci. Environ Epidemiol. 21:3-4.

Incardona JP, Vines CA, Linbo TL, Myers MS, Sloan CA, Anulacion BF, Boyd D, Collier TK, Morgan SG, Cherr GN, Scholz NL. 2012a. Potent phototoxicity of marine bunker oil to translucent herring embryos after prolonged weathering. PLoS ONE 7(2):e30116.

Incardona JP, Vines CA, Anulacion BF, Baldwin DH, Day HL, French BL, Labenia JS, Linbo TL, Myers MS, Olson OP, Sloan CA, Sol S, Griffin FJ, Menard K, Morgan SG, West JE, Collier TK, Ylitalo GM, Cherr GN, Scholz NL. 2012b. Unexpectedly high mortality in Pacific herring embryos exposed to the 2007 Cosco Busan oil spill in San Francisco Bay. Proc Natl Acad Sci 109:E51-E58.

Jacobson KC, Arkoosh MR, Kagley AN, Clemons ER, Collier TK, Casillas E. 2003. Cumulative effects of natural and anthropogenic stress on immune function and disease resistance in juvenile chinook salmon: *Oncorhynchus tshawytscha*. J Aquat Anim Health 15:1-12.

Johnson LL, Misitano D, Sol SY, Nelson GM, French B, Ylitalo GM, Hom T. 1998. Contaminant effects on ovarian development and spawning success in rock sole from Puget Sound, Washington. Trans Am Fish Soc 127:375–392.

Johnson LL, Lomax DP, Myers MS, Olson OP, Sol SY, O'Neill SM, West JE, Collier TK. 2008. Xenoestrogen exposure and effects in English sole (*Parophrys vetulus*) from Puget Sound, WA. Aquatic Toxicol. 88: 29-38.

Johnson LL, Ylitalo GM, Myers MS, Anulacion BF, Buzitis J, and Collier TK. 2015. Aluminum smelter-derived polycyclic aromatic hydrocarbons and flatfish health in the Kitimat marine ecosystem, British Columbia, Canada. Sci Total Environ 512-513:227-239.

Kennedy CJ, Farrell AP. 2008. Immunological alternations in juvenile Pacific herring, *Clupea pallasi*, exposed to aqueous hydrocarbons derived from crude oil. Environ Pollut 153:638-648.

Khan RA. 1991. Influence of concurrent exposure to crude oil and infection with *Trypanosoma murmanensis* (Protozoa, Mastigophora) on mortality in winter flounder, *Pseudopleuronectes americanus*. Can J Zool 69:876-880.

Khan RA. 1995. Histopathology in winter flounder, *Pleuronectes americanus*, following chronic exposure to crude oil. Bull Environ Contam Toxicol 54:297-301.

Kiceniuk JW, Khan RA. 1987. Effect of petroleum hydrocarbons on Atlantic cod, *Gadus morhua*, following chronic exposure. Can J Zool 65:490-494.

Law RJ, Kelly C, Baker K, Jones J, McIntosh AD, Moffat CF. 2002. Toxic equivalency factors for PAH and their applicability in shellfish pollution monitoring studies. J Environ Monitor 4:383–388.

Lee K, Prince R, Greer C, Doe K, Wilson J, Cobanli S, Wohlgeschaffen G, Alroumi D, King T, Tremblay G. 2003. Composition and toxicity of residual bunker C fuel oil in intertidal sediments after 30 years. Spill Sci Technol Bull 8:187-199.

Little EE, Cleveland L, Calfee R, Barron MG. 2000. Assessment of the photoenhanced toxicity of a weathered oil to the tidewater silverside. Environ Toxicol Chem 19:926-932.

Logan DT. 2007. Perspective on ecotoxicology of PAHs to fish. Hum Ecol Risk Assess 13:302-316.

Martin J. 2011. Comparative toxicity and bioavailability of heavy fuel oils to fish using different exposure scenarios. MSc thesis, Queen's University, Kingston, ON.

Marty GD, Hose JE, McGurk MD, Brown ED, Hinton DE. 1997a. Histopathology and cytogenetic evaluation of Pacific herring larvae exposed to petroleum hydrocarbons in the laboratory or in Prince William Sound, Alaska, after the Exxon Valdez oil spill. Can J Fish Aquat Sci 54:1846-1857.

Marty GD, Short JW, Dambach DM, Willits NH, Heintz RA, Rice SD, Stegeman JJ, Hinton DE. 1997b. Ascites, premature emergence, increased gonadal cell apoptosis, and cytochrome P4501A induction in pink salmon larvae continuously exposed to oil-contaminated gravel during development. Can J Zool 75:989-1007.

Maynard DJ, Weber DD. 1981. Avoidance reactions of juvenile coho salmon (*Oncorhynchus kisutch*) to monocyclic aromatics. Can J Fish Aquatic Sci 38:772-778.

McAuliffe CD. 1977. Dispersal and alteration of oil discharged on a water surface. Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms, Seattle WA, pp. 19-35.

McGrath JA, Di Toro DM. 2009. Validation of the target lipid model for toxicity assessment of residual petroleum constituents: Monocyclic and polycyclic aromatic hydrocarbons. Environ Sci Technol 28:1130-1148.

McIntosh S, King T, Wu D, Hodson PV. 2010. Toxicity of dispersed weathered crude oil to early life stages of Atlantic herring (*Clupea harengus*). Environ Toxicol Chem 29:1160-1167.

Middaugh DP, Chapman PJ, Shelton ME, McKenney CL, Courtney LA. 2002. Effects of fractions from biodegraded Alaska North Slope crude oil on embryonic inland silversides, *Menidia beryllina*. Arch Environ Contam Toxicol 42:236-243.

Moles A, Norcross BL. 1998. Effects of oil-laden sediments on growth and health of juvenile flatfishes. Can J Fish Aquat Sci 55:605-610.

Neff JM, Burns WA. 1996. Estimation of polycyclic aromatic hydrocarbon concentrations in the water column based on tissue residues in mussels and salmon: an equilibrium partitioning approach. Environ Toxicol Chem 15:2240-2253.

Parker-Hall HA, Owens EH. 2006. Lake Wabamun derailment. Fate and persistence of the spilled oil. Report prepared for CN Polaris Applied Sciences, Inc., Bainbridge Island, WA.

Payne JF, Fancey LF. 1989. Effect of polycyclic aromatic hydrocarbons on immune responses in fish: change in melanomacrophage centers in flounder exposed to hydrocarbon-contaminated sediments. Mar Environ Res 28:431-435.

Payne JF, Mathieu A, Collier TK. 2003. Ecotoxicological studies focusing on marine and freshwater fish. In PAHs: An Ecotoxicological Perspective (ed PET Douben), John Wiley & Sons, Ltd, Chichester, UK.

Perrin, C.J., A Heaton, and M.A. Laynes. 2000. White sturgeon (*Acipenser transmontanus*) spawning habitat in the lower Fraser River,1999. Report prepared by Limnotek Research and Development Inc. for BC Fisheries. 72p.

PSMFC. 1992. White sturgeon management framework plan. Pacific States Marine Fisheries Commission. Portland, OR. 201p

Quinn TP. 2005. The Behavior and Ecology of Pacific Salmon and Trout. University of Washington Press. 378 p.

Raatz SK, Silverstein J, Jahns L, Picklo Sr SJ. 2013. Issues of fish consumption for cardiovascular disease risk reduction. Nurtients 5:1081-1097.

Ramachandran SD, Hodson PV, Khan CW, Lee K. 2004. Oil dispersant increases PAH uptake by fish exposed to crude oil. Ecotox Environ Safe 59:300-308.

Rien, T.A., Beamesderfer,R.C.,1994. Accuracy and precision of white sturgeon age estimates from pectoral fin rays. Trans Am Fish Soc 123:255–265

Reynaud S, Deschaux P. 2006. The effects of polycyclic aromatic hydrocarbons on the immune system of fish: A review. Aquat Toxicol 77:229-238.

Rhodes S, Farwell A, Hewitt LM, MacKinnon M, Dixon DG. 2005. The effects of dimethylated and alkylated polycyclic aromatic hydrocarbons on the embryonic development of the Japanese medaka. Ecotox Environ Safe 60:247-258.

Saunders RL, Sprague JB. 1967. Effects of copper-zinc mining pollution on a spawning migration of Atlantic salmon. Water Res 1:419-432.

Schein A, Scott JA, Mos L, Hodson PV. 2009. Oil dispersion increases the apparent bioavailability and toxicity of diesel to rainbow trout (*Oncorhynchus mykiss*). Environ Toxicol Chem 28:595-602.

Scott JA, Hodson PV. 2008. Evidence for multiple mechanisms of toxicity in larval rainbow trout (*Oncorhynchus mykiss*) co-treated with retene and α-naphthoflavone. Aquat Toxicol 88:200-206.

Scott JA, Ross M, Lemire BC, Hodson PV. 2009. Embryotoxicity of retene in cotreatment with 2-aminoanthracene, a cytochrome P4501A inhibitor, in rainbow trout (*Oncorhynchus mykis*s). Environ Toxicol Chem 28:1304-1310.

Semakula SN, Larkin PA. 1968. Age, growth, food and yield of the white sturgeon (Acipenser transmontanus) of the lower Fraser River, British Columbia. J Fish Res Bd Canada. 25:2589-2602.

Shaw DG, Cheek LM, Paul AJ. 1977. Uptake and release of petroleum by intertidal sediments at Port Valdez, Alaska. Estuar Coast Mar Sci 5:429-436.

Sol SY, Johnson LL, Horness BH, Collier TK. 2000. Relationship between oil exposure and reproductive parameters in fish collected following the Exxon Valdez oil spill. Mar Pollut Bull 40:1139-1147.

Sun J, Zheng X. 2009. A review of oil-suspended particulate matter aggregation-a natural process of cleansing spilled oil in the aquatic environment. J Environ Monitor 11:1801- 1809.

Thomas P, Budiantara L. 1995. Reproductive life-history stages sensitive to oil and naphthalene in Atlantic croaker. Mar Environ Res 39:147-50.

Thorne RE, Thomas GL. 2008. Herring and the "Exxon Valdez" oil spill: an investigation into historical data conflicts. ICES J Mar Sci 65:44-50.

Tonina D, Buffington JM. 2007. Hyporheic exchange in gravel bed rivers with pool-riffle morphology: Laboratory experiments and three-dimensional modeling. Wat Resources Res 34.

Tonina D, Buffington JM. 2009a. Hyporheic exchange in mountain rivers I: Mechanics and environmental effects. Geogr Comp 3:1063-1086.

Tonina D, Buffington JM. 2009b. A three-dimensional model for analyzing the effects of salmon redds on hyporheic exchange and egg pocket habitat. Can J Fish Aquat Sci 66:2157-2173.

Truscott B, Walsh JM, Burton MP, Payne JF, Idler DR. 1983. Effect of acute exposure to crude petroleum on some reproductive hormones in salmon and flounder. Comp Biochem Phys C 75:121-130.

Turcotte D, Akhtar P, Bowerman M, Kiparissis Y, Brown RS, Hodson PV. 2011. Measuring the toxicity of alkyl-phenanthrenes to early life stages of medaka (*Oryzias latipes*) using partition-controlled delivery. Environ Toxicol Chem 30:487-495.

Tuvikene A. 1995. Responses of fish to polycyclic aromatic hydrocarbons (PAHs). Ann Zool Fennici 32:295-309.

USEPA 1993. *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons*. EAP/600/R-93/089.

USEPA 2000. *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories*. EPA-823-B-00-007. Washington, DC: U.S. Environmental Protection Agency, Office of Water. Available at http://www.epa.gov/waterscience/fish/guidance.html. Last accessed April 2015.

Vandermeulen JH, Gordon DC. 1976. Reentry of 5-year-old stranded Bunker C fuel oil from a low-energy beach into the water, sediments, and biota of Chedabucto Bay, Nova Scotia. J Fish Res Can 33:2002-2010.

Vehniainen, ER, Hakkinen J, and Oikari A. 2003. Photoinduced lethal and sublethal toxicityof retene, a polycyclic aromatic hydrocarbon derived from resin acid, to coregonid larvae. Environ Toxicol Chem 22:2995-3000.

Wang. Y.L., F.P. Binkowski, and S.I. Doroshov. 1985. Effect of temperature on earlydevelopment of white and lake sturgeon, *Acipenser transmontanus* and *Acipenserfulvescens*. In: N. Am. Sturgeons: Biol Aqua Potential. F.P. Binkowski and S.I. Doroshov eds. Dr. W. Junk Publishers, Dordrecht, p. 43-50.

Weber DD, Maynard DJ, Gronlund WD, Konchin V. 1981. Avoidance reactions of migrating adult salmon to petroleum hydrocarbons. Can J Fish Aquat Sci 38:779-781.

Wheeler B, White MP, Fleming LE, Taylor T, Harvey A, Depledge MH. 2014. Influences of the oceans on human health and well-being. In Bowen RE, Depledge MH, Carlarne CP, Fleming LE (eds) *Oceans and Human Health: Implications for Society and Well-Being*. John Wiley and Sons, pp 3-22.

Wickliffe J, Overton E, Frickel S, Howard J, Wilson M, Simon B, Echsner S, Nguyen D, Gauthe D, Blake D, Miller C, Elferink C, Ansari S, Fernando H, Trapido E, Kane A. 2014. Evaluation of polycyclic aromatic hydrocarbons using analytical methods, toxicology, and risk assessment research: seafood safety after a petroleum spill as an example. Environ Health Perspect 122:6–9.

Wilson MJ, Frickel S, Nguyen D, Bui T, Echsner S, Simon BR, Howard JL, Miller K, Wickliffe JK. 2015. A targeted health risk assessment following the *Deepwater Horizon* oil spill: Polycyclic aromatic hydrocarbon exposure in Vietnamese-American shrimp consumers. Env Health Perspect 123:152-159.

Wu D, Wang Z, Hollebone B, McIntosh S, King T, Hodson PV. 2012. Comparative toxicity of four chemically-dispersed and undispersed crude oils to rainbow trout embryos. Environ Toxicol Chem 31:754-765.

Yang C, Wang Z, Yang Z, Hollebone B, Brown CE, Landriault M, Fieldhouse B. 2011. Chemical fingerprints of Alberta oil sands and related petroleum products. Environ Forensics 12:173-188.

Yender R, Michel J, Lord C. 2002. Managing Seafood Safety after an Oil Spill. Seattle: Hazardous Materials Response Division, Office of Response and Restoration, National Oceanic and Atmospheric Administration. 72 pp.

Appendix A – Statement of Qualifications

This report has been prepared in accordance with my duty as an expert to assist: the Shxw'ōwhámel First Nation in conducting its assessment of the Project; (ii) provincial or federal authorities with powers, duties or functions in relation to an assessment of the Project's environmental and socio-economic effects and impacts on the Shxw'ōwhámel First Nation's Aboriginal title, rights, and interests; and (iii) any court seized with an action, judicial review, appeal, or any other matter in relation to the Project. A signed copy of my Certificate of Expert's Duty is attached as Appendix "B".

Appendix “B”: Certificate of Expert’s Duty

I, _Tracy Collier____of __Bainbridge Island WA USA_____have been engaged on behalf of Shxw’ōwhámel First Nation to provide evidence in relation to Trans Mountain Pipeline ULC’s Trans Mountain Expansion Project application currently before the National Energy Board.

In providing evidence in relation to the above-noted proceeding, I acknowledge that it is my duty to provide evidence as follows:

1. to provide evidence that is fair, objective, and non-partisan;

2. to provide evidence that is related only to matters within my area of expertise; and

3. to provide such additional assistance as the tribunal may reasonably require to determine a matter in issue.

I acknowledge that my duty is to assist the tribunal, not act as an advocate for any particular party. This duty to the tribunal prevails over any obligation I may owe any other party, including the parties on whose behalf I am engaged.

Date: __22_May 2015_____ Signature: _  __

PCURRICULUM VITAE

TRACY K. COLLIER

Science Director
Puget Sound Partnership
326 East D Street
Tacoma, WA 98421

Email: tkcoll@gmail.com
Mobile: (206) 369-2779

Education

University of Washington, Seattle, WA	- B.Sc.	1976	Fisheries
University of Washington, Seattle, WA	- M.Sc.	1978	Fisheries
University of Washington, Seattle, WA	- Ph.D.	1988	Fisheries

Positions held

2012-present	**Science Director**, Puget Sound Partnership
2013-present	**Chair**, Delta Independent Science Board, Delta Stewardship Council
2012-2013	**Vice-chair**, Delta Independent Science Board, Delta Stewardship Council
2011-2012	**Member**, Puget Sound Science Panel, Puget Sound Partnership
2011-2012	**Member**, Board of Directors, People for Puget Sound
2011-2012	**Member**, Great Lakes Restoration Initiative Action Plan Review Panel, USEPA Science Advisory Board
2010-present	**Member**, Delta Independent Science Board, Delta Stewardship Council
2010-present	**Technical Advisor**, Marine Mammal/Sea Turtle Technical Working Group for Natural Resource Damage Assessment, Deepwater Horizon Oil Spill
2010-2014	**Science Advisor**, NOAA Oceans and Human Health Initiative
2003-2010	**Division Director,** Environmental Conservation Division, Northwest Fisheries Science Center (NWFSC), NOAA Fisheries
2004-2008	**Adjunct Professor**, Dept. Veterinary Microbiology and Pathology Washington State University
2004-2010	**Affiliate Faculty**, Dept. Molecular and Environmental Toxicology, Oregon State University
2004-2006	**Principal Investigator**, Oceans and Human Health Initiative, NWFSC
2003-2004	**Courtesy Faculty**, Dept. Molecular and Environmental Toxicology, Oregon State University
1999-2000	**Salmon Recovery Team**, NWFSC
1996-1997	**Principal Investigator** on studies to determine injury to fishery resources in Rhode Island salt ponds following the *North Cape*oil spill.
1995-2005	**Center Coordinator** for habitat science, NWFSC
1994-2003	**Program Manager**, Ecotoxicology and Environmental Fish Health Program, NWFSC

1994-1996	**Principal Investigator** on study funded by NMFS Restoration Center and USEPA to determine efficacy of subtidal remediation.
1994-2010	**Research Advisor** for National Research Council Postdoctoral Research Associate Program
1993-2003	**Division Coordinator** for Natural Resource Damage Assessment and Restoration Science
1992-1996	**Research Group Leader**, Biochemical Pathology Group, ECD
1992-2010	**Supervisory Research Chemist**, ECD, NMFS
1992-1994	**Assistant Branch Manager**, Ecotoxicology Branch of the ECD, NMFS
1990-1994	**Division Coordinator** for Natural Resource Damage Assessment/Restoration after the EXXON Valdez oil spill
1993	**Visiting Scientist**, Laboratory of Marine Molecular Biology, Bergen, Norway.
1991	**Chief Scientist**, Natural Resources Damage Assessment project in Prince William Sound
1988-1992	**Assistant Task Manager**, Biochemistry Task of the ECD
1978-1992	**Research Chemist**, ECD
1976-1978	**Fishery Biologist**, ECD
1972-1976	**Biological Aid/Physical Science Technician,** ECD

Honors

2006	Silver Medal for Exceptional Federal Service, Dept. of Commerce, Hurricane Katrina Emergency Response
2006	NOAA Fisheries Employee of the Year
2005	Bronze Medal for Superior Federal Service, Dept. of Commerce. NOAA Coastal Storms Program
2004	Special Act Award for proposal establishing a NOAA Center of Excellence in Oceans and Human Health.
2004	Award from NOAA Fisheries HQ, for scientific contributions to NOAA Oil and Dispersant Summit.
2004	Bronze Medal from US Environmental Protection Agency, for contributions to the West Region National Coastal Assessment Program.
2003	Special Act Award, US Delegate to T/V Prestige Oil Spill, Galicia, Spain
1999	Bronze Medal for Superior Federal Service, Dept. of Commerce. Essential Fish Habitat Team
1994, 1996, 2002	Outstanding Performance Award, NMFS, NOAA
1987, 1991, 1998	Sustained Superior Performance Award, NMFS, NOAA
1984, 1990	Outstanding Performance Award, NMFS, NOAA
1986	Best Student Platform Presentation, PANWAT Annual Meeting
1984	Elected to Society of the Sigma Xi
1972	National Merit Scholar

Committee and Public Service

2014	Mentor for Interagency Ecological Program, Early career mentoring luncheon 2/28/14.
2013-present	Advisory Committee for Washington Sea Grant
2013-2014	Chair of Independent Science Advisory Panel, Ecosystem Restoration Program, Dept. Fish and Wildlife, State of California
2013-present	Editorial Board, Environments
2012-present	Advisory Panel/Coordinating Committee member, Salish Sea Marine Survival of Salmon project.
2012	External Review Panel for Hollings Marine Laboratory
2011-present	Chair of Toxicology Sub-group, 2010-2011 Cetacean Unusual Mortality Event in Northern Gulf of Mexico Working Group
2010-2012	Steering Committee, Cumulative Effects of Underwater Anthropogenic Sound on Marine Mammals, NCEAS
2010-2012	Ocean Policy Task Force member for Water Quality, NOAA
2010	Chair of Advisory Panel, Contaminants of Emerging Concern for Coastal and Marine Ecosystems in California, David and Lucile Packard Foundation
2009-present	Scientific Committee, International Symposium on Toxicity Assessment
2009-present	Appointed to pool of experts for GESAMP (Group of Experts on Scientific Aspects of Marine Protection), advising the United Nations.
2008-2010	Technical Working Group, Sacramento River fall chinook 'collapse'
2008-2009	Governance committee, Puget Sound Monitoring Consortium
2008-present	Editorial Board, Journal of Marine Biology
2007-2010	Aquatic Toxicology Editor, Environmental Toxicology and Chemistry
2006-2010	Executive Committee for Puget Sound Nearshore Partnership (PSNP).
2006-2010	Scientific Advisory Board, Unexplained Neurotoxicity in Biological Organisms of Northern Europe. Stockholm University, Stockholm, Sweden.
2006	Ad-Hoc Committee for Water Quality, Puget Sound Partnership
2005-2011	Steering Committee for APEC Marine Environmental Training and Education Center (AMETEC), Geoje Island, South Korea.
2004-2012	Science Advisor for 'EarthSky', national radio series.
2004-2005	Biological Review Team, Cherry Point herring status review
2004-2009	Management Committee, Puget Sound Assessment and Monitoring Program
2003-2005	Habitat Restoration Implementation Team, NOAA
1999-2001	Chair, Promotion Review Board, ECD, NWFSC
1999	PMAC Review Board, Conservation Biology Division, NWFSC
1999-2004	Steering Committee, Puget Sound Ambient Monitoring Program
1996-2000	Essential Fish Habitat Team, National Marine Fisheries Service
1996-2000	Technical Advisory Panel to Cook Inlet Keeper Program
1996-1998	Controlling Toxic Inputs Working Group of Puget Sound/Georgia Basin Transboundary Task Force
1996-1998	Salt Pond Injury Technical Working Group for natural resource injury assessment following the North Cape Oil Spill
1995-1996	Strategic Planning Team for Northwest Fisheries Science Center

1995-1997	Management Committee for Puget Sound Estuary Program
1994-2000	Environmental Monitoring Committee, Cook Inlet Regional Citizens Advisory Council
1994-1996	Scientific Advisory Committee, Marine Science Society of the Pacific Northwest
1993-1994	Chair, Technical Advisory Panel for Damage Assessment Regulations Team of NOAA
1992-1995	Reports Review Board, ECD
1991-1995	Personnel Evaluation and Guidance Committee, ECD
1991-1993	Restoration Science Committee, ECD
1988-1993	Computer Committee, ECD
1987	Science Curriculum Review Committee, Bainbridge Island High School

Graduate students/Post-docs

PhD students

Louise Kiel Jensen	2011	University of Tromsø	Opponent
Tomas Hansson	2008	Stockholm University	Opponent
David West	2006	Waikato University	External Examiner
Claudia Bravo	2003-2006	Oregon State University	Committee Member
Henrik Sundberg	2005	Stockholm University	Opponent
Gunilla Ericson	1999	Stockholm University	Opponent

Post-docs

Dr. Erin Oleson	2005
Dr. John Incardona	2002-2003
Dr. David Baldwin	2000-2002
Dr. Nat Scholz	1999
Dr. Jim Moore	1996-1997

Professional Memberships (past and present)

American Fisheries Society, American Meteorological Society, Marine Technology Society, The Ocean Society, Society of the Sigma Xi, Society of Environmental Toxicology and Chemistry (SETAC), Pacific Northwest SETAC, Taiwan Cetacean Society (first foreign member).

Peer reviews provided for

Journals

Aquatic Toxicology, Archives of Environmental Contamination and Toxicology, Bulletin of Environmental Contamination and Toxicology, Canadian Journal of Fisheries and Aquatic Sciences, Carcinogenesis, CRC Press, Chemosphere, Comparative Biochemistry and Physiology, Environmental Health Perspectives, Environmental Science and Technology, Environmental Toxicology and Chemistry, Ecotoxicology, Fish Physiology and Biochemistry,

Genetics and Molecular Biology, Human and Ecological Risk Assessment, Integrated Environmental Assessment and Management, Journal of Coastal Research, Marine Environmental Research, Pakistan Veterinary Journal, Pesticide Biochemistry and Physiology, Polycyclic Aromatic Compounds, Proceedings of the National Academy of Sciences, Reviews in Fisheries Science, Science of the Total Environment, Toxicology and Applied Pharmacology, Transactions of the American Fisheries Society.

Institutions
CALFED, Clemson University, City University of Hong Kong, Consortium for Ocean Leadership, Great Lakes Fishery Trust, Hudson River Foundation, Marine Board of the European Science Foundation, Natural Sciences and Engineering Research Council of Canada, NIEHS, NOAA Sea Grant, NOAA Damage Assessment Regulations Team, NOAA Oceans and Human Health Initiative, NOAA Saltonstall-Kennedy Grants, Oregon Sea Grant, Prince William Sound Regional Citizen's Advisory Council, Royal Melbourne Institute of Technology, San Francisco Estuary Institute, Stockholm University, U.S. Environmental Protection Agency, U.S. Fish and Wildlife Service, University of Washington, University of Wyoming, Washington Sea Grant, WHOI Sea Grant

Scientific Publications

155. **Collier, T.K.** 2014. Integration of environmental health and marine ecosystem services. In: Understanding the Connections Between Coastal Waters and Ocean Ecosystem Services and Human Health: Workshop Summary. R.M. Martinez and E. Rusch (Eds.) Institute of Medicine, National Academies Press. pp 13-19. http://www.iom.edu/Reports/2014/Understanding-the-Connections-Between-Coastal-Waters.aspx
154. Schwacke, L.H., C.R. Smith, F.I. Townsend, R.S. Wells, L.B. Hart, B.C. Balmer, **T.K. Collier**, S. De Guise, M.M. Fry, L.J. Guillette, Jr., S.V. Lamb, S.M. Lane, W.E. McFee, N.J. Place, M.C. Tumlin, G.M. Ylitalo, E.S. Zolman, and T.K. Rowles. 2014. Response to comment on health of common bottlenose dolphins (*Tursiops truncatus*) in Barataria Bay, Louisiana following the *Deepwater Horizon* oil spill. Environmental Science and Technology 48(7). DOI:10.1021/es5009278
153. **Collier, T.K.**, N. Denslow, E. Gallagher, M. Kostich, and D. Lattier. 2014. Evaluating Stressors in the San Francisco Estuary Using Biomarkers. Final Report of an Independent Scientific Advisory Panel to California Department of Fish and Wildlife, Ecological Research Program. 41 pp. https://www.dfg.ca.gov/erp/biomarkers.asp
152. **Collier, T.K.**, B.F. Anulacion, M.R. Arkoosh, J.P. Dietrich, J.P. Incardona, L.L. Johnson, G.M. Ylitalo, and M.S. Myers. 2014. Effects on fish of polycyclic aromatic hydrocarbon (PAH) and naphthenic acid exposures. In Tierney, K.B. (ed.) *Fish Physiology Volume 33:Organic Chemical Toxicology of Fishes.* Elsevier. pp 195-255.
151. Schwacke, L.H., C.R. Smith, F.I. Townsend, R.S. Wells, L.B. Hart, B.C. Balmer, **T.K. Collier**, S. De Guise, M.M. Fry, L.J. Guillette, Jr., S.V. Lamb, S.M. Lane, W.E. McFee, N.J. Place, M.C. Tumlin, G.M. Ylitalo, E.S. Zolman, and T.K. Rowles. 2014. Health of common bottlenose dolphins (*Tursiops truncatus*) in Barataria

Bay, Louisiana, following the *Deepwater Horizon* oil spill. Environmental Science and Technology 48:93-103.

150. Sandifer P.A., **T.K. Collier**, and J.M. Trtanj. 2014. Linking ocean, organism, and human health for sustainable management of coastal ecosystems. In Meyers RA (ed.) *Encyclopedia of Sustainability Science and Technology*. Springer Science + Business Media, LLC. (in press).
149. Redman, S., K. Stiles, M. Neuman, A. Knaster, K. Dzinbal, A. Mitchell, K. Boyd, R. Ponzio, K. Currens, and **T.K. Collier**. 2013. Puget Sound Partnership Adaptive Management Framework. Puget Sound Partnership Technical Report 2013-01. Available at psp.wa.gov. 26 pp.
148. **Collier, T.K**. 2013. Forensic ecotoxicology. In Aris, A.Z., T.H. Tengku Ismail, R. Harun, A.M. Abdullah, and M.Y. Ishak (eds) *From Sources to Solutions*. Springer Science+ Business Media Singapore. pp 503-508.
147. da Silva D.A.M., J. Buzitis, W.L. Reichert, J.E. West, S.M. O'Neill, L.L. Johnson, **T.K. Collier**, and G.M. Ylitalo. 2013. Endocrine disrupting chemicals in fish bile: A rapid method of analysis using English sole (*Parophrys vetulus*) from Puget Sound, WA, USA. Chemosphere 92:1550-1556.
146. Sandifer, P.A., J.M. Trtanj, and **T.K. Collier**. 2013. A perspective on the history and evolution of an Oceans and Human Health "metadiscipline" in the USA. Microbial Ecology 65:880-888.
145. **Collier, T.K.,** M.W.L. Chang, D.W.T. Au, and P.S. Rainbow. 2013. Biomarkers currently used in environmental monitoring. Chapter 15. In Amiard-Triquet, C., P.S. Rainbow, and J.-C. Amiard (eds.) *Ecological Biomarkers: Indicators of Ecotoxicological Effects*. CRC Press, Taylor and Francis Group, LLC. pp 385-409.
144. Trtanj, J.M., J.R. Davis, and **T.K. Collier**. 2013. Oceans and human health. In Férard, J.F. and C. Blaise (eds.) *Encyclopedia of Aquatic Ecotoxicology*. Springer Science + Business Media, LLC. pp 797-804.
143. **Collier, T.K**. 2013. Forensic ecotoxicology. In Férard, J.F. and C. Blaise (eds.) *Encyclopedia of Aquatic Ecotoxicology*. Springer Science + Business Media, LLC. pp 533-538.
142. Incardona, J.P., C.A. Vines, T.L. Linbo, M.S. Myers, C.A. Sloan, B.F. Anulacion, D. Boyd, **T.K. Collier**, S.G. Morgan, G.N. Cherr, and N.L. Scholz. 2012. Potent phototoxicity of marine bunker oil to translucent herring embryos after prolonged weathering. PLoS ONE 7(2):e30116.
141. Incardona, J.P., C.A.Vines, B.F. Anulacion, D.H. Baldwin, H.L. Day, B.L. French, J.S. Labenia, T.L. Linbo, M.S. Myers, O.P. Olson, C.A. Sloan, S. Sol, F.J. Griffin, K. Menard, S.G. Morgan, J.E. West, **T.K. Collier**, G.M. Ylitalo, G.N. Cherr, and N.L. Scholz. 2012. Unexpectedly high mortality in Pacific herring embryos exposed to the 2007 Cosco Busan oil spill in San Francisco Bay. Proc. Natl. Acad. Sci. 109:E51-E58.
140. Scholz, N.L., M.S. Myers, S.G. McCarthy, J.S. Labenia, J.K. McIntyre, G.M. Ylitalo, L.D. Rhodes, C.A. Laetz, C.M. Stehr, B.L. French, B. McMillan, D. Wilson, L. Reed, K.D. Lynch, S. Damm, J.W. Davis, and **T.K. Collier**. 2011. Recurrent die-offs of adult coho salmon returning to spawn in Puget Sound lowland urban streams. PLoS ONE 6(12):e28013.
139. Incardona, J.P., G.M. Ylitalo, M.S. Myers, N.L. Scholz, **T.K. Collier** (NOAA) and C.A. Vines, F.J. Griffin, E. Smith, G.N. Cherr (BML). 2011. The 2007 *Cosco Busan* oil spill:

Field and laboratory assessment of toxic injury to Pacific herring embryos and larvae in the San Francisco estuary: Final Report. 82 p. Available online at: http://www.fws.gov/contaminants/Restorationplans/CoscoBusan/Cosco_Settlement/App_D_Herring_Injury_Study.pdf

138. Nash, S.B., J. Bolton, R. Brownell, **T. K. Collier**, P.R. Dorneles, C. Godard-Codding, F. Gulland, J. Kucklick, L. Schwacke, S. Venn-Watson, P. Burkhardt-Holm, G. Donovan, C. Fossi, A. Hall, C. Rosa, M. Simmonds, T. Rowles, and G. Ylitalo. 2011. Report of the IWC pollution 2000+ phase II workshop. J. Cetacean Res. 12 (Suppl):423-436.
137. Curtis, L.R, M.R. Arkoosh, C.B. Garzon, **T.K. Collier**, M.S. Myers, J. Buzitis, and M.E. Hahn. 2011. Reduced cytochrome P4501A activity and recovery from oxidative stress during subchronic benzo[a]pyrene and benzo[e]pyrene treatment of rainbow trout. Toxicol. Appl. Pharmacol. 254:1-7.
136. Arkoosh, M.R., S. Strickland, A. Van Gaest, G.M. Ylitalo, L.L. Johnson, G.K. Yanagida, **T.K. Collier**, and J.P. Dietrich. 2011. Trends in organic pollutants and lipids in juvenile Snake River spring Chinook salmon with different outmigrating histories through the Lower Snake and Middle Columbia Rivers. Sci. Tot. Environ. 409:5086-5100.
135. Hicken, C.L., T.L. Linbo, D.W. Baldwin, M.L. Willis, M.S. Myers, L. Holland, M. Larsen, N.L. Scholz, **T.K. Collier**, G.S. Rice, M.S. Stekoll, and J.P. Incardona. 2011. Sublethal exposure to crude oil during embryonic development alters cardiac morphology and reduces aerobic capacity in adult fish. Proc. Natl. Acad. Sci. 108:7086-7090.
134. Van Gaest, A.L., J.P. Dietrich, D.E. Thompson, D.A. Boylen, S.A. Strickland**, T.K. Collier**, F.J. Loge, and M.R. Arkoosh. 2011. Survey of pathogens in hatchery Chinook salmon with different outmigration histories through the Snake and Columbia Rivers. J. Aquatic Animal Health 23:62-77.
133. Dietrich, J.P., D.A. Boylen, D.E. Thompson, D.K. Spangenberg, C.F. Bravo, E.J. Loboschefsky, G.M. Ylitalo, D.S. Fryer, **T.K. Collier**, M.R. Arkoosh, and F.J. Loge. 2011. An evaluation of the influence of stock origin and outmigration history on the disease susceptibility and survival of juvenile Chinook salmon. J. Aquatic Animal Health 23:35-47.
132. Bravo C.F., L.R. Curtis, M.S.Myers, J.P.Meador, L.L. Johnson, J. Buzitis, **T.K. Collier**, J.D. Morrow, C.A. Laetz, M.R. Arkoosh. 2011. Biomarker response and disease susceptibility in juvenile rainbow trout *Oncorhynchus mykiss* fed a high molecular weight polycyclic aromatic (PAH) mixture. Environ. Toxicol. Chem. 30:1-11.
131. Incardona J.P., **T.K. Collier**, and N.L. Scholz. 2011. Oil spills and fish health: exposing the heart of the matter. J. Exposure Sci. and Environmental Epidemiol. 21:3-4.
130. Hatlen, K., C.A. Sloan, D.G. Burrows, **T.K. Collier**, N.L. Scholz, and J.P. Incardona. 2010. Natural sunlight and residual fuel oils are an acutely lethal combination for fish embryos. Aquat. Toxicol. 99:56-64.
129. Arkoosh, M.R., D. Boylen, J. Dietrich, B.F. Anulacion, G. Ylitalo, C.F. Bravo, L.L. Johnson, F.J. Loge, and **T.K. Collier**. 2010. Disease susceptibility of salmon exposed to polybrominated diphenyl ethers (PBDEs). Aquat. Toxicol. 98:51-59.
128. Lindley, S.T., C.B. Grimes, M.S. Mohr, W. Peterson, J. Stein, J.T. Anderson, L.W. Botsford, D.L. Bottom, C.A. Busack, **T.K. Collier**, J. Ferguson, J.C. Garza, A.M. Grover, D.G. Hankin, R.G. Kope, P.W. Lawson, A. Low, R.B. MacFarlane, K. Moore, M. Palmer-Zwahlen, F.B. Schwing, J. Smith, C. Tracy, R. Webb, B.K. Wells, and T.H. Williams.

2009. What caused the Sacramento River fall Chinook stock collapse? U.S. Dept. Commerce, NOAA Tech. Memo., NMFS-SWFSC-447, 62 p.

127. Baldwin, D.H., J.A. Spromberg, **T.K. Collier**, and N.L. Scholz. 2009. A fish of many scales: extrapolating sublethal pesticide exposures to the productivity of wild salmon populations. Ecol. Appl. 19:2004-2015.
126. Johnson, L. L., G. M. Ylitalo, M. S. Myers, B. F. Anulacion, J. Buzitis, W. L. Reichert, and **T. K. Collier**. 2009. Polycyclic aromatic hydrocarbons and fish health indicators in the marine ecosystem in Kitimat, British Columbia. U.S. Dept. Commerce, NOAA Tech. Memo., NMFS-NWFSC-98, 123 p.
125. Laetz, C.A., D.H. Baldwin, **T.K. Collier**, V. Herbert, J.D. Stark, and N.L. Scholz. 2009. The synergistic toxicity of pesticide mixtures: Implications for risk assessment and the conservation of Pacific salmon. Env. Hlth. Perspect. 117:348-353.
124. Incardona, J.P., Carls, M.G., Day, H.L., Sloan, C.A., Bolton, J.L., **Collier, T.K.**, and Scholz N.L. 2009. Cardiac arrhythmia is the primary response of embryonic Pacific herring (*Clupea pallasi*) exposed to crude oil during weathering. Env. Sci. Technol., 43:201-207.
123. O'Neill, S.M., C.F. Bravo, and **T.K. Collier**. 2008. Environmental indicators for the Puget Sound Partnership: A regional effort to select provisional indicators (Phase 1). Report submitted to Puget Sound Partnership, 64 p. Available online at: Environmental indicators for the puget sound partnership: a regional effort to select provisional indicators (Phase 1)
122. Stewart, J.R., R.J. Gast, R.S. Fujioka, H.M. Solo-Gabriele, J.S. Meschke, L.A. Amaral-Zettler, E. del Castillo, M.F. Polz, **T.K. Collier**, M.S. Strom, C.D. Sinigalliano, P.D.R. Moeller, and A.F. Holland. 2008. The coastal environment and human health: microbial indicators, pathogens, sentinels and reservoirs. Environmental Health 7(Suppl. 2):S3. doi:10.1186/1476-069X-7-S2-S3.
121. Sol, S.Y., L.L. Johnson, D. Boyd, O.P. Olson, D.P. Lomax, and **T.K. Collier**. 2008. Relationships between anthropogenic chemical contaminant exposure and associated changes in reproductive parameters in male English sole (*Parophrys vetulus)* collected from Hylebos Waterway, Puget Sound, Washington. Arch Environ. Contam. Toxicol. 55:627-638.
120. Johnson, L.L., D.P. Lomax, M.S. Myers, O.P. Olson, S.Y. Sol, S.M. O'Neill, J.E. West, and **T.K Collier**. 2008. Xenoestrogen exposure and effects in English sole (*Parophrys vetulus*) from Puget Sound, WA. Aquatic Toxicol. 88: 29-38.
119. Carls, M.G., L. Holland, M. Larsen, **T.K. Collier**, N.L. Scholz, and J.P. Incardona. 2008. Fish embryos are damaged by dissolved PAHs, not oil particles. Aquatic Toxicol. 88:121-127.
118. Olson, O. P., L. Johnson, G. Ylitalo, C. Rice, J. Cordell, **T. K. Collier**, and J. Steger. 2008. Fish habitat use and chemical contaminant exposure at restoration sites in Commencement Bay, Washington. U.S. Dept. Commerce, NOAA Tech. Memo. NMFS-NWFSC-88.
117. Myers, M.S., B.F. Anulacion, B.L. French, W.L. Reichert, C.A. Laetz, J. Buzitis, O.P. Olson, S. Sol, and **T.K. Collier**. 2008. Improved flatfish health following remediation of a PAH-contaminated site in Eagle Harbor, Washington. Aquatic Toxicol. 88:277-288.

116. Hom, T., **T.K. Collier**, M.M. Krahn, M.S. Strom, G.M. Ylitalo, W.B. Nilsson, R.N. Paranjpye, and U. Varanasi. 2008. Assessing seafood safety in the aftermath of Hurricane Katrina. Page 73-94. In: *Mitigating Impacts of Natural Hazards on Fishery Ecosystems*, McLaughlin, K.D. (Ed.), American Fisheries Society Symposium 64, 446 pp.
115. Johnson L.L., M.R. Arkoosh, **T.K. Collier**, M.M. Krahn, J.P. Meador, M.S. Myers, W.R. Reichert, and J.E. Stein. 2008. The effects of polycyclic aromatic hydrocarbons in fish from Puget Sound Washington. In: *The Toxicology of Fishes*. Di Giulio R and Hinton D (eds). Taylor & Francis, New York, London.
114. Krahn, M.M., M.B. Hanson, R.W. Baird, R.H. Boyer, D.G. Burrows, C.K. Emmons, J.K.B. Ford, L.L. Jones, D.P. Noren, P.S. Ross, G.S. Schorr and **T.K. Collier.** 2007. Persistent organic pollutants and stable isotope ratios in biopsy samples (2004-2006) from Southern Resident killer whales. Marine Pollution Bulletin 54:190-211.
113. **Collier, T.K.,** S.M. O'Neill, J.E. West, and N.L. Scholz 2007. Toxic chemical contaminants and Puget Sound. In: Proc. 2007 Georgia Basin Puget Sound Research Conference. Available online at: http://depts.washington.edu/uwconf/2007psgb/2007proceedings/papers/12e_colli.pdf
112. Dickhoff, W.D., **T.K. Collier**, and U. Varanasi. 2007. The Seafood Dilemma: A Way Forward. *Guest Director's Line*. Fisheries 32:244-246.
111. Johnson L. L., G. M. Ylitalo, C. A. Sloan, B. F. Anulacion, A. N. Kagley, M. R. Arkoosh, T. A. Lundrigan, K. Larson, M. Siipola and **T. K. Collier**. 2007. Persistent organic pollutants in outmigrant juvenile chinook salmon from the Lower Columbia Estuary, USA. Science of The Total Environment, 374:342-366.
110. Arkoosh, M.R., A.N. Kagley, B.F. Anulacion, D.A. Bohlen, B.P. Sandford, F.J. Loge, L.L. Johnson, and **T.K. Collier**. 2007. Disease susceptibility of hatchery Snake River spring/summer Chinook salmon (*Oncorhynchus tshawytscha*) with different juvenile migration histories in the Columbia River. J Aquat. Animal Health 18:223-231.
109. Uravitch, J.A., **T.K. Collier**, E. Moore, T. Noji, L. Wenzel, S. White, and R. Zimmerman. 2006. Marine zoning and coastal management. pp 40-51. In: S.A. Murawski and G.C. Matlock (eds). *Ecosystem Science Capabilities Required to Support NOAA's Mission in the Year 2020*. U.S. Dept. Commerce, NOAA Tech. Memo. NMFS-F/SPO-74, 97 p.
108. Incardona, J.P., H.L. Day, **T.K. Collier**, and N.L. Scholz. 2006. Developmental toxicity of 4-ring polycyclic aromatic hydrocarbons in zebrafish is differentially dependent on Ah receptor isoforms and hepatic cytochrome P450 1A metabolism. Toxicol. App. Pharmacol. 217:308-321.
107. Johnson, L.L., G.M. Ylitalo, M.R. Arkoosh, A.N. Kagley, C. Stafford, J.L. Bolton, J. Buzitis, B.F. Anulacion, and **T.K. Collier**. 2006. Contaminant exposure in outmigrant juvenile salmon from Pacific Northwest estuaries. Environ. Monitoring Assess. 124:167-194.
106. Bus, J.S., R.A. Canady, **T.K. Collier**, J.W. Owens, S.D. Pettit, N.L. Scholz, and A.C. Street. 2006. The extension of molecular and computational information to risk assessment and regulatory decision-making. in (W. Benson and R.T. DiGiulio, eds) *Genomic Approaches for Cross-Species Extrapolations in Toxicology*. SETAC Press, Pensacola, FL. pp 151-180.
105. Hellou, J., **T.K. Collier**, and F. Ariese. 2006. Assessing PAH exposure in feral finfish from the Northwest Atlantic. Marine Pollution Bulletin 52: 433-441.

104. Scholz, N.L., N.K. Truelove, J.S. Labenia, D.H. Baldwin, and **T.K. Collier**. 2006. Dose-additive inhibition of chinook salmon acetylcholinesterase activity by mixtures of organophosphate and carbamate insecticides. Environ. Toxicol. Chem. 5:1200-1207.

103. Boland, M., M. Carls, E. Chavez, B. Chesney, B. Cluer, **T.K. Collier**, N. Consentino-Manning, J. Dillon, B. Donnelly, J. Hanson, M. Helvey, R.A. Heintz, B. Hoffman, T. Hooper, D. Kirkpatrick, K Koski, B. Lance, Stacey Li, M. Liverman, M. Longenbaugh, J. Mann, L. Mahan, B. Meyer, A. Moles, N. Munn, L. Peltz, E. Phillips, K. Phippen, S. Rice, M. Sommer, J. Stadler, D. Tonnes. 2005. Appendix G Non-fishing Impacts to Essential Fish Habitat and Recommended Conservation Measures. 88 p.

102. Incardona, J.P., M.G. Carls, H. Teraoka, C.A. Sloan, **T.K. Collier**, and N.L. Scholz. 2005. Aryl hydrocarbon receptor-independent toxicity of weathered crude oil during fish development. Environ. Hlth. Perspect. 113:1755-1762.

101. Arkoosh, M.R., D. Bohlen, C.L. Stafford, L.L. Johnson, and **T.K. Collier**. 2005. Use of disease challenge assay to assess immunotoxicity of xenobiotics in fish. In Techniques in Aquatic Toxicology, Vol. 2 (G. Ostrander, ed.) CRC Press, Inc. Boca Raton, FL. pp. 19-38.

100. Loge, F. J., M. R. Arkoosh, T. R. Ginn, L. L. Johnson, and **T. K. Collier**. 2005. Impact of environmental stressors on the dynamics of disease transmission. Environ. Sci. Technol. 39:7329-7336.

99. Baker, J.E., W. F. Bohlen, R. Bopp, B. Brownawell, **T. K. Collier**, K. J. Farley, W. R. Geyer, L. Rosman, and R. Nairn. PCBs in the Upper Hudson River: The science behind the dredging controversy. 2005. in (J. Levinton and J. Waldman, eds.) *The Hudson River Estuary*. Cambridge University Press, New York, NY. pp 349-367.

98. Elskus, A.M., **T.K. Collier**, and E. Monnosson. 2005. Interactions between lipids and persistent organic pollutants in fish. In: *Biochemistry and Molecular Biology of Fishes, Volume 6. Environmental Toxicology*. (T.P. Mommsen and T.W. Moon, eds). Elsevier Science. pp 119-152.

97. Arkoosh, M.R., E. Clemons, A. Kagley, C. Stafford, A. Glass, K. Jacobson, P. Reno, M. Myers, E. Casillas, L. Johnson, F. Loge, and **T.K. Collier**. 2004. Survey of pathogens in juvenile salmon *Oncorhynchus spp*. migrating through Pacific Northwest estuaries. Journal of Aquatic Animal Health 16:186-196.

96. Arkoosh, M.R., L. Johnson, P. A. Rossignol and **T.K. Collier**. 2004. Predicting the impacts of perturbations on salmon communities: Implications for monitoring. Canadian Journal of Fisheries and Aquatic Sciences 61:1166-1175.

95. Incardona, J.P., **T.K. Collier**, and N. L. Scholz. 2004. Defects in cardiac function precede morphological abnormalities in fish embryos exposed to polycyclic aromatic hydrocarbons. Toxicol. Appl. Pharmacol. 196:191-205.

94. **Collier, T.K.** 2004. Guest Editor. Mar. Environ. Res. 58(2-5). Special issue, Proceedings of PRIMO-12. M.O. James, Guest Editor-in-Chief.

93. Jacobson, K.C., M.R. Arkoosh, A.N. Kagley, E.R. Clemons, **T.K. Collier** and E. Casillas. 2003. Cumulative effects of natural and anthropogenic stress on immune function and disease resistance in juvenile chinook salmon: *Oncorhynchus tshawytscha*. J Aquatic Animal Health 15:1-12.

92. Payne, J.F., Mathieu, A., and **T.K. Collier**. 2003. Ecotoxicological studies focusing on marine and freshwater fish. In: PAHs—An Ecotoxicological Perspective (P.E.T. Dauben, ed). John Wiley and Sons. pp 191-224.
91. **Collier, T.K.** and S.M. Adams. 2003. Special Issue Editors. Causal relationships between exposure and effects in field studies. Human and Ecological Risk Assessment 9:15-266.
90. **Collier, T.K.** 2003. Forensic ecotoxicology: Establishing causality between contaminants and biological effects in field studies. Human and Ecological Risk Assessment 9:259-266.
89. Myers M.S., L.L. Johnson, and **T.K. Collier**. 2003. Establishing the causal relationship between polycyclic aromatic hydrocarbon (PAH) exposure and hepatic neoplasms and neoplasia-related liver lesions in English sole (*Pleuronectes vetulus*). Human and Ecological Risk Assessment 9:67-94.
88. **Collier, T.K.,** L.L. Johnson, and J.P. Meador. 2002. Introduction: Fish tissue and sediment effects thresholds for polychlorinated biphenyls, polycyclic aromatic hydrocarbons, and tributyltin. Aquatic Conservation: Marine and Freshwater Ecosystems. 12:489-492.
87. Meador, J.P., **T.K. Collier**, and J.E. Stein. 2002. Use of tissue and sediment-based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the US Endangered Species Act. Aquatic Conservation: Marine and Freshwater Ecosystems. 12:493-516.
86. Johnson, L.L, **T.K. Collier**, and J.E. Stein. 2002. An analysis in support of sediment quality thresholds for polycyclic aromatic hydrocarbons (PAHs) to protect estuarine fish. Aquatic Conservation: Marine and Freshwater Ecosystems. 12:517-538.
85. Meador, J.P., **T.K. Collier**, and J.E. Stein. 2002. Determination of a tissue and sediment threshold for tributyltin to protect prey species of juvenile salmonids listed under the US Endangered Species Act. Aquatic Conservation: Marine and Freshwater Ecosystems. 12:539-551.
84. Johnson L.L. and **T.K. Collier** 2002. Assessing contaminant-induced stress across levels of biological organization. In: Adams SM (ed), Biological Indicators of Aquatic Ecosystem Stress, pp 533-564. American Fisheries Society, Bethesda, MD, USA.
83. Arkoosh, M.R. and **T.K. Collier.** 2002. Ecological risk assessment paradigm for salmon: Analyzing immune function to evaluate risk. Human and Ecological Risk Assessment. 8:265-276.
82. Arkoosh, M. R., E. Casillas, E. Clemons, P. Huffman, A. N. Kagley, **T.K. Collier** and J. E. Stein. 2001. Increased susceptibility of juvenile chinook salmon (*Oncorhynchus tshawytscha*) to vibriosis after exposure to chlorinated and aromatic compounds found in contaminated urban estuaries. *Journal of Aquatic Animal Health* 13:257-268.
81. Scholz N.L. and **T.K. Collier**. 2001. Endangered salmon and probabilistic risk assessments for organophosphate pesticides. SETAC Globe 1:32-33.
80. Stehr C.M., D.W. Brown, T. Hom, B.F. Anulacion, W.L. Reichert, and **T.K. Collier**. 2000. Exposure of juvenile chinook and chum salmon to chemical contaminants in the Hylebos Waterway of Commencement Bay, Tacoma Washington. Journal of Aquatic Ecosystem Stress and Recovery 7:215-227.

79. Arukwe, A., R. Male, L.L. Johnson, K.A. Peck-Miller, **T.K. Collier**, and A. Goksøyr. 2000. Fish zona radiata (eggshell) proteins: evaluation and validation as a biomarker for xenoestrogen monitoring. Mar. Environ. Res. 50:195-196.
78. Sol, S. S., L. L. Johnson, B. H. Horness, and **T. K. Collier**. 2000. Relationship between oil exposure and reproductive parameters in fish collected following the EXXON VALDEZ oil spill. Mar. Poll. Bull. 40:1139-1147.
77. Scholz NL, Truelove N, French B, Berejikian B, Quinn T, Casillas E, and **Collier TK**. 2000. Diazinon disrupts antipredator and homing behaviors in chinook salmon. Canadian Journal of Fisheries and Aquatic Sciences 57:1911-1918.
76. Johnson L.L., S.Y. Sol, G.M. Ylitalo, T. Hom, B. French, O.P. Olson, and **T.K. Collier**. 1999. Reproductive injury in English Sole (*Pleuronectes vetulus*) from the Hylebos Waterway, Commencement Bay, Washington. Journal of Aquatic Ecosystem Stress and Recovery 6:289-310.
75. **Collier, T.K.**, K. Grodzinska, L. Lagadic, J.F. Børseth, J. Gonzalez-Lopez, A. Lebedev, and M. Leinerte. 1999. Assessing environmental quality and efficacy of remedial activities in the Upper Vistula Basin of Poland, using biomarkers. in Biomarkers: A Pragmatic Basis for Remediation of Severe Pollution in Eastern Europe (D.B. Peakall et al., eds.). Kluwer Academic Publishers, The Netherlands, pp. 211-227. .
74. **Collier, T.K.** and M.S. Myers. 1999. Using biomarkers to monitor remedial actions at a subtidal estuarine site severely contaminated with polynuclear aromatic hydrocarbons. in Biomarkers: A Pragmatic Basis for Remediation of Severe Pollution in Eastern Europe (D.B. Peakall et al., eds.). Kluwer Academic Publishers, The Netherlands, pp. 301-304.
73. Gallagher, E.P, K. Sheehy, P. Janssen, D.L. Eaton, and **T.K. Collier**. 1999. Isolation and cloning of homologous glutathione S-transferase cDNAs from English sole and starry flounder. Aquat. Toxicol. 44:171-182.
72. Myers, M.S., C.M. Stehr, O.P. Olson, D. Boyd, C.A. Krone, B. French, B.F. Anulacion, S.M. Pierce, and **T.K. Collier**. 1998. Toxicopathic liver pathology in flatfish from the Hylebos Waterway in Tacoma, Washington. Proc. 1998 Puget Sound Research Conference. pp 695-711. (http//www.wa.gov/ puget_sound)
71. Stehr, C.M., D.W. Brown, T. Hom, B.F. Anulacion, W.L. Reichert, **and T.K. Collier**. 1998. Exposure of juvenile chinook and chum salmon to chemical contaminants in the Hylebos Waterway of Commencement Bay. Proc. 1998 Puget Sound Research Conference. pp 712-722. (http//www.wa.gov /puget_sound)
70. Johnson L.L., S.Y. Sol, G.M. Ylitalo, T. Hom, B. French, O.P. Olson, and **T.K. Collier**. 1998. Reproductive anomalies in bottomfish from the Hylebos Waterway. Proc. 1998 Puget Sound Research Conference. pp 712-722. (http//www.wa.gov/puget_sound)
69. Peck-Miller, K.A., M.S. Myers, **T.K. Collier**, and J.E. Stein. 1998. Complete cDNA sequence of the Ki-ras proto-oncogene in liver of wild English sole (*Pleuronectes vetulus*) and mutation analysis of hepatic neoplasms and other toxicopathic liver lesions Molec. Carcinogenesis 23:207-216.
68. Myers, M.S., L.L. Johnson, O.P. Olson, C.M. Stehr, B.H. Horness, **T.K. Collier,** and B.B. McCain. 1998. Toxicopathic hepatic lesions as biomarkers of chemical contaminant exposure and effects in marine bottomfish species from the Northeast and Pacific coasts, USA. Mar. Poll. Bull. 37:92-113.

67. **Collier, T.K.**, B.F. Anulacion, and B.D. Bill. 1998. Hepatic CYP1A in winter flounder (*Pleuronectes americanus*) along the Northeast coast: Results from the National Benthic Surveillance Project. Mar. Poll. Bull. 37:86-91.
66. Myers, M.S., B. French, W.L. Reichert, M.L. Willis, B.F. Anulacion, **T.K. Collier**, and J.E. Stein. 1998. Reductions in CYP1A expression and hydrophobic DNA adducts concentration in liver neoplasms in English sole: Further evidence supporting the "resistant hepatocyte" model of hepatocarcinogenesis in this species. Mar. Environ. Res. 46:197-202.
65. **Collier, T.K**., L.L. Johnson, C.M. Stehr, M.S. Myers, and J.E. Stein. 1998. A comprehensive assessment of the impacts of contaminants on fish from an urban waterway. Mar. Environ. Res. 46:243-247.
64. Reichert, W.L., M.S. Myers, K. Peck-Miller, B. French, B.F. Anulacion, **T.K. Collier**, J.E. Stein and U. Varanasi. 1998. Genotoxic events in marine fish from exposure to complex mixtures of environmental contaminants. Mutation Research 411:215-225.
63. Anulacion, B.F., M.S. Myers, M.L. Willis, and **T.K. Collier.** 1998. Quantitation of CYP1A expression in two flatfish species showing different prevalences of contaminant-induced hepatic disease. Mar. Environ. Res. 46:7-11.
62. Horness, B.H., D.P. Lomax, L.L. Johnson, M.S. Myers, S.M. Pierce, and **T.K. Collier**. 1998. Use of hockey stick regression for determination of sediment quality thresholds: aromatic hydrocarbons versus liver lesions in a benthic fish species. Environ. Toxicol. Chem. 17:872-882.
61. Johnson, L.L., J.T. Landahl, L.A. Kubin, B.H. Horness, M.S. Myers, **T.K. Collier**, and J.E. Stein. 1998. Assessing the effects of anthropogenic stressors on Puget Sound flatfish populations. *J. Sea Res*. 39:125-137.
60. **Collier, T.K**., L.L. Johnson, C.M. Stehr, M.S. Myers, M.M. Krahn, and J.E. Stein. 1998. Fish Injury in the Hylebos Waterway of Commencement Bay, Washington. U.S. Dep. Commer., NOAA Tech. Memo. NMFS-NWFSC-36, 576 p.
59. Myers, M.S., L.L. Johnson, T. Hom, **T.K. Collier**, J.E. Stein, and U. Varanasi. 1998. Toxicopathic hepatic lesions in subadult English sole (*Pleuronectes vetulus*) from Puget Sound, Washington, U.S.A.: relationships with other biomarkers of contaminant exposure. *Mar. Environ. Res.* 45:47-67.
58. Landahl, J.T., L.L. Johnson, J.E. Stein, **T.K. Collier**, and U. Varanasi. 1997. Approaches for determining effects of pollution on fish populations in Puget Sound. Trans. Am. Fish. Soc. 126:519-535.
57. Anulacion, B.F., B.D. Bill, and **T.K. Collier**. 1997. Stability of cytochrome P4501A-associated enzyme activity in cryogenically stored teleost liver samples. Chemosphere 34:2029-2039.
56. Eufemia, N.A., **T.K. Collier**, J.E. Stein, D.E. Watson, and R.T. Di Giulio. 1997. Biochemical responses to sediment-associated contaminants in brown bullhead (*Ameriurus nebulosus*) from the Niagara River ecosystem. *Ecotoxicology* 5:1-22.
55. **Collier, T.K.**, C.A. Krone, M.M. Krahn, J.E. Stein, S.-L. Chan, and U. Varanasi. 1996. Petroleum exposure and associated biochemical effects in subtidal fish after the *EXXON Valdez* oil spill. Am. Fish. Soc. Symp. 18:671-683.
54. McCain, B.B., D.W. Brown, T. Hom, M.S. Myers, S.M. Pierce, **T.K. Collier**, J.E. Stein, S.-L. Chan, and U. Varanasi. 1996. Chemical contaminant exposure and effects in four fish species from Tampa Bay. *Estuaries* 19:86-104.

53. Varanasi, U., **T.K. Collier**, C.A. Krone, M.M. Krahn, L.L. Johnson, M.S. Myers, and S-L. Chan. 1995. Assessment of oil spill impacts on fishery resources: Measurement of hydrocarbons and their metabolites, and their effects, in important species. *Exxon Valdez* Oil Spill State/Federal Natural Resource Damage Assessment Final Report (Subtidal 7), National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, Washington.

52. Sol, S.-Y., L. L. Johnson, **T. K. Collier**, M. M. Krahn, and U. Varanasi. 1995. Contaminant effects on reproductive output in North Pacific flatfish. Proceedings of the International Pacific Flatfish Conference. Alaska Sea Grant College Program AK-SG-95-04. pp 547-560.

51. Myers, M.S., M.L. Willis, A.-M. Husøy, A. Goksøyr, and **T.K. Collier**. 1995. Immunochemical localization of cytochrome P4501A in contaminant-associated hepatic lesions of English sole (*Pleuronectes vetulus*). *Mar. Environ. Res.* 39:283-288.

50. Stein, J.E., T. Hom, **T.K. Collier**, D.W. Brown, and U. Varanasi. 1995. Contaminant exposure and biochemical effects in outmigrant juvenile Chinook salmon from urban and non-urban estuaries of Puget Sound, WA. *Environ. Toxicol. Chem.* 14:1019-1029.

49. Johnson, L. L., J. E. Stein, T. Hom, S. Sol, **T. K. Collier**, and U. Varanasi. 1995. Effects of exposure to Prudhoe Bay crude oil on reproductive function in gravid female flatfish. *Environmental Science* 3:67-81.

48. Casillas, E., M.R. Arkoosh, E. Clemons, T. Hom, D. Misitano, **T.K. Collier**, J.E. Stein, and U. Varanasi. 1995. Chemical contaminant exposure and physiological effects in outmigrant juvenile salmon from urban estuaries of Puget Sound, Washington. pp 657-665. In: *Proc. Puget Sound Research '95*, Vol. 2.

47. **Collier, T.K.**, B. F. Anulacion, J. E. Stein, A. Goksøyr, and U. Varanasi. 1995. A field evaluation of cytochrome P4501A as a biomarker of contaminant exposure in three species of flatfish. *Environ. Toxicol. Chem.* 14:154-162.

46. Myers, M.S., L.L. Johnson, O.P. Olson, C.M. Stehr, D.P. Lomax, B.H. Horness, B.F. Anulacion, M.L. Willis, **T.K. Collier**, B.B. McCain, J.E. Stein, and U. Varanasi. 1994. Toxicopathic hepatic lesions and other biomarkers of exposure to chemical contaminants in marine bottomfish species from the Northeast and Pacific Coasts, U.S.A. Baltic Marine Biologists, Publication No. 15, pp 81-98.

45. Casillas, E., M.R. Arkoosh, E. Clemons, T.Hom, D.Misitano, **T.K. Collier**, J.E. Stein, and U. Varanasi. 1994. Chemical contaminant exposure and physiological effects in outmigrant Chinook salmon from selected estuaries of Puget Sound, Washington. In: Salmon Ecosystem Restoration: Myth and Reality. Proceedings of the 1994 Northeast Pacific Chinook and Coho Workshop, M. Keefe (Ed.). pp 86-102.

44. Husøy, A.-M., M.S. Myers, M.J. Willis, **T.K. Collier**, M. Celander, and A. Goksøyr. 1994. Immunohistochemical localization of CYP1A and CYP3A-like isozymes in hepatic and extrahepatic tissues of Atlantic cod (*Gadus morhua* L.), a marine fish. *Toxicol. Appl. Pharmacol.* 129:294-308.

43. Johnson, L.L., J.E. Stein, **T.K. Collier**, E. Casillas, and U. Varanasi. 1994. Ovarian development, plasma estradiol concentrations, egg weight and fecundity in winter flounder (*Pseudopleuronectes americanus*) from urban and nonurban estuaries on the northeast Atlantic coast. *Sci Total Environm.* 141:241-260.

42. Husøy, A.-M., M.J. Willis, M.S. Myers, **T.K. Collier**, and A. Goksøyr. 1993. Immunohistochemical localization of cytochrome P4501A1 in different organs of Atlantic cod (*Gadus morhua*). *Sci Total Environm.* Suppl. 1993: 671-680.
41. Egaas, E., J.U. Skåre, N.O. Svendsen, M. Sandvik, J. Netland, J.G. Falls, W.C. Dauterman, and **T.K. Collier**. 1993. A comparative study of the effects of atrazine on some xenobiotic metabolizing enzymes in fish and insects, and of the in vitro Phase II atrazine metabolism in fish, insects, mammals, and one plant species. *Comp. Biochem. Physiol.* 106C:141-149.
40. **Collier, T.K.**, J.E. Stein, A. Goksøyr, M.S. Myers, J.W. Gooch, R.J. Huggett, and U. Varanasi. 1993. Biomarkers of PAH exposure and effects in oyster toadfish (*Opsanis tau*) from the Elizabeth River, Virginia. *Environ. Sci.* 2:161-177.
39. Varanasi, U., E. Casillas, M.R. Arkoosh, T. Hom, D.A. Misitano, D.W. Brown, S-L. Chan, **T.K. Collier**, B.B. McCain, and J.E. Stein. 1993. Contaminant exposure and associated biological effects in juvenile chinook salmon (*Oncorhynchus tshawytscha*) from urban and nonurban estuaries of Puget Sound. U.S. Dep. Commer., NOAA Tech. Memo. NMFS-NWFSC-8, 112 p.
38. **Collier, T.K.**, M.M. Krahn, C.A. Krone, L.L. Johnson, M.S. Myers, S.-L. Chan, and U. Varanasi. 1993. Survey of oil exposure and effects in subtidal fish following the EXXON Valdez oil spill: 1989-1991. In: *Exxon Valdez* oil spill symposium, Abstract book,. p 235-238. (Available from the Oil Spill Public Information Center, 645 G. Street, Anchorage, AK 99501).
37. **Collier, T.K.**, M.M. Krahn, C.A. Krone, L.L. Johnson, M.S. Myers, S.-L. Chan, and U. Varanasi. 1993. Oil exposure and effects in subtidal fish following the EXXON Valdez oil spill. In: *Proceedings 1993 International Oil Spill Conference* pp 301-305.
36. **Collier, T.K.**, J.E. Stein, H.R. Sanborn, T. Hom, M.S. Myers, and U. Varanasi. 1993. A field study of the relationship between bioindicators of maternal contaminant exposure and egg and larval viability of English sole (*Parophrys vetulus*). *Mar. Environ. Res.* 35:171-176.
35. Stein, J.E., **T.K. Collier**, W.L. Reichert, E. Casillas, T. Hom, and U. Varanasi. 1993. Bioindicators of contaminant exposure and sublethal effects in benthic fish from Puget Sound, WA. *Mar. Environ. Res.* 35:95-100.
34. Johnson, L.L., E. Casillas, J.E. Stein, **T.K. Collier**, and U. Varanasi. 1993. Contaminant effects on reproductive success in selected benthic fish species. *Mar. Environ. Res.* 35:165-170.
33. **Collier, T.K.**, S.D. Connor, B.-T.L. Eberhart, B.F. Anulacion, A. Goksøyr, and U. Varanasi. 1992. Using cytochrome P450 to monitor the aquatic environment: Initial results from regional and national surveys. *Mar. Environ. Res.* 34:195-199.
32. Varanasi, U., J.E. Stein, L.L. Johnson, **T.K. Collier**, E. Casillas, and M.S. Myers. 1992. Evaluation of bioindicators of contaminant exposure and effects in coastal ecosystems. In: *Ecological Indicators*, Vol. 1. D.H. McKenzie, D.E. Hyatt, and V.J. McDonald, (Eds.). pp 461-498.
31. **Collier, T.K.**, S.V. Singh, Y.C. Awasthi, and U. Varanasi. 1992. Hepatic xenobiotic metabolizing enzymes in two species of benthic fish showing different prevalences of contaminant-associated liver neoplasms. *Toxicol. Appl. Pharmacol.* 113:319-324.

30. **Collier, T.K.**, J.E. Stein, H.R. Sanborn, T. Hom, M.S. Myers, and U. Varanasi. 1992. Field studies of reproductive success in English sole (*Parophrys vetulus*): Correlations with bioindicators of maternal contaminant exposure. *Sci Total Environm.* 116:169-185.
29. Johnson, L.L., J.E. Stein, **T.K. Collier**, E. Casillas, B. McCain, and U. Varanasi 1992. Bioindicators of contaminant exposure, liver pathology, and reproductive development in prespawning female winter flounder (*Pleuronectes americanus*) from urban and nonurban estuaries on the Northeast Atlantic coast. NOAA Technical Memorandum NMFS-NWFSC-1, 76 p.
28. Krahn, M.M., D.G. Burrows, G.M. Ylitalo, D.W. Brown, C.A. Wigren, **T.K. Collier**, S.-L. Chan, and U. Varanasi. 1992. Mass spectrometric analysis for aromatic compounds in bile of fish sampled after the *EXXON Valdez* oil spill. *Environ. Sci. Technol.* 26:116-126.
27. Stein, J.E., **T.K. Collier**, W.L. Reichert, E. Casillas, T. Hom, and U. Varanasi. 1992. Bioindicators of contaminant exposure and sublethal effects: studies with benthic fish in Puget Sound, WA. *Environ. Toxicol. Chem.* 11:701-714.
26. **Collier, T.K.** and U. Varanasi. 1991. Hepatic activities of xenobiotic metabolizing enzymes and biliary levels of xenobiotics in English sole *(Parophrys vetulus)* exposed to environmental contaminants. *Arch. Environ. Contam. Toxicol.* 20:462-473.
25. Casillas, E., D. Misitano, L. Johnson, L. Rhodes, **T.K. Collier,** J.E. Stein, B. McCain and U. Varanasi. 1991. Inducibility of spawning and reproductive success of female English sole (*Parophrys vetulus*) from urban and nonurban areas of Puget Sound, Washington. *Mar. Environ. Res*. 31:99-122.
24. **Collier, T.K**., B.-T. L. Eberhart, J.E. Stein and U. Varanasi. 1989. Aryl hydrocarbon hydroxylase-a 'new' monitoring tool in the Status & Trends Program. pp. 608-610. In: *Proceedings Oceans '89* IEEE, Washington, D.C.
23. Johnson, L., E. Casillas, D Misitano, **T.K. Collier,** J.E. Stein, B.B. McCain and U. Varanasi. 1989. Bioindicators of reproductive success in female English sole (*Parophrys vetulus*) exposed to environmental contaminants. pp. 391-396. In: *Proceedings Oceans '89* IEEE, Washington, D.C.
22. **Collier, T.K**. 1988. Xenobiotic metabolizing enzymes in liver of flatfish from Puget Sound, Washington. Doctoral dissertation, University of Washington. 110 p.
21. **Collier, T.K**., J.E. Stein, W.L. Reichert, B.-T.L. Eberhart and U. Varanasi. 1988. Using bioindicators to assess contaminant exposure in flatfish from Puget Sound, WA. pp. 567-576. In: *Proc. First Ann. Mtg. on Puget Sound Research*, Vol. 2.
20. Johnson, L., E. Casillas, **T.K. Collier**, B.B. McCain, U. Varanasi**.** 1988. Contaminant effects on ovarian maturation in English sole (*Parophrys vetulus*) from Puget Sound. pp. 651-661. In: *Proc. First Ann. Mtg. on Puget Sound Research*, Vol. 2.
19. Johnson, L., E. Casillas, **T.K. Collier**, B.B. McCain and U. Varanasi**.** 1988. Contaminant effects on ovarian maturation in English sole (*Parophrys vetulus*) from Puget Sound, Washington. *Canadian J. Fish Aquatic Sci.* 45:2133-2146.
18. **Collier, T.K**. and U. Varanasi. 1987. Biochemical indicators of contaminant exposure in flatfish from Puget Sound, WA. pp. 1544-1549. In: *Proceedings Oceans '87* IEEE, Washington, D.C.
17. Varanasi, U., J.E. Stein, M. Nishimoto, W.L. Reichert and **T.K. Collier.** 1987. Chemical carcinogenesis in feral fish--uptake, activation and detoxication of organic xenobiotics. *Environ. Hlth. Persp*. 71: 155-170.

16. **Collier, T.K**., J.E. Stein, R.J. Wallace and U. Varanasi. 1986. Xenobiotic metabolizing enzymes in spawning English sole (*Parophrys vetulus*) exposed to organic-solvent extracts of marine sediments from contaminated and reference areas. *Comp. Biochem. Physiol.* 84C:291-298.
15. Varanasi, U., **T K. Collier**, D.E. Williams and D.R. Buhler. 1986. Hepatic cytochrome P-450 isozymes and aryl hydrocarbon hydroxylase in English sole (*Parophrys vetulus*). *Biochem. Pharmacol.* 35:2967-2971.
14. **Collier, T.K**., E.H. Gruger and U. Varanasi. 1985. Effect of Aroclor 1254 on the biological fate of 2,6-dimethylnaphthalene in coho salmon (*Oncorhynchus kisutch*). *Bull. Environ. Contam. Toxicol.* 34:114-120.
13. Krahn, M.M., **T.K. Collier** and D.C. Malins. 1982. Aromatic hydrocarbons metabolites in fish: Automated extraction and high-performance liquid chromatographic separation into conjugate and non-conjugate fractions. *J. Chrom.* 236:441-452.
12. Malins, D.C. and **T.K. Collier**. 1981. Xenobiotic interactions in aquatic organisms: Effects on biological systems. *Aquatic Toxicology* 1:257-268.
11. **Collier**, **T.K**., M.M. Krahn and D.C. Malins. 1980. Disposition of naphthalene and its metabolites in brain of rainbow trout (*Salmo gairdneri*) *Environ. Research* 23:35-41.
10. Krahn, M.M., D.W. Brown, **T.K. Collier**, A.J. Friedman, R.G. Jenkins and D.C. Malins. 1980. Determination of naphthalene and its metabolites in biological systems by liquid chromatography with fluorescence detection. *Chromatog. Newsletter* 8:29-31.
9. Krahn, M.M., D.W. Brown, **T.K. Collier**, A.J. Friedman, R.G. Jenkins and D.C. Malins. 1980. Rapid analysis of naphthalene and its metabolites in biological systems: Determination by high performance liquid chromatography /fluorescence detection and by plasma desorption/chemical ionization mass spectrometry. *J. Biochem. Biophys. Methods*. 2:223-246.
8. Malins, D.C., M.M. Krahn, D.W. Brown, W.D. MacLeod and **T.K. Collier.** 1980. Analysis for petroleum products in marine environments. *Helgolander Meeresunters.* 33:257-271.
7. Malins, D.C., **T.K. Collier** and H.R. Sanborn. 1979. Disposition and metabolism of aromatic hydrocarbons in marine organisms. In: *Metabolism and Disposition of Pesticides and Other Chemicals in Aquatic Organism*s. American Chemical Society Symposium Series. 99:57-75.
6. Malins, D.C., **T.K. Collier**, L.C. Thomas and W.T. Roubal. 1979. Metabolic fate of aromatic hydrocarbons in aquatic organisms: Analysis of metabolites by thin-layer chromatography and high-pressure liquid chromatography. *Intl. J. Environ. Anal. Chem*. 6:55-66.
5. **Collier, T.K.,** L.C. Thomas and D.C. Malins. 1978. Influence of environmental temperature on disposition of dietary naphthalene in coho salmon (*Oncorhynchus kisutch*): Isolation and identification of individual metabolites. *Comp. Biochem. Physiol.* 61C:23-28.
4. **Collier, T.K.** 1978. Disposition and metabolism of metabolism of naphthalene in rainbow trout (Salmo gairdneri). M.S. Thesis, University of Washington, Seattle. 43 pp.
3. Roubal, W.T, D.H. Bovee, **T.K. Collier** and S.I. Stranahan. 1977. Flow-through system for chronic exposure of aquatic organisms to seawater soluble hydrocarbons from crude oil: Construction and applications. In: Proceedings 1977 Oil Spill Conference

(Prevention, Behavior, Control, Clean-up), p. 551-555. American Petroleum Institute, Washington, D.C.

2. Roubal, W.T, **T.K. Collier** and D.C. Malins. 1977. Accumulation and metabolism of carbon-14 labeled benzene, naphthalene, and anthracene by young coho salmon (*Oncorhynchus kisutch*). *Arch. Environ. Contam. Toxicol.* 5: 513-529.
1. Roubal, W.T. and **T.K. Collier**. 1975. Spin-labeling techniques for studying mode of action of petroleum hydrocarbons on marine organisms. *Fish. Bull.* 73:299-305.

Presentations/Panels/Sessions chaired/Conferences organized
(partial list, > 300 total)

Correa, L., and **T.K. Collier.** 2014. Communication between scientists and decision makers: the science/policy interface. Invited presentation, Cyberinfrastructure and Water Resources in the Lower Mekong Region. Hanoi, Vietnam. 8/21/14.

Collier, T.K. 2014. Selecting and using environmental indicators to assess ecosystem state. Invited presentation, Cyberinfrastructure and Water Resources in the Lower Mekong Region. Hanoi, Vietnam. 8/19/14.

Collier, T.K. 2014. Organizer and US co-chair, 2014 Salish Sea Ecosystem Conference. Seattle, WA. 4/29/2014-5/2/2014.

Collier, T.K. 2014. A U.S. perspective on Oceans and Human Health efforts in the European Union. Invited presentation, Oceans and Human Health: A rising tide of opportunities and challenges for Europe. Mawgan Porth, Cornwall, UK. 3/21/14.

Collier, T.K. 2014. Chemical contaminants, seafood safety, and public health. Keynote presentation, International Conference on Seafood Safety, Quality, and Traceability. Muscat, Sultanate of Oman, 3/4/14.

Collier, T.K. 2014. Evaluating stressors in the San Francisco Estuary using biomarkers. Invited presentation, Interagency Ecological Program Annual Workshop. Folsom, CA 2/27/14.

Collier, T.K. 2013. Science in support of large ecosystem recovery: Perspectives from California's Delta and Puget Sound. Invited seminar, Monster Seminar Jam series, Northwest Fisheries Science Center, Seattle, WA. 12/5/13.

Collier, T.K. 2013. Forensic ecotoxicology: Linking contaminant exposures to biological effects in field studies. Keynote presentation, 2013 International Conference on Environmental Forensics. Kuala Lumpur, Malaysia. 11/13/13.

Collier, T.K. 2013. Effects of PAHs on fish: Relevance for determination of sediment quality criteria and oil spill response. Invited presentation, International Conference on Environmental Mutagens, Iguassu Falls, Brazil. 11/4/13.

Collier, T.K. 2013. Exposure and sub-lethal effects of PAHs in Puget Sound fish: Relevance for determination of sediment quality criteria. Invited lecture, University of Campinas, School of Technology, Campinas, Brazil. 11/1/13.

Collier, T.K. 2013. Chemical contaminants and the life cycle of Pacific salmon in Puget Sound. Invited presentation, Swiss Center for Applied Ecotoxicology. Zurich, Switzerland. 10/18/13.

Collier, T.K. 2013. Chemical contaminants and their effects on salmon and other biota in Puget Sound. Invited presentation, WA Dept. Ecology Local Source Control Specialists workshop. Bellevue, WA 9/11/13.

Collier, T.K. 2013. Puget Sound Science 201: Polycyclic aromatic hydrocarbons and shoreline armoring as threats to the health of Puget Sound. Invited presentation, King County ECO Net Quarterly meeting, Seattle, WA 3/22/13.

Collier, T.K. 2013. Oceans and human health (and climate change). Invited presentation, Colloquium on Human Dimensions and Ocean Health in a Changing Climate, Univ. S. California, Los Angeles, CA. 3/12/13.

Collier, T.K. 2013. The environmental and public health implications of the Deepwater Horizon oil spill. Invited presentation, 16th Biennial Conference of the International Society for Toxicity Assessment. Cape Town, South Africa. 2/22/13.

Collier, T.K. 2012. Invited panel member, OR Sea Grant review panel on contaminants of emerging concern and tsunami debris from Japan. Corvallis, OR 12/5/12.

Collier, T.K. 2012. Integration of environmental health and marine ecosystem services. Invited presentation, Institute of Medicine Roundtable on Environmental Health Sciences, Research, and Medicine. Washington, DC. 11/13/12.

Collier, T.K. 2012. The Puget Sound Partnership monitoring program, and Science Panel recommendations. Invited presentation, Workshop on Marine Survival of Salmon and Steelhead in the Salish Sea. Bellingham, WA. 11/6/12.

Collier, T.K. 2012. Invited moderator, 4th South Sound Science Symposium. Shelton, WA. 10/30/12.

Collier, T.K. 2012. The environmental and public health implications of the Deepwater Horizon oil spill. Invited plenary presentation, 12th Brazilian Congress of Ecotoxicology, Porto de Galinhas, Pernambuco, Brazil. 9/27/12.

Collier, T.K. 2012. Applying toxicology and chemistry during responses to oil spills and other releases of hazardous substances. Invited mini-course, 12th Brazilian Congress of Ecotoxicology, Porto de Galinhas, Pernambuco, Brazil. 9/25/12.

Collier, T.K. 2012. Invited member, review panel for Coastal Response Research Center. Durham, NH. 9/11-12/12.

Collier, T.K. 2012. Monitoring of major spills – the importance and challenge – an overview of the developing landscape. Invited presentation, The PREMIAM Conference (Pollution Response in Emergencies: Marine Impact Assessment and Monitoring), London, England. 7/4/12.

Collier, T.K. 2012. Chemical contaminants, pathogens, and algal toxins and their effects on human and ecosystem health. Invited course with 14 lectures, CETESB (Environmental Company of São Paulo), São Paulo, Brazil. 6/26-29/12.

Collier, T.K. 2012. Impacts on human and ecosystem health of chemical contaminants in aquatic systems – evaluation and risk management. Invited seminar, CETESB (Environmental Company of São Paulo), São Paulo, Brazil. 6/25/12.

Collier, T.K. 2012. Assessing seafood safety after the BP Deepwater Horizon oil release. Invited presentation, 2012 Gordon Research Conference on Oceans and Human Health. Biddeford, ME 6/6/12.

Collier, T.K. 2012. Hazardous chemical releases associated with Hurricanes Katrina and Rita, and assessment of seafood safety. Invited presentation, 2012 Gordon Research Conference on Oceans and Human Health. Biddeford, ME 6/5/12.

Collier, T.K. and D.J. Grimes. 2012. Chairs, 2012 Gordon Research Conference on Oceans and Human Health. Biddeford, ME 6/3-6/8/12.

Collier, T.K. 2012. Considerations for a career in Oceans and Human Health. Invited presentation, 2012 Gordon Research Seminar on Oceans and Human Health. Biddeford, ME 6/3/12.

Collier, T.K. 2012. The importance of understanding thiamine deficiency syndrome. Invited presentation to Swedish businesses and philanthropists. Stockholm, Sweden 5/24/12.

Collier, T.K. 2012. Member of External Panel for review of Hollings Marine Laboratory. Charleston, SC 2/28-29/12.

Collier, T.K. 2012. Organizing committee for National Climate Assessment Health Sector Workshop, Northwest Region. Seattle, WA. 2/23-24/12.

Collier, T.K. 2012. Oil spills, seafood safety, and public health. Invited presentation, Winter Meeting of the Norwegian Society for Pharmacology and Toxicology, Beitostølen, Norway. 1/27/12.

Collier, T.K. 2012. Polycyclic aromatic hydrocarbons (PAHs) as threats to environmental and human health. Invited presentation, Winter Meeting of the Norwegian Society for Pharmacology and Toxicology, Beitostølen, Norway. 1/26/12.

Collier, T.K. 2012. Invited participant, Oil In Ice workshop, to develop research strategy for Fram Centre, Tromsø, Norway. 1/23-24/12.

Collier, T.K. 2011. Why fish need clean water too. Invited mini-course, taught in conjunction with the 1st International Workshop on Environmental Health, at the University of Sao Paulo, Sao Paulo, Brazil. 12/16/11.

Collier, T.K. 2011. Chemical contaminants, seafood safety, and public health. Invited speaker, 1st International Workshop on Environmental Health. University of Sao Paulo, Sao Paulo, Brazil. 12/14/11.

Collier, T.K. 2011. External opponent, Ph.D. defense for Louise Kiel Jensen, University of Tromsø, Norway. 11/11/11.

Collier, T.K., S. M. Moore, and J.M. Trtanj. 2011. Training the next generation of scientists in Oceans and Human Health. 21st Biennial Conference of the Coastal and Estuarine Research Federation, Daytona Beach, FL. 11/7/11.

Collier, T.K. and S. M. Moore. 2011. Session chairs, Training the next generation of scientists in Oceans and Human Health. 21st Biennial Conference of the Coastal and Estuarine Research Federation, Daytona Beach, FL. 11/7/11.

Collier, T.K. 2011. Chemical contaminants, seafood safety, and public health. Invited keynote speaker, 15th International Symposium on Toxicity Assessment. Hong Kong, 7/7/11.

Collier, T.K. 2011. Chemical contaminants and seafood safety. Invited keynote presentation, Symposium on Seafood Safety and Quality, sponsored by Oman Ministry of Agriculture and Fisheries Wealth. Muscat, Sultanate of Oman. 5/31/11.

Collier, T.K. 2011. Reopening fisheries after an oil spill: Tainting, chemical analysis, and risks to human health. PRIMO-16, Long Beach, CA. 5/18/11.

Collier, T.K., T.K. Rowles, L.H. Schwacke, and J.M. Trtanj. 2011. Using sentinel species to better inform human health assessments following the Deepwater Horizon oil spill. Invited presentation, Aerospace Medical Association, Anchorage, AK. 5/10/11.

Collier, T.K. and A.H. Ringwood. 2011. Invited session moderators, Ecosystem Effects of Oil Spills. Gulf Oil Spill Focused Topic Meeting, SETAC. Pensacola, FL. 4/26-27/11.

Collier, T.K. 2011. Emerging concerns about the effects and fate of chemical contaminants in marine biota. Invited conference plenary, Envirotox 2011. Darwin, Australia, 4/18/11.

Collier, T.K. 2011. Interactions between Puget Sound and human health. Invited lecture, WA Children's Environmental Health Working Group, Seattle, WA. 4/14/11.

Collier, T.K. 2011. Contributions of marine products to global nutrition, and implications of changing climate. Invited seminar, University of Washington/NOAA Oceans and Human Health Seminar Series, Seattle, WA. 3/1/11.

Collier, T.K. 2011. Overview of NOAA's Oceans and Human Health Initiative. Invited presentation, NSF Workshop on Ecology of Marine Infectious Diseases, San Juan, Puerto Rico. 2/12/11

Collier, T.K. 2011. Session chair, Fate and Effects of Oil Spills. Sponsored by National Research Council of Norway. Houston, TX. 2/8/11.

Collier, T.K. 2011. Invited panelist, Human Health Impacts of the Gulf Oil Spill, Nat'l Council for Science and the Environment, Washington, DC, 1/21/11.

Collier, T.K. 2011. Invited panelist on NPR's Science Friday, Washington, DC, 1/21/11.

Collier, T.K. 2010. New science regarding the effects of oil spills on marine resources. Invited presentation, King County Bar Association, Seattle, WA, 12/8/10.

Collier, T.K. and T.K. Rowles. 2010. NOAA data to support investigations of human health effects resulting from the Deepwater Horizon oil spill. Invited presentation, Nat'l Institutes of Health Interagency Meeting on Data Needs for Assessing Human Health Impacts of the Deepwater Horizon Oil Spill, Washington, DC 11/17/10.

Collier, T.K. 2010. The seafood 'dilemma': Balancing human health benefits and both real and perceived risk—a case in point, re-opening fisheries after an oil spill. Invited seminar, Center for Food Safety and Applied Nutrition, FDA, Gaithersburg MD. 11/16/10.

Collier, T.K. 2010. Represented NOAA at NY Academy of Sciences workshop and evening gala on Global Nutrition Initiative. New York, NY. 11/15/10.

Collier, T.K. 2010. Toxic chemical contaminants and Puget Sound. Invited presentation, SETAC North America, Portland, OR. 11/10/10.

Collier, T.K. 2010. What a long, strange trip it's been: Striving to understand the stress posed by toxic chemicals in Puget Sound. Invited departmental seminar, School of Aquatic and Fisheries Sciences, University of Washington, Seattle, WA 11/04/10.

Collier, T.K. 2010. Reopening fisheries after an oil spill: Tainting, chemical analysis, and human health. Invited presentation, 45th Session of the UJNR Panel on Toxic Microorganisms, Scientific Sessions. Seattle, WA 11/02/10.

Collier, T.K. 2010. Understanding the connections between ocean health and human health. Invited class lecture, Aichi Prefectural University, Nagoya, Japan. 10/27/10.

Collier, T.K. 2010. Environmental criteria for PAHs and implications of recent findings on aquatic animal health. Invited presentation, Physical and Chemical Impacts of Marine Organisms – for Conservation of Biodiversity and Sustainability. Aichi Prefectural University, Nagoya, Japan. 10/25/10.

Walker, S., and **T.K.Collier**. 2010. Panelists for Institute of Medicine workshop on Gulf Long-term Follow-up of Workers. Tampa, FL 9/22/10.

Collier, T.K. 2010. Potential for marine ecological and human health impacts from chemicals of emerging concern (CECs). Invited presentation, California and the World Ocean 2010. San Francisco, CA. 9/9/10.

Collier, T.K. 2010. Polycyclic aromatic hydrocarbons (PAHs) as threats to environmental and human health. Invited presentation, 3rd Summer Course on Environmental and Human Health, City University of Hong Kong. 7/15/10.

Collier, T.K. 2010. Sentinel species concepts in NOAA's Oceans and Human Health Program. Invited presentation, 3rd Summer Course on Environmental and Human Health, City University of Hong Kong. 7/15/10.

Collier, T.K. 2010. Seafood safety and human health. Invited presentation, 3rd Summer Course on Environmental and Human Health, City University of Hong Kong. 7/14/10.

Collier, T.K. and J. Turner. 2010. Oceans and Human Health: An Emerging Discipline. Invited presentation, 3rd Summer Course on Environmental and Human Health, City University of Hong Kong. 7/14/10.

Collier, T.K. 2010. Review of sea turtle/oil spill literature. Invited presentation, Sea Turtle and Marine Mammal NRDA Workshop, New Orleans, LA. 6/29/10.

Collier, T.K. 2010. Reopening fisheries after an oil spill: Tainting, chemical analysis, and human health. Keynote presentation, Assoc. Official Analytical Chemists, Annual Meeting of Pac NW Section, Tacoma, WA 6/22/10.

Collier, T.K., T. Noji, J. Kurland, B. Hoffman, and J. Rapp. 2010. An assessment of current processes for providing habitat science for management. Invited presentation, NOAA Fisheries 1st National Habitat Assessment Workshop. St. Petersburg, FL. 5/19/10.

Collier, T.K. 2010. Potential sea turtle exposure to chemical contaminants: Contaminants of emerging concern (CECs). Invited presentation, NOAA Sea Turtle and Contaminants Workshop. Washington, DC. 5/3/10.

Collier, T.K. 2010. Contaminants of emerging concern in coastal and marine ecosystems. Invited presentation, Pollution 2000. Sausalito, CA. 2/23/10.

Collier, T.K. 2010. Impacts of copper on the sensory biology and behavior of salmon. Invited presentation, Alaska Forum on the Environment. Anchorage, AK. 2/9/10.

Collier, T.K. 2009. Contaminated sediments and fish health: The Bad, the Good, and the Ugly. Invited presentation, Green Sturgeon Workshop. Oakland, CA. 12/2/09.

Collier, T.K. 2009. Monitoring for biological effects of oil spills in sub-Arctic U.S. water: Added effects of biomarkers? Invited presentation at workshop on 'Biomarkers—what role can they play in environmental management related to oil and gas activities offshore?' Paris, France. 11/27/09.

Collier, T.K. 2009. Report and conclusions from July 2009 Sousa conservation workshop. Invited presentation, Indo-Pacific Humpback Dolphin Conservation Action Plan meeting, Taipei, Taiwan. 11/1/09.

Collier, T.K. 2009. Sentinel species concepts in NOAA's OHHI. Invited presentation, NOAA OHHI All-PI meeting. Seattle, WA. 10/7/09.

Collier, T.K. 2009. Toxic chemicals and our aquatic ecosystems: how bad is it? Invited presentation, Pacific Fisheries Legislative Task Force. Astoria, OR. 9/18/09.

Collier, T.K. 2009. Introduction to the subject of toxic chemicals and salmon in the Columbia River. Invited presentation, Northwest Power and Conservation Council, Columbia River Estuary Science-Policy Exchange. Astoria, OR. 9/10/09.

Collier, T.K., L.L. Johnson, and J.P. Incardona. 2009. Environmental criteria for PAHs and implications of recent findings on aquatic animal health. Invited plenary presentation, International Society for Toxicity Assessment, 14th meeting. Metz, France. 8/31/09.

Collier, T.K. 2009. Polycyclic aromatic hydrocarbons (PAHs) as threats to environmental and human health. Invited plenary presentation, International Symposium on Environmental Pollution, Ecology, and Human Health. Tirupati, India. 7/26/09.

Collier, T.K. 2009. Opening keynote address, International Symposium on Environmental Pollution, Ecology, and Human Health. Tirupati, India. 7/25/09.

Collier, T.K. 2009. Working group chair, Group of experts evaluating conservation strategies for *Sousa chinensis.* Taipei, Taiwan, 7/21/09.

Collier, T.K. 2009. Strategies for habitat protection in coastal areas. Invited presentation (for Tom Bigford) at Symposium on Conservation Strategies for *Sousa chinensis* in Taiwan 2009. Taipei, Taiwan. 7/20/09.

Collier, T.K. 2009. Effect of petrochemicals on cetaceans and their prey base. Invited presentation at Symposium on Conservation Strategies for *Sousa chinensis* in Taiwan 2009. Taipei, Taiwan. 7/20/09.

Collier, T.K. 2009. NOAA's One Health Approach and its use in early warning systems. Invited presentation to GEO workshop on Priority Earth Observation Needs for Environmental Change and Human Health. Geneva, Switzerland. 7/10/09.

Collier, T.K. 2009. Environmental stressors and disease. Invited presentation to GEOSS Workshop XXVII - Health and the Environment. Geneva, Switzerland. 7/7/09.

Collier, T.K. 2009. Presented testimony on "Effects of endocrine disrupting compounds on fish and wildlife" before U.S. House of Representatives Subcommittee on Insular Affairs, Oceans, and Wildlife. Washington, DC. 6/09/09.

Collier, T.K. 2009. Session chair, Ecological Relevance, at PRIMO-15, Bordeaux, France. 5/19/09.

Collier, T.K., D.A. Baldwin, J.A. Spromberg, M.R. Arkoosh, L.L. Johnson, S. Hecht, and N.L. Scholz 2009. Linking contaminant-associated reductions in fish health to impaired population viability, in order to turn PRIMO into action. Platform presentation at PRIMO-15, Bordeaux, France. 5/19/09.

Collier, T.K. 2009. Co-organizer of workshop "Chemicals of emerging concern" in Costa Mesa, CA. 4/27-29/09.

Collier, T.K. 2009. Linking contaminant-associated reductions in fish health to impaired population viability, in order to support management actions.
Invited presentation, annual meeting of Pacific Northwest Chapter of SETAC. Port Townsend, WA. 4/18/09.

Collier, T.K. 2009. Organizer (in conjunction with NOS), Contaminants of Emerging Concern (CEC) Workshop: Adapting NOAA monitoring and research to address CEC management in coastal, marine, and Great Lakes environments. Costa Mesa, CA. 3/11-12/09.

Collier, T.K. 2009. Developing programs to ensure seafood safety and enhance human health: A U.S. perspective. Invited seminar, Symposium on Recent Trends in Environmental Pollution and Impacts. Burapha University, Chonburi, Thailand. 3/4/09.

Collier, T.K. 2009. Fish health, from mechanistic studies to population models, as a tool for assessing impacts of chemical contaminants. Invited seminar, Symposium on Recent Trends in Environmental Pollution and Impacts. Burapha University, Chonburi, Thailand. 3/4/09.

Collier, T.K. 2009. Invited review panel member, Hudson River Foundation. New York, NY. 2/12/09.

Collier, T.K. 2009. Toxic chemicals in the biota of the Salish Sea. Session chair, Puget Sound/Georgia Basin research conference. Seattle, WA. 2/10/09.

Collier. T.K. 2009. Activities and capabilities of environmental and human health programs at the Northwest Fisheries Science Center. Invited presentation, A One Health Approach? Integrating NOAA's Marine Organism and Human Health Related Data for Use in Early Warning Systems. Silver Spring, MD. 2/3/09.

Collier, T.K. 2009. Fish health, from mechanistic studies to population models, as a tool for assessing impacts of chemical contaminants. Invited seminar, National Institute for Environmental Studies, Center for Environmental Risk Research, Tsukuba, Japan. 1/14/09.

Collier, T.K. 2009. Fish health, from mechanistic studies to population models, as a tool for assessing impacts of chemical contaminants. Invited departmental seminar, Stanford University, Hopkins Marine Station, Pacific Grove, CA 1/9/09.

Collier, T.K. 2009. Toxic stormwater runoff: impacts on salmon in Seattle creeks. Invited presentation to the Seattle City Council. Seattle, WA 1/5/09.

Collier, T.K. 2008. Developing programs to ensure seafood safety and enhance human health: A U.S. perspective. Invited lecture, KORDI/AMETEC. Biochemical responses of fish to oil spills and other marine pollutants. Geoje Island, Korea. 11/4/08.

Anulacion, B.F., S.Y. Sol, and **T.K. Collier**. 2008. Obtaining high quality environmental samples. Invited lecture, KORDI/AMETEC. Biochemical responses of fish to oil spills and other marine pollutants. Geoje Island, Korea. 11/3/08.

Collier, T.K. 2008. The impacts of oil spills on both wild and cultured marine organisms. Invited lecture, KORDI/AMETEC. Biochemical responses of fish to oil spills and other marine pollutants. Geoje Island, Korea. 11/3/08.

Collier, T.K. 2008. Workshop organizer. KORDI/AMETEC. Biochemical responses of fish to oil spills and other marine pollutants. Geoje Island, Korea. 11/2-14/08.

Collier, T.K. 2008. Developing programs to ensure seafood safety and enhance human health: A U.S. perspective. Invited seminar, Ås, Norway. 10/20/08.

Collier, T.K. 2008. The effects of chemical contaminants on fish habitats, fish health, and fish populations. Invited seminar, Marine Environmental Laboratory, Int'l Atomic Energy Administration. Monaco. 10/15/08.

Collier, T.K. 2008. External opponent, Ph.D. defense for Tomas Hansson, Stockholm University. Stockholm, Sweden. 10/3/08.

Collier, T.K. and L.L. Johnson. 2008. Toxic chemical contaminants and salmon in the Columbia River. Invited presentation to the Northwest Power and Conservation Council. Kalispell, MT. 7/15/08.

Collier, T.K. 2008. Working together to protect human and environmental health from toxic chemicals. Invited presentation, Society of American Indians in Government, 5th Annual Training Conference. Traverse City MI. 6/4/08.

Collier, T.K. 2008. Overview of NOAA's Oceans and Human Health Initiative and the West Coast Center for Oceans and Human Health. Invited presentation, Society of American Indians in Government, 5th Annual Training Conference. Traverse City MI. 6/4/08.

Collier, T.K. 2008. Recent findings on algal toxins, organic chemicals, and marine mammal health (with some chemical ecology for dessert). Invited 'brown bag' seminar, NOAA Fisheries Office of Protected Resources, Silver Spring, MD. 5/28/08.

Collier, T.K. 2008. Session chair, Oil Spills and Aquaculture. World Aquaculture 2008. Busan, Korea. 5/23/08.

Collier, T.K. 2008. The impacts of oil spills on both wild and cultured marine organisms. Invited keynote speaker, World Aquaculture 2008. Busan, Korea. 5/23/08.

Collier, T.K. 2008. Breakout group lead for discussion of NOAA Climate Change priorities. Invited participant at NOAA Climate Change and LMR workshop. Seattle, WA. 5/14/08.

Collier, T.K. 2008. Toxic chemical contaminants and Puget Sound. Invited presentation at Puget Sound Symposium hosted by CH2M Hill, Bellevue, WA. 4/16/08.

Collier, T.K. 2008. Group lead for discussion of NMFS Habitat Science initiative. Invited participant at NMFS' national meeting of Assistant Regional Administrators for Habitat. Silver Spring, MD. 4/8/08.

O'Neill, S.M and **Collier, T.K.** 2008. Selecting provisional environmental indicators to determine progress in protecting and restoring the Puget Sound ecosystem. Invited presentation to the Leadership Council of the Puget Sound Partnership. 3/26/08.

Collier, T.K. 2008. Invited participant, Water Environment Research Foundation sponsored workshop: Developing a cross-sector research collaboration to address trace organics in receiving water. Alexandria, VA. 3/12-13/08.

Collier, T.K., W.W. Dickhoff, and U. Varanasi. 2008. The seafood dilemma: A way forward. Invited presentation in Oceans and Human Health session, Ocean Sciences 2008. Orlando, FL, 3/3/08.

Collier, T.K. 2008. Fish health, from mechanistic studies to population models, as a tool for assessing effects of chemical contaminants and other stressors. Invited conference keynote address, 37th annual meeting of the Interagency Ecological Program. Asilomar, CA, 2/27/08.

Collier, T.K. 2008. Invited review panel member, National Oceans and Human Health external grants review panel. Arlington, VA. 2/20-22/08.

Collier, T.K. 2008. Working our way up the biological scale from biomarkers to population dynamics: effects of pesticides on fish. Guest lecturer, Environmental Health 590, University of Washington, Seattle, WA. 2/13/08.

Collier, T.K. and S.M. O'Neill. 2008. Selecting provisional environmental indicators to determine progress in protecting and restoring the Puget Sound ecosystem. Invited presentation to the Ecosystem Coordination Board of the Puget Sound Partnership. 2/8/08.

O'Neill, S.M and **Collier, T.K.** 2008. Selecting provisional environmental indicators to determine progress in protecting and restoring the Puget Sound ecosystem. Invited presentation to the Leadership Council of the Puget Sound Partnership. 1/28/08.

Collier, T.K. D. Baldwin, C. Laetz, J. Incardona, J. Spromberg, and N. Scholz. 2007. Assessing the effects of pesticides, singly and in combination, on fish health and fish populations. Invited conference keynote presentation, "Modern Agriculture: Issues and the Way Forward". Kuala Lumpur, Malaysia. 12/5/07.

Collier, T.K., W.W. Dickhoff, and U. Varanasi. 2008. The seafood dilemma: A way forward. Invited presentation at OHH All P-I Annual Meeting, Muskegon, MI. 10/23/07.

Collier, T.K. and S.M. O'Neill. 2007. Protecting estuaries from toxic contaminants: What role is there for Biological Observing Systems (BiOS)? Invited presentation at 2007 State of the Estuary Conference, Oakland, CA, 10/18/07.

Collier, T.K. 2007. Considerations for use and/or removal of creosote-treated wood along the shoreline of Bainbridge Island, WA. Invited presentation at public town hall meeting by the City of Bainbridge Island. Bainbridge Island, WA. 10/15/07.

Collier, T.K., G.M. Ylitalo, and M.M. Krahn. 2007. Chemical contaminants and southern resident killer whales (*Orcinus orca*). Invited presentation at *12th Annual Symposium/Workshop on Cetacean Ecology and Conservation. Threats and Strategies: Reflections of the Year of the Dolphin*. Taipei, Taiwan. 10/9/07.

Collier, T.K., Assessing seafood safety in the aftermath of Hurricane Katrina. Invited presentation, AFS Special Symposium *Mitigating impacts of natural disasters on fisheries ecosytems*. 137th Annual Meeting, American Fisheries Society, San Francisco, CA. 9/5/07.

Collier, T.K. 2007. Restored urban streams in the Pacific Northwest as indicators of ecosystem health. Invited presentation, Fort Johnson Seminar Series. Charleston, SC. 8/31/07.

Collier, T.K. 2007. Putting the 'eco' into ecotoxicology. Invited presentation, Hollings Marine Laboratory Brown Bag Seminar Series. Charleston, SC. 8/30/07.

Collier, T.K. 2007. Urbanizing coastal streams in the Pacific Northwest as sentinel habitats. Invited presentation, Coastal Zone '07. Portland, OR. 7/26/07.

Collier, T.K. 2007. Session chair, Oceans and Human Health. Coastal Zone '07. Portland, OR. 7/23/07.

Collier, T.K. 2007. Pesticides and Pacific salmon: linking exposure to physiology, behavior, growth, survival, and long-term productivity. 14th Symposium on Pollutant Responses in Marine Organisms PRIMO-14. Florianopolis, Brazil. 5/9/07.

Collier, T.K. 2007. Session chair (with Sue Codi), Endocrine Disruption and Biological Modeling. 14th Symposium on Pollutant Responses in Marine Organisms PRIMO-14. Florianopolis, Brazil. 5/9/07.

Collier, T.K. 2007. National Core Coastal Indicators Workshop. Invited participant and breakout group (Water Quality) rapporteur. Linthicum Heights, MD. 5/1-3/07.

Collier, T.K., S.M. O'Neill, J.E. West, and N.L. Scholz. 2007. Toxic chemical contaminants and Puget Sound. 2007 Georgia Basin/Puget Sound Research Conference. Vancouver, BC. 3/29/07.

Collier, T.K. 2006. Establishing programs to ensure seafood safety and enhance human health. Invited keynote address, 2006 Qingdao Symposium on Marine Environmental Monitoring. 11/10/06.

Collier, TK., F.J. Loge, M.R. Arkoosh, L.L. Johnson, and T.R. Ginn. 2006. Increased disease-induced mortalities in outmigrant salmon due to environmental stressors. VIIth International Congress on the Biology of Fish, St. John's, Newfoundland. 7/22/06.

Collier, TK. 2006. Fish health, from mechanistic studies to population models, as a tool for assessing effects of chemical contaminants. Invited presentation, VIIth International Congress on the Biology of Fish, St. John's, Newfoundland. 7/20/06.

Collier, TK. 2006. Course Organizer. Methods and strategies for conducting environmental assessments in developing economies. AMETEC Training Course, Geoje Island, Korea. 6/12-6/22/06.

Collier, TK. 2006. Connecting ecosystems to human health in Puget Sound. Invited speaker, Puget Sound Toxics Forum, Seattle, WA. 4/5/06.

Collier, TK. 2006. Using modeling and empirical data to estimate population level effects from sub-lethal stress. Invited seminar, San Francisco Estuary Institute, Oakland, CA. 3/14/06.

Collier, TK. 2006. Using biology to assess the effectiveness of remediation and restoration activities. Invited presentation, Assoc. State and Territorial Solid Waste Management Officials. San Francisco, CA. 3/14/06.

Collier, TK. 2006. Assessing seafood safety after Hurricane Katrina: Is there a role for OHHI in environmental emergencies? Invited presentation, OHH Principal Investigators Meeting. Charleston, SC. 1/19/06.

Collier, TK. 2006. Sentinel species concepts in NOAA's WCCOHH Program. Invited presentation, OHH Principal Investigators Meeting. Charleston, SC. 1/19/06.

Collier, TK. 2005. Effects of Hurricane Katrina on fisheries in the northern Gulf of Mexico. Invited speaker and panelist for special session 'The Science of Environmental Catastrophes: 2005, the Year of the Hurricane'. SETAC National Conference, Baltimore MD 11/16/05.

Johnson LL, OP Olson, GY Ylitalo, C Rice, and **TK Collier.** 2005. Fish habitat use and chemical contaminant exposure at restoration sites in Commencement Bay, Washington. Interactive presentation by Tracy Collier. SETAC National Conference, Baltimore MD 11/15/05.

Collier, TK. 2005. Effects of land-based runoff on the health of anadromous and marine fish: From Seattle rain to hurricanes. Invited presentation. APEC Symposium, Protection of the Marine Environment from Land-based Sources of Pollution. Busan, Korea. 11/3/05.

Collier TK. 2005. Session chair. Contaminants, Oceans, and Human Health. Estuarine Research Federation Annual Meeting, Norfolk, VA. 10/18/05.

Incardona, **TK Collier**, and NL Scholz. 2005. Invited presentation. Estuarine Research Federation Annual Meeting, Norfolk, VA. 10/18/05.

Collier**, T.K.,** BF Anulacion, SY Sol, GM Ylitalo, and LL Johnson. 2005. PAH-induced impairment of fish health in Kitimat Arm, BC: Contaminant sources vs contaminant effects. Invited presentation. Estuarine Research Federation Annual Meeting, Norfolk, VA. 10/18/05.

Loge, F, MR Arkoosh, T Ginn, LL Johnson, **TK Collier**. 2005. Impact of environmental stressors on the dynamics of disease transmission. Invited presentation, presented by **TK Collier**. Estuarine Research Federation Annual Meeting, Norfolk, VA. 10/17/05.

Collier, T.K. (2005). Assessing the effects of storm runoff on fish and their ecosystems. Presented at Coastal Storms Project Final Wrap-up and Constituent Outreach workshops, in Jacksonville and Daytona Beach, FL 1/25 and 1/26/05.

Collier, T.K. (2004). Toxic chemicals affect both ocean ecosystems and human health. Invited departmental seminar. Xiamen University, Department of Chemistry. Xiamen, Fujian, China. 12/10/04.

Collier, T.K. (2004). Environmental monitoring for toxic chemicals in aquatic ecosystems. Short course taught at Xiamen University, Department of Chemistry. Xiamen, Fujian, China. 12/8-9/04.

Collier, T.K. (2004). Session chair. Water quality restoration in and around the built environment. 2nd National Conference on Coastal and Estuarine Habitat Restoration. Seattle, WA 9/14/04.

Collier, T.K. (2004). Northwest Fisheries Science Center research in Puget Sound. Seminar presented to NOAA's Chesapeake Bay Office, Annapolis, MD 8/24/04.

Collier, T.K. (2004). Impacts of oil and dispersants on NOAA trust resources. Presented at NOAA Dispersants Summit, Washington DC 7/27/04.

Collier, T.K. (2004). Current aquatic toxicology issues related to Superfund site management decisions. Presented to USEPA Region IX Biological Technical Assessment Group, Oakland, CA 7/21/04.

Collier, T.K. (2004). Workgroup co-chair. "Extension of molecular and computational information to risk assessment and regulatory decision-making". Pellston Workshop sponsored by SETAC/SOT "Emerging Molecular and Computational Approaches for Cross-Species Extrapolations". Portland, OR 7/18-7/23/04.

Collier, T.K. (2004). Modern approaches for linking contaminant exposure to meaningful biological endpoints. Presented at APEC workshop "Modern Approaches to Linking Exposure to Toxic Compounds and Biological Effects". Brisbane, Australia. 7/13/04.

Collier, T.K. (2004). Co-organizer, "Modern Approaches to Linking Exposure to Toxic Compounds and Biological Effects". Workshop sponsored by Asia-Pacific Economic Cooperation, Brisbane, Australia. 7/10-7/17/04.

Collier, T.K., J.P. Meador, and N.L. Scholz (2004). Toxicology issues associated with the use of treated wood in surface waters. Presented at a workshop entitled 'Use and permitting of treated wood in the San Francisco estuary', sponsored by the Bay Planning Coalition. Oakland, CA 6/3/04.

Collier, T.K. (2004). Overview of NWFSC research plans under NOAA's Oceans and Human Health Initiative. Presented to NOAA OHH workshop, Silver Spring, MD. 5/4/04.

Collier, T.K. (2004). ECD capabilities for environmental assessments and seafood safety research. Presented to Tsleil-Waututh First Nation and EcoTrust. Seattle, WA. 4/27/04.

Collier, T.K. (2004). Effects of oil on fisheries: Lessons from the EXXON Valdez and beyond. Symposium Plenary Address, 11th Symposium, Sample Handling for Environmental and Biological Analysis. Baiona, Spain, April 19, 2004.

Collier, T.K. (2004). Session chair, Special Session on the Prestige Oil Spill. 11th Symposium, Sample Handling for Environmental and Biological Analysis. Baiona, Spain, April 19, 2004.

Collier, T.K. (2004). Overview of ECD research pertinent to natural resource injury assessment in Portland Harbor. Presented to Portland Harbor Trustees. April 8, 2004.

Collier, T.K. (2004). Toxic chemicals and endangered species. Invited departmental seminar at Duke University. Durham, NC, 2/27/04.

Collier, T.K., L.L. Johnson, M.R. Arkoosh, N.L. Scholz, and J.P. Meador (2004). Toxic contaminants in PNW estuaries, and effects on Pacific salmon. Invited presentation, Annual Meeting of Oregon Chapter, AFS. Sunriver, OR, 2/18/04.

Collier, T.K. (2004). Endocrine disruption and developmental abnormalities: 'New' toxicology threats for estuarine fish. Invited presentation at CALFED Workshop 'Contaminant Stressors in the Bay-Delta Watershed'. Sacramento, CA. 2/4/04

Collier, T.K. J Labenia, MS Myers, JA Spromberg, and NL Scholz (2004). Prespawn mortality

in adult coho salmon: Interaction between urban environments and weather. Invited presentation at 2004 national meeting of the American Meteorological Society, Symposium on 'The Nexus Between Urban and Coastal Environments'. Seattle, WA. 01/14/04.

Collier, T.K. (2003). Session organizer and chair (with S. Marshall Adams). Making Ecotoxicology Studies Relevant: The 'So What' Question. SETAC North America conference, Austin, TX. 11/12/03.

Collier, T.K. J.S. Labenia, J.A. Spromberg, N.L. Scholz (2003). Prespawn mortality in coho salmon of hatchery origin: So what? Invited presentation at SETAC North America conference, Austin, TX. 11/11/03.

Collier, T.K. (2003). Session organizer and chair (with Jocelyne Hellou). Predicting Field Effects of PAHs from Laboratory Studies. SETAC North America conference, Austin, TX. 11/11/03.

Collier, T.K. (2003). Salmon Toxicology 101. Invited presentation at NW Salmon Recovery Conference. Seattle, WA 11/05/03.

Collier, T.K. (2003). Possible management actions for dealing with contaminant loads in southern resident killer whales. Presented at 'Disease, Pollution and Contaminants: A Workshop to Discuss Management Measures for Puget Sound Killer Whales'. Seattle, WA 10/24/03

Collier, T.K. (2003). Endocrine disruption and developmental abnormalities: 'New' toxicology threats for estuarine fish. Invited plenary presentation at 6th Biennial State of the Estuary Conference, Oakland, CA 10/21/03.

Collier, T.K. (2003). Do toxic chemicals limit the productivity of aquatic ecosystems? Invited departmental lecture at University of Stockholm, Stockholm, Sweden. 10/1/03.

Collier, T.K. and G.M. Ylitalo. (2003). Chemical contaminants in cultured fish: Risks, perceptions, and precautions. Invited lecture at Norges Landbrukshøgskole, Ås, Norway. 9/29/03.

Collier, T.K. and G.M. Ylitalo. (2003). Invited short course on seafood as a source of contaminant exposure for Native Americans. Presented at Water Quality Training course sponsored by Northwest Environmental Training Center, Randle, WA, 9/17/03.

Collier, T.K. and M.R. Arkoosh (2003). Contaminant effects on immune function of endangered fish. Invited platform presentation, 133rd American Fisheries Society Annual Meeting. Quebec City, Quebec, Canada. 8/13/03.

Collier, T.K. (2003). Ecotoxicology at the Northwest Fisheries Science Center. AMS-NOAA Teacher Workshop, NWFSC, Seattle, WA 6/26/03.

Collier, T.K. (2003). Ecotoxicology at the Northwest Fisheries Science Center. Kenmore Senior tour group. NWFSC, Seattle, WA 6/24/03.

Collier, T.K. (2003). Presentation to student orientation session for NOAA's Educational Partnership Program. Seattle, WA. 6/10/03

Collier, T.K. J Labenia, MS Myers, GM Ylitalo, and NL Scholz (2003). Extreme PRIMO—Premature mortality of adult coho salmon associated with stormwater discharges. Platform presentation, PRIMO 12. Safety Harbor, FL. 5/9/03.

Collier, T.K. (2003). Determining tissue and sediment effects thresholds for protecting aquatic species from harmful effects of toxic contaminants (PAHs, TBT, PCBs). Invited lecture, PRIMO 12 Short Course on Aquatic Toxicology. Safety Harbor, FL. 5/9/03.

Collier, T.K. (2003). Do toxic chemicals limit the productivity of our aquatic ecosystems? Invited departmental seminar. Alfred Wegener Institute for Polar and Sea Research. Bremerhaven, Germany. 4/25/03.

Ylitalo, G.M., J. Buzitis, K. Lynch, L. Reed, S.M. O'Neill, J.E. West, M.M. Krahn, and **T.K. Collier**. (2003). Recent exposure of adult coho salmon from Puget Sound to polycyclic aromatic hydrocarbons. Puget Sound/Georgia Basin Research Conference.

Collier, T.K. (2003). Lessons from the T/V Prestige oil spill in Galicia, Spain. Invited lecture for 'Science of Oil Spills' course, HAZMAT/ORR. Seattle, WA 3/20/03.

Collier, T.K. (2003). Toxic contaminants and environmental fish health. Invited presentation to the Columbia River Inter-Tribal Fisheries Commission Water Quality Workshop. Warm Springs Indian Reservation. 3/14/03.

Collier, T.K. (2003). Pre-spawn mortality in Pacific salmon on the northwest coast of North America. Invited departmental seminar, Oregon State University, Corvallis, OR 3/5/03.

Collier, T.K. (2003). A traditional Thanksgiving on the Spanish coast—response to the Prestige oil spill. ECD seminar series, Seattle, WA. 1/26/03.

Collier, T.K. (2002). Member of US Delegation to assist in response to T/V Prestige oil spill. La Coruña, Spain. 11/26/02-12/06/02.

Collier, T.K. (2002). Salmon, pesticides, and other toxic compounds. Invited presentation to the Pacific Fisheries Legislative Task Force. Catalina Island, CA. 11/23/02.

Collier, T.K. and L.L. Johnson (2002). Assessing effects of PAH contamination in a marine system and communicating risk across cultures. Presented at SETAC, Salt Lake City UT. 11/17/02.

Collier, T.K. (2002). Conference organizer. Workshop on Modern Approach to Linking Exposure to Toxic Compounds and Biological Health, Ansan, Korea. 9/02.

Collier, T.K. (2002). Linking exposure to toxic compounds to impaired biological health of Pacific salmon. Invited presentation. Ansan, Korea. 9/02.

Collier, T.K. (2002). Determining causality between contaminant exposure and biological responses in field studies in the marine environment. Invited presentation. Ansan, Korea. 9/02.

Collier, T.K. (2002). Conference organizer. Workshop on PCBs in the Puget Sound/Georgia Basin ecosytem. Sponsored by PNWSETAC, NOAA, and USEPA. Friday Harbor, WA 9/02.

Collier, T.K. (2002). Chemical habitat quality and salmon health. Invited speaker. Independent Multidisciplinary Science Team. Corvallis, OR. 5/20/02.

Collier, T.K. (2002). Chemical contaminants and salmon health: Issues for the Willamette River. Invited speaker. Pacific Northwest SETAC annual meeting. Portland, OR. 5/18/02.

Collier, T.K. (2002). Effects of contaminated sediments on marine and estuarine animals. Invited speaker. Pacific Estuarine Research Society. Portland, OR 5/3/02.

Collier, T.K. (2002). Polycyclic aromatic hydrocarbons in the aquatic environment: Still an emerging issue after 25 years? Invited departmental seminar, UC Riverside, Riverside, CA. 4/24/02.

Collier, T.K. (2002). Overview of Ecotoxicology and Environmental Fish Health Program. Presented at NWFSC/CIMRS collaboration planning meeting. Newport, OR. 4/3/02.

Collier, T.K. (2002). Chemical habitat quality and salmon health. Invited speaker. Salmonid Restoration Federation Annual Meeting. Ukiah, CA. 2/28/02.

Collier, T.K. (2002). Panel reviewer. CALFED Ecosystem Restoration Program. Davis CA. 2/25-2/27/02.

Collier, T.K. (2000). Review of NOAA/NMFS white papers on sediment and tissue thresholds for chemical contaminants. Invited speaker at PNWSETAC Annual meeting. Lacey, WA. 5/11/00.

Collier, T.K. (2000). After listing, then what? The salmon recovery process. Presentation at ECD Science Forum, 5/24/00.

Collier, T.K. (1999). The science of contaminated sediments. Invited presentation at Northwest Regional Conference on Contaminated Sediments. Portland, OR. June 24, 1999.

Collier, T.K. (1998). Hosted NMFS National Habitat Managers meeting, Seattle, WA. 10/5-10/8/98.

(presentations incomplete between 1997 and 2002)

Collier, T.K. (1997). Conference organizer. 3rd Annual Conference on Fisheries, Habitat, and Pollution. Charleston, SC. 11/6-11/8/97.

Collier, T.K. (1997). Invited participant and rapporteur, NATO Advanced Regional Study Workshop. Biomarkers: A Pragmatic Basis for Remediation of Severe Pollution in Eastern Europe. Cieszyn, Poland. September 20-25, 1997.

Collier, T.K. (1997). Session chair, Xenobiotic transformation enzymes. Ninth Int'l Symposium on Pollutant Responses in Marine Organisms, Bergen, Norway, 4/28/97.

Myers, M.S., B. French, W.L. Reichert, M.L. Willis, B.F. Anulacion, **T.K. Collier**, and J.E. Stein. (1997). Reductions in CYP1A expression and hydrophobic DNA adducts concentration in liver neoplasms in English sole: Further evidence supporting the "resistant hepatocyte" model of hepatocarcinogenesis in this species. Presented at Ninth Int'l Symposium on Pollutant Responses in Marine Organisms, Bergen, Norway, 4/28/97.

Anulacion, B.F., M.S. Myers, M.L. Willis, and **T.K. Collier.** (1997). Quantitation of CYP1A expression in two flatfish species showing different prevalences of contaminant-induced hepatic disease. Presented at Ninth Int'l Symposium on Pollutant Responses in Marine Organisms, Bergen, Norway, 4/28/97.

Collier, T.K., L.L. Johnson, C.M. Stehr, M.S. Myers, and J.E. Stein. A comprehensive assessment of the impacts of contaminants on fish from an urban waterway. Presented at 9th Int'l Symposium on Pollutant Responses in Marine Organisms, Bergen, Norway, 4/30/97.

Collier, T.K. Injury endpoints in natural resource damage assessment: A case study. Invited paper and panel discussion at ASTM-sponsored Seventh Symposium on Environmental Toxicology and Risk Assessment: Biomarkers and Risk Assessment. St. Louis, MO. 4/9/97.

Anulacion, B.F., B.D. Bill and **T.K. Collier** (1996). Stability of cytochrome P4501A-associated enzyme activity in frozen tissue specimens from teleosts. Presented at 17th Annual Meeting SETAC, 11/96, Washington, DC.

Collier, T.K., B.F. Anulacion, B.D. Bill, and A. Goksøyr (1996). Hepatic CYP1A in winter flounder along the Eastern seaboard: Results from seven years of monitoring. Presented at 17th Annual Meeting SETAC, 11/96, Washington, DC.

Collier, T.K., L.L. Johnson, M.S. Myers, and J.E. Stein (1996). The ecotoxicological consequences of increasing urbanization of coastal areas. Presented at Int'l Symposium on Environ. Chem. and Toxicol., 7/15/96. Sydney, Australia.

Collier, T.K. (1996). The ecotoxicological consequences of increasing urbanization of coastal areas. Invited Faculty Seminar, presented at the Royal Melbourne Institute of Technology. 7/11/96, Melbourne, Australia.

Collier, T.K. (1996). Invited Departmental Seminar, presented at the Royal Melbourne Institute of Technology. 7/12/96, Melbourne, Australia.

Anulacion, B.F., T. Hom, W.L. Reichert, and **T.K. Collier**. (1995). Temporal changes in CYP1A, DNA adducts, and contaminant residues in fish removed from contaminated sites. Presented at 2nd SETAC World Congress, Vancouver, B.C. November 9, 1995.

Collier, T.K. (1996). Injuries to natural resources in the Duwamish River. Presentation to Elliott Bay Natural Resource Trustees and the Boeing Company. Seattle, WA, 11/6/96.

Collier, T.K. (1996). **Session chair**. Processes of contaminant cycling. Fifth Annual Meeting of the North Pacific Marine Science Organization (PICES). Nanaimo, B.C. October, 1996.

Collier, T.K. (1995). National review panel member for five-year review of Puget Sound Ambient Monitoring Program. Seattle, WA. 9/26-9/28/95.

Collier, T.K. (1995). Biological indicators of oil exposure and effects in vertebrate species. Presentation to State of Alaska personnel responsible for Natural Resource Injury and Damage Assessment. Presented at request of NOAA's Damage Assessment Center. Anchorage, AK. 9/27/95.

Collier, T.K. (1995). NOAA's restoration research program, problems, and goals: Providing a scientific framework for restoration. Presented at the First Annual Northwest Natural Resource Damages Conference. Bellevue, WA. 9/21/95.

Collier, T.K. (1995). Biological indicators of contaminant exposure and effects. Presentation to Oregon and Washington personnel responsible for Natural Resource Injury and Damage Assessment. Presented at request of NOAA's Damage Assessment Center. Seattle, WA. 9/18/95.

Collier, T.K. (1995). The science of habitat restoration. Lecture presented at Seabeck Annual Adult Education/Family Retreat seminar series. Seabeck, WA. 7/27/95.

Collier, T.K. (1995). Salmon issues in the Pacific Northwest. Lecture presented at Seabeck Annual Adult Education/Family Retreat seminar series. Seabeck, WA. 7/26/95.

Collier, T.K. (1995). Marine mammal issues facing the National Marine Fisheries Service. Lecture presented at Seabeck Annual Adult Education/Family Retreat seminar series. Seabeck, WA. 7/25/95.

Collier, T.K. (1995). The EXXON Valdez oil spill: an example of NWFSC science in action. Lecture presented at Seabeck Annual Adult Education/Family Retreat seminar series. Seabeck, WA. 7/24/95.

Collier, T.K. (1995). Assessing the efficacy of remediation of contaminated estuarine sediments. **Keynote address** at joint meeting of Pacific Estuarine Research Society and Pacific Northwest Society of Environmental Toxicology and Chemistry. Seattle, WA. 5/12/95.

Collier, T.K. (1995). Presentation to Hylebos Waterway/Commencement Bay Trustees on preliminary results of fish injury assessments and proposal for continued work. Tacoma, WA. 4/11/95.

Collier, T.K., LL Johnson, MS Myers, E Casillas, JE Stein, and U Varanasi (1995). Incorporation of biomarkers into ecological risk assessments of contaminated nearshore marine habitats. Invited paper at ASTM-sponsored Fifth Symposium on Environmental Toxicology and Risk Assessment: Biomarkers and Risk Assessment. Denver, CO. 4/5/95.

Collier, T.K., LL Johnson, MS Myers, E Casillas, JE Stein, and U Varanasi (1995). Incorporation of biomarkers into ecological risk assessments of contaminated nearshore marine habitats. Presented at PRIMO-8, Monterey, CA. 4/3/95.

Collier, T.K., (1995). **Session Chair**. Xenobiotic biotransformation enzymes: Molecular Biological Aspects. PRIMO-8, Monterey, CA. 4/3/95.

Gallagher, E.P., P.L. Stapleton, **T.K. Collier**, U. Varanasi, and D.L. Eaton. (1995). Biochemical and molecular characterization of glutathione S-transferase expression in English sole and starry flounder. PRIMO-8, Monterey, CA. 4/3/95.

Collier, T.K.. 1995. Incorporation of biomarkers into ecological risk assessments of contaminated nearshore marine habitats. Presented at 1995 Marine Ecosystem Monitoring Network Meeting, Nanaimo, B.C. 3/29/95.

Collier, T.K. (1995). Presentation to Washington State Dept. of Ecology staff on use of biomarkers and screening methods for performing environmental assessment. Olympia, WA, 3/9/95.

Collier, T.K. (1995). Participant in South Florida Coastal Ocean Ecosystem Workshop. Miami, FL. 2/27-2/28/95.

Collier, T.K. (1995). Use of flatfish for monitoring Alyeska ballast water treatment plant in Valdez, AK. Invited participant at meeting between Prince William Sound RCAC, Alyeska, and USEPA. Seattle, WA, 1/18/95.

Anulacion, B.F., M.S. Myers, M.L. Willis, and **T.K. Collier**. (1994). Quantitation of immunohistochemically localized CYP1A in two flatfish species showing different prevalences of contaminant-induced neoplasia. Presented at PANWAT Annual Meeting, Newport, OR, 10/8/94.

Anulacion, B.F., M.S. Myers, M.L. Willis, and **T.K. Collier**. (1994). CYP1A expression in two flatfish species showing different prevalences of contaminant-induced hepatic disease. Presented at Int'l Symposium on Aquatic Animal Health, Seattle, WA. 9/7/94.

Myers, M.S., L. L. Johnson, O.P. Olson, C. M. Stehr, M.L. Willis, **T.K. Collier**, B. B. McCain, S.-L. Chan, and U. Varanasi. (1994). Relationships between toxicopathic hepatic lesions and exposure to chemical contaminants in marine Bottomfish species from the Northeast and Pacific Coasts of the United States. Presented at Int'l Symposium on Aquatic Animal Health, Seattle, WA. 9/6/94.

Collier, T.K. (1994). Remediation of contaminated subtidal sediments: How do we proceed? Invited keynote address at 3rd International Marine Biotechnology Conference. Tromsø, Norway. 8/11/94.

Collier, T.K. and L.J. Field. (1994). Eagle Harbor: A case study for remediation of a subtidal Superfund site. Presented at 6th Annual Superfund Environmental Evaluation Workshop. Boston, MA 4/28/94.

Collier, T.K., J.E. Stein, A. Goksøyr, M.S. Myers, J.W. Gooch, R.J. Huggett, and U. Varanasi. (1994). Biomarkers of PAH exposure and effects in fish from the Elizabeth River, Virginia. Presented at 6th Annual Superfund Environmental Evaluation Workshop. Boston, MA 4/27/94.

Collier, T.K. (1994). Participant at NOAA Science Review for Coastal Ecosystem Health. Miami, FL. 4/19-4/22/94.

Reichert, W. L., M. S. Myers, B. French, **T. K. Collier**, J. E. Stein, and U. Varanasi. (1994). Molecular epizootiology: Relationships among hepatic DNA adducts, cytochrome P4501A levels and lesions involved in hepatic neoplasia in marine fish. Presented at 85th Annual Meeting of the American Association for Cancer Research in vol 35 of the AACR Proceedings pg 102 , Abstract #607

Collier, T.K. (1994). Invited participant at Implementation Management Structure workshop for designing ecosystem approaches to restoration following the EXXON Valdez oil spill. Sponsored by Exxon Valdez Oil Spill Trustee Council, Anchorage, AK. 4/13-4/15/94.

Varanasi, U., **T.K. Collier**, M.M. Krahn, and S.-L. Chan. (1994). Incorporation of screening technologies into early response and sampling strategies: Lessons from the *EXXON Valdez* oil spill. Presented at Prevention, Response, and Oversight 5 years after the *Exxon Valdez* Oil Spill. Anchorage, AK. 3/25/94. Presented by T.K. Collier.

Collier, T.K. (1994). Invited participant at Implementation Management Structure workshop for designing ecosystem approaches to restoration following the EXXON Valdez oil spill. Sponsored by Exxon Valdez Oil Spill Trustee Council, Anchorage, AK. 3/21-3/23/94.

Collier, T.K. (1994). NRDA/Restoration studies being conducted and planned by Environmental Conservation Division. Presented at EC Seminar Series, Seattle, WA. 2/16/94.

Collier, T.K. (1994). Overview of Environmental Conservation Division research directed towards NRDA studies of Commencement Bay/Hylebos Waterway. Presented to the Commencement Bay Natural Resource Trustees, Seattle, WA. 2/8/94.

Collier, T.K. (1993). Invited participant, Prince William Sound Ecosystem Study Planning Workshop. Cordova, AK. 12/4-12/6/93.

Collier, T.K., B. F. Anulacion, J. E. Stein, A. Goksøyr, and U. Varanasi. (1993). A field evaluation of cytochrome P4501A as a biomarker of contaminant exposure in three species of flatfish. Presented at 14th Annual Meeting SETAC, 11/18/93, Houston, TX.

Collier, T.K. (1993). **Panel Chair**. Technical advisory panel to review guidance documentation developed by Univ. Wyoming/WEST, Inc. for Natural Resource Injury/Damage Assessment under OPA90. Laramie, WY, 11/4/93-11/5/93, and Houston, TX, 11/15/93.

Varanasi, U., J.E. Stein, L.L. Johnson, **T.K. Collier**, M.S. Myers, and E. Casillas. (1993). Evaluation of biomarkers of contaminant exposure and effects in coastal ecosystems. Presented at ICES, Dublin, Ireland, September, 1993.

Johansen, L.K., W.T. Roubal, and **T.K. Collier**. (1993). Characterization of glutathione S-transferases (GST) in two benthic fish species with differing susceptibilities to chemical carcinogenesis. Presented at PNWSETAC Annual Meeting, Newport, OR, May 20, 1993.

Anulacion, B.F. and **T.K. Collier**. (1993). Cytochrome P4501A and associated enzyme activities in fish removed from polluted environments and from fish exposed and depurated in the laboratory. Presented at PNWSETAC Annual Meeting, Newport, OR, May 21, 1993.

Collier, T.K. (1993). Using biomarkers to determine contaminant exposure and associated biological effects in aquatic organisms. Lecture presented at the High Technology Center in Bergen, Norway. May 6, 1993.

Collier, T.K. (1993). ECD research and assessment of the EXXON Valdez oil spill. Lecture presented at the High Technology Center in Bergen, Laboratory of Marine Molecular Biology. Bergen, Norway. April 30, 1993.

Collier, T.K. (1993). The utilization of species differences for understanding contaminant-induced disease processes. Lecture presented at National Veterinary Institute, Oslo, Norway. April 23, 1993.

Collier, T.K, B.F. Anulacion, A. Goksøyr, C. Grunwald, I. Wirgin, and U. Varanasi. (1993). Relationships between induction of cytochrome P4501A and contaminant residues in feral teleosts. Paper presented at 7th International Symposium on Pollutant Responses in Marine Organisms, Göteborg, Sweden, April 21, 1993.

Collier, T.K., J.E. Stein, A. Goksøyr, M.S. Myers, J.W. Gooch, R.J. Huggett, and U. Varanasi. (1993). Biomarkers of PAH exposure and effects in oyster toadfish (*Opsanis tau*) from the Elizabeth River, Virginia. Paper presented at 7th International Symposium on Pollutant Responses in Marine Organisms, Göteborg, Sweden, April 21, 1993.

Myers, M.S., M.L. Willis, A.-M. Husøy, A. Goksøyr, and **T.K. Collier**. (1993). Immunochemical localization of cytochrome P4501A in contaminant-associated hepatic lesions of English sole (*Pleuronectes vetulus*). Paper presented at 7th International Symposium on Pollutant Responses in Marine Organisms, Göteborg, Sweden, April 21, 1993.

Collier, T.K. (1993). **Session Chair**, CYP1A Induction. 7th International Symposium on Pollutant Responses in Marine Organisms, Göteborg, Sweden, April 20, 1993

Egaas, E., J.G. Falls, W.C. Dauterman, **T.K. Collier**, and J.U. Skåre (1993). The comparative in vitro glutathione S-transferase activity in selected plants, fish, insects, and mammalian species measured with four different second substrates. Paper presented at 7th International Symposium on Pollutant Responses in Marine Organisms, Göteborg, Sweden, April 20, 1993.

Collier, T.K. (1993). Using biomarkers to determine contaminant exposure and associated biological effects in aquatic organisms. Lecture presented at the Norwegian Institute for Water Research, Oslo, Norway, April 19, 1993.

Collier, T.K., M.M. Krahn, C.A. Krone, L.L. Johnson, M.S. Myers, S.-L. Chan, and U. Varanasi. (1993). Oil exposure and effects in subtidal fish following the EXXON Valdez oil spill. Presented at the 1993 International Oil Spill conference, March 31, 1993, Tampa Bay, FL.

Collier, T.K., W.T. Roubal, L.K. Johansen, E.P. Gallagher, E. Egaas, and U. Varanasi (1993). Characterization of glutathione S-transferases (GST) in feral species with different susceptibilities to chemical carcinogenesis. Presented at Society of Toxicology 32nd Annual Meeting, March 16, 1993, New Orleans, LA.

Egaas, E., J.G. Falls, W.C. Dauterman, **T.K. Collier**, and J.U. Skaare (1993). The glutathione S-transferase-mediated metabolism of atrazine in selected plants, fish, insects and mammalian species. Presented at SETAC World Conference, March, 1993 in Lisbon, Portugal.

Collier, T.K. (1993). Participation in strategic planning workshop to develop predictive capabilities under NOAA's Comprehensive Coastal Initiative. Silver Spring, MD, 2/15/93-2/17/93.

Casillas, E., L.L. Johnson, D. Misitano, **T.K. Collier,** J.E. Stein, B.B. McCain, and U. Varanasi. (1993). Inducibility of spawning, reproductive success, and egg production of flatfish species from urban and nonurban areas of Puget Sound, Washington. *Mar. Environ. Res.* 35:227 (Abstract).

Collier, T.K. (1993). **Session Chair**, Subtidal Session, EXXON Valdez Oil Spill Symposium, February 3, 1993, Anchorage, AK.

Collier, T.K., M. M. Krahn, C. A. Krone, L. L. Johnson, M. S. Myers, S.-L. Chan, and U. Varanasi (1993). Survey of oil exposure and effects in subtidal fish following the *EXXON Valdez* oil spill (EVOS): 1989-1991. Presented at EXXON Valdez Oil Spill Symposium, February 4, 1993, Anchorage, AK.

Myers, M.S., M.L. Willis, A.-M. Husøy, A. Goksøyr, and **T.K. Collier**. (1992). Immunochemical localization of cytochrome P4501A in contaminant-associated hepatic lesions of English sole (*Pleuronectes vetulus*). In: *Proc 13h Annual Mtg SETAC* (Abstract #92). Presented 11/92 in Cincinnati, OH.

Collier, T.K. and B.F. Anulacion (1992). Bioindicators of contaminant exposure and effects in fish removed from polluted environments in Puget Sound. PANWAT Proceedings 9:15. Richland, WA 9/26/92.

Collier, T.K. (1992). Using species differences to understand cancer in fish. Presented at Ira C. Darling Marine Center, Walpole, ME. 7/20/92.

Collier, T.K., M.S. Myers, W.T. Roubal, L.K. Johansen, and U. Varanasi (1992). Differences in chemical carcinogenesis in two benthic fish species: possible influence of xenobiotic metabolizing enzymes. Presented at Gordon Research Conference on Drug Metabolism. Plymouth, New Hampshire. 7/13/92.

Roubal, W.T., L.K. Johansen, and **T.K. Collier**. (1992). Methods for isolation and partial characterization of glutathione S-transferases from English sole (*Parophrys vetulus*) and starry flounder (*Platichthys stellatus*) from Puget Sound, Washington. Presented at 1st Annual Meeting of the Pacific Northwest Society of Environmental Toxicology and Chemistry, Bellingham, WA 7/26-7/27/92.

Collier, T.K. (1992). Participation in EXXON Valdez Restoration Science workshop to plan and coordinate subtidal studies. Anchorage, AK. 1/23/92.

Eufemia, N.A., M.S. Kroen, D.E. Watson, **T.K. Collier,** J.E. Stein, and R.T. DiGiulio (1992). Cytochrome P450 induction and genotoxicity in feral fish collected from the Niagara River ecosystem. *Toxicologist* 12:407. Presented at Society of Toxicology 31st Annual Meeting, Seattle, WA.

Collier, T.K. (1991). Participation in Damage Assessment Regulations Team workshop to determine early sampling strategies to be employed after oil spills. Washington, DC. 12/17-12/18/91.

Collier, T.K. (1991). Results of ECD research on the effects of the EXXON Valdez oil spill in fish species, and proposed restoration efforts. Presentation to NRDA Management Team and Restoration Planning Work Group, Anchorage, AK. 12/5/91

Collier, T.K., M.S. Myers, and U. Varanasi. (1991). Differences in chemical carcinogenesis in two benthic fish species: Activation, detoxication, and lesion prevalences. In: *Proc 12th Annual Mtg SETAC* (Abstract #92). Presented 11/91 in Seattle, WA.