BEFORE THE STATE OF WASHINGTON
ENERGY FACILITY SITE EVALUATION COUNCIL

IN THE MATTER OF: APPLICATION NO. 2013-01 TESORO SAVAGE, LLC VANCOUVER ENERGY DISTRIBUTION TERMINAL	CASE NO. 15-001 EXHIBIT NO. _____ (-TRB) PREFILED DIRECT TESTIMONY OF: STANLEY RICE, PhD SPONSORS: CONFEDERATED TRIBES OF THE UMATILLA INDIAN RESERVATION, CONFEDERATED TRIBES AND BANDS OF THE YAKAMA NATION AND COLUMBIA RIVER INTER-TRIBAL FISH COMMISSION

REGARDING EFFECTS OF OIL SPILLS ON AQUATIC ORGANISMS

Q. PLEASE SUMMARIZE YOUR QUALIFICATIONS.

A. I was hired by NOAA Fisheries in 1971 to initiate an oil effects study program in Alaska, relative to Alaska Fisheries, and to comment for the Agency on the marine portion of the draft Environmental Impact statement for the TransAlaska Pipeline and terminal at Valdez. The decisions for these facilities became final about six months after I was hired.

For NOAA, I have worked on damage assessment following several spills*: Ixtoc, Exxon Valdez, Kuroshima, Selendang Ayu, and Deepwater Horizon*.

As a scientist at Auke Bay Lab for NOAA, I supervised and managed a team of chemists and biologists, initially on the toxicity of oil prior to Exxon Valdez, and then from 1989 to my retirement in 2014. I spent 25 years on post spill damage assessment of the Exxon Valdez.

In this regard, I led or participated in studies of oil persistence, embryo sensitivity, and long term effects. I have worked on many species- from invertebrates to fish to whales.

I have published more than 130 peer reviewed scientific papers, the most recent in 2015, and continue to participate in publications from continuing work, with some still in draft form. About 90% of those are on oil toxicity and effects. A copy of my resume is attached to this testimony.

In 2014-15, I consulted with the U.S. Department of Justice in its prosecution of British Petroleum for environmental damages resulting from their 2010 *Deepwater Horizon* blowout in in the Gulf of Mexico. I testified before Judge Barbier in the *Deepwater Horizon* case regarding natural resources damage assessment on behalf of the United States. During that proceeding I evaluated the risk of the spill to aquatic resources and provided a critique of the methods used by witnesses for British Petroleum in conducting its risk assessment.

Q. PLEASE DESCRIBE YOUR CONCERNS WITH THE DRAFT ENVIRONMENTAL IMPACT STATEMENT (DEIS).

A. My concerns fall into two broad categories.

- First, the extent of possible oil exposure is not well portrayed in the DEIS, primarily because of the reliance on weathering models to predict the spread of spilled oil rather than using available literature, such as the report on the 1984 *Mobiloil* spill at river mile 88 of the Columbia River, which has real observations that contrast sharply with the modeled characterizations in the DEIS.

- Second the sensitivity of aquatic species to oil contamination is not well portrayed because of the reliance on acute toxicity information. The DEIS does not suitably address available literature coming from recent oil spill studies.

An oil spill risk analysis needs to evaluate two halves of the risk equation that addresses exposure risk, and environmental sensitivity risk. Oil spills are classic "low-probability/high-consequence" events, requiring careful assessment of the risk of exposure, as well as the resulting consequences to species and habitats. The overall risk is confounded when either half of the equation is under-represented. In this case, neither half was portrayed well, primarily because DEIS did not utilize key pieces of literature in discussions of the exposure and sensitivity risks.

Risk evaluation also must address temporal considerations. The evaluations of both exposure and sensitivity risk in the DEIS did not portray the sense of long-term effect time scales adequately. Spilled oil can persist for years in some contaminated habitats, and affected species can suffer long lasting impacts. Available literature was not used to address the possible length of exposure or of persistent impacts from an oil spill.

Q. HOW WOULD YOU CHARACTERIZE THE EXPOSURE RISK ASSESSMENT FOR AQUATIC RESOURCES IN THE DEIS?

A. I believe the exposure risk assessment portrayed in the DEIS has several critical weaknesses.

- It under-represents the extent of toxic oil spreading, particularly down river, in the water column and at the river bottom.

- The DEIS does not use available, pertinent literature
- The time-scale of impacts is under-emphasized.

The spreading of the oil in the DEIS, based on a weathering model and suggests that only a few river miles may be affected. This portrayal starkly conflicts with the real life spill that occurred in 1984, when the *Mobiloil* vessel ran aground at river mile 88 on the Columbia River. Table 4-13 of the DEIS suggests that a small/medium vessel spill of 2200 barrels will only affect two river miles; or a large facility spill of 5000 barrels will only affect seven river miles. In contrast, the 1984 *Mobiloil* spill, of 3000 barrels of oil, had oil swept down stream, as fast as 50 miles in 24 hours. Oil reached the mouth of the Columbia River in 3 days, and was transported up the Washington coastal shoreline to Grays Harbor and Willapa Bay over the next few days.

River current is a key dominant factor, because it causes spilled oil to be carried down stream rapidly. Additionally, oil will mix into the water because of the turbulence from the current; measurements from the *Mobiloil* spill found oil on the river surface, in the water column, on river bottom sediments, and in the mouths of white sturgeon (Kennedy and Baca, 1985; Krahn et al. 1986). The current will also impact response efforts, because of the rapid spread oil downstream due to current, and because the effectiveness of booms to contain oil is curtailed at currents above one knot (Fang and Wong, 2006). Currents in the Columbia River will exceed one knot at many times even above Bonneville Dam. Rice Exhibit 1.

Clearly, more than two river miles of contamination can be expected to result from a spill of one rail car (750 barrels) to the Columbia River. In addition to the 1984 *Mobiloil* spill on the Columbia River, the expectation for rapid oil spreading and down stream contamination is

corroborated by a Bakken crude oil spill in the lower Mississippi where *Barge E2MS 303* released 750 barrels from a gash. In this case, oil was swept downstream as far as 65 miles over the next two days (Doeling et al. 2014 - Rice Exhibit 2). Less than 1 % of the spilled oil was recovered. The lack of use of the real life *Mobiloil* spill in the Columbia River and the *Barge E2MS 303* casts doubt on the validity of exposure risk in the DEIS and related model estimates.

Q. HOW WOULD YOU COMPARE THE INFORMATION FROM THE *MOBILOIL* SPILL, THE DRAFT ENVIRONMENTAL IMPACT STATEMENT AND IT'S APPENDICES?

A. The *Mobiloil* spill report used actual field data and observations, and included chemical measurements of the oil below the surface and on the river bottom sediments.

- Appendices E and J are based on output of a weathering model [ADIOS2], used to predict possibilities for the extent of oiling. The output can vary with the input variables. It is not clear what specific variables were inputed into the model for Appendices E and J, but the output of the model contrasts extremely with the real life *Mobiloil* spill, suggesting that river current may have been under estimated or devalued.
- The draft environmental impact statement selectively emphasizes the extent of oil spill distribution to a few limited river miles, primarily reporting only oil present at a thickness of ½ mm. *Mobiloil* spill report documented oil traveling down stream on the surface, rapidly; contamination was documented on river banks and Washington

ocean shorelines, and oil was documented in the water column, on river bottom sediments, as well as in mouths and tissues of white sturgeon.

The net result of the choices in the DEIS is that it under-reports the down stream spread of oil via the current.

Q. WHY IS UNDER-REPORTING OF EXPOSURE A CRITICAL ISSUE?

A.

- Under predicting the spread of the oil under predicts the habitats and species that can be expected to be exposed in a spill event, and would then under-predict the response needed to cope with a spill event.
- Under-predicting the spread of oil, along with reliance on acute toxicity thresholds for effects will under predict the adverse effects of oil exposure to species and habitats, particularly early life stages that can be impacted in the low parts per billion polycyclic aromatic hydrocarbons (PAH). (this will be discussed in more detail later under sensitivity issues).
- Limiting the discussion of effects to oil distributions ½ mm thick is inappropriate as oil of this thickness primarily relates only to birds, marine mammals, and shoreline habitats. Further, it only addresses acute toxicity, a flawed concept when evaluating biological risks to sensitive species (discussed subsequently in greater detail).

Q. HOW DOES APPENDIX J TREAT EXPOSURE RISK TO AQUATIC RESOURCES?

A. Poorly. As mentioned previously, there are two parts of the risk assessment: the chances of exposure, and the chances of effect from that exposure. Appendix J attempts to quantify the chances of exposure, based on vessel and transfer operations, and concludes the risk of an

individual event is very low, but nevertheless concludes that vessel related spills will almost certainly occur during the life of the project.

The low risk of exposure is then inappropriately minimized by the modeling of the oil spreading, and river miles that might be impacted. The percent of oil remaining after 120 hours (35% for Bakken crude, Table 21 and 75% for Dilbit, Table 22; 0.1 mm thickness of oil from Appendix J) is based on a model of evaporation and spreading, to arrive at an estimate of 0.31 RM or 0.66 RM impacted by 1000 barrel spill of Bakken crude or Dilbit, respectfully. Real life experience with the 3000 barrel *Mobiloil* spill contrast sharply. This spill contaminated many miles of river shoreline between river mile 88 and the mouth of the Columbia River in three days, because of the river current. River current is the dominant factor in determining the river miles impacted. The under-estimate of 0.31 and 0.66 river miles impacted is misleading.

The estimate of 35% oil remaining after 120 hours is also misleading. First, some of the oil will mix into the water column because of the current, and not be available to evaporation, but nevertheless will be available to organisms in the water column. Disappearance from the surface does not translate to disappearance from the river ecosystem (e.g. oil in the mouths of white sturgeon from the *Mobiloil* spill report). Second, the 120 hours used in the table is sufficient time for the oil to be transported as far down stream as the river mouth from the terminal facility, and is sufficient time to exert effects to embryos and other tiny organisms, such as zooplankton and invertebratesin the water column. Once habitat is contaminated, however, the persistence in shorelines, particularly wetlands, will be quite significant. Oil in contaminated wetlands from the *Florida* spill at West Falmouth has persisted more than

three decades (Reddy et al 2002), and oil from the Exxon Valdez spill continues to persist in contaminated beaches after 25 years.

The risk is communicated in terms of exposure risk (chances for a spill event; river miles that could be exposed), but the risks to sensitive life stages of aquatic organisms for example are not covered in Appendix J. The risk for a specific spill event is low, but this is a multi-decade project, and the risk of spill over the life of the project is significant. In 10 years, 1.8 million rail tank cars may be expected to travel to the port facility for transfer to vessels.

Q. IF RIVER CURRENT IS A DOMINANT FACTOR IN A POTENTIAL SPILL IN THE COLUMBIA RIVER, IS IT A GOOD THING OR BAD THING?

A. Both. The current is a good thing in that it will transport new clean water from upstream, on a steady basis, bringing continual dilution, and uncontaminated prey items. Exposures are likely to be transient for many species, thus minimizing the potential exposure to acute toxic doses of oil, particularly for the larger organisms.

It is bad in the sense that current will rapidly carry surface oil down-stream, and most importantly, aid in the mixing of oil down into the water column. The "mixing issue" is critical, because mixing oil into the water column means that the subsurface species are now vulnerable to exposure. The risk expands from primarily birds and marine mammals on the surface to fish, invertebrates, and vulnerable life stages in the water column. For the largest organisms, the transient nature of spills may be tolerated, but for the smallest and most fragile embryonic life stages, "transient" exposures can be more than adequate to absorb toxic PAH, and harm critical developmental processes (e.g. heart development, as shown later).

From a response viewpoint, the current is a very bad thing. The spill will expand downstream rapidly, and hinder responders from containing or limiting the geography of the spill. More wetlands, tidal areas, and even bottom sediments (because of the mixing) will be more vulnerable to contamination. Contamination of any of these habitats translates to greater difficulty in cleanup, and expands the time scale for chronic oil exposure to many organisms. Once oil is in the water column, cleanup operations will do little or nothing to protect exposed aquatic organisms.

Q. HOW WOULD YOU CHARACTERIZE THE SECOND HALF OF THE RISK EQUATION, SENSITIVITY OF SPECIES AND HABITATS TO OIL EXPOSURE?

A. There are several shortcomings in the DEIS relative to discussion of risk to species and habitats.

- Assessing risk to species and habitats is more complex than measuring the statistics of spill events
- Reliance on acute toxicity measures to gauge or predict sensitivity issues is a flawed concept
- Avoidance of recent spill study literature focusing on high sensitive embryonic life stages is inappropriate.
- Time scales relative to sensitivity are not treated meaningfully, because of reliance on acute toxicity as a measure of risk.

Measuring the risk to species is a complex task compared to measuring the risk of a spill event (such as derailment of a tank car) where there are hard statistical data to work with.

Biological sensitivities are a much more complex, and while we certainly have spill events in the past to examine, none have a complete understanding of the impacts from such a spill. The two most studied spills in world history, Exxon Valdez and Deepwater Horizon, have produced hundreds of scientific papers, yet were not studied satisfactorily: Pre-spill chemical and biological baselines are largely absent; there are no spill replicates; and only the *Exxon Valdez* spill has been tracked for a long time period, but only on limited species and habitats. The spill most relevant to the Columbia river DEIS, the 1984 *Mobiloil* spill is a good example of inadequate studies of biological consequences: the report (Kennedy and Baca, 1985) documents response effort, oil tracking, but biological effects were under studied in general (e.g. oiled bird carcasses were collected and disposed of, but the numbers and the species were not recorded). Some larger spills, such as *Exxon Valdez, Cosco Busan*, and *Deepwater Horizon* (Gulf of Mexico blowout) have been tracked with more scrutiny, and have revealed a wealth of information in the form of many scientific publications: oil persists in many habitats, embryos are extremely sensitive, some species have impacts that last for decades (see reviews by Peterson et al 2003; Rice 2009; Bodkin et al. 2014). The DEIS did not take advantage of this important literature. Instead it relies heavily on acute toxicity information, a flawed concept when predicting potential impacts from spilled oil.

Q. PLEASE EXPLAIN YOUR CONCERNS ABOUT EMPHASIZING ACUTE TOXICITY MEASURES WITHOUT COMPARABLE TREATMENT OF SUBLETHAL EFFECTS.

A.

- Acute toxicity, typically measured as an LD50, is a flawed concept for predicting biological effects from an oil spill. Standard 1-4 day acute toxicity tests will describe

a lethal toxic dose ("the ceiling"), but they do not describe what a safe exposure is. Standard acute toxicity tests are a scientific tool designed to determine the relative acute toxicity of different toxicants. The concept was originally applied to pesticides to see which formulation was most toxic and rapid. The method was not developed to assess the sensitivities of different organisms, or different life stages, or predict survival potential if exposures were not acutely toxic.

- Acute toxicity is often a relatively rapid death because the mechanism of toxic action is usually at respiratory (gills, lungs) and/or neural functions. When these critical functions are impaired, death follows rapidly, and is easily observed.

- There are many different mechanisms of toxic action, from impacts to DNA in a nucleus, to subcellular structures and function in a cell, or to tissues and organs. Some of these mechanisms are captured by the term sublethal effect, a term that is very inclusive, meaning that some damages may be slight and repairable with no measureable loss in survival potential, while other sublethal effects may not show as visible damages, but the survival potential can be severely impacted. In the latter case, death may be assured, either physiological, or more likely, as result of predation because the organism is impaired in ways not easily measured.

- The term sublethal is easily misinterpreted, because there is an implication that this level of dosage is not deadly. However, survival in nature is more complicated. Any sublethal effect from exposure to an organism that makes it more difficult to acquire energy (be a predator) or avoid predation (e.g. grows slower, swims slower) will diminish the survival potential. A population effect can occur if the net result of

sublethal effects is increased predation, decreases in recruitment, or impairment to reproduction. Acute toxicity tests determine the "ceiling" of negative effects, but never determine the "floor" of negative effects. For example, a crustacean larvae that is still alive after a 4 day exposure tests, but fails to molt weeks later is ecologically dead. The same holds true for an exposed fish larvae that can not swim as fast as their unexposed cohorts; they are eaten.

Q. HAVE YOU STUDIED SUBLETHAL EFFECTS? PLEASE PRESENT RELEVANT INFORMATION FROM YOUR STUDIES.

A. Yes, I have studied sublethal effects in many studies, most dating back to the *Exxon Valdez* spill, when I led a group of biologists and chemists studying the persistence of oil and the impacts for over 25 years in my career with the NOAA. Our studies along with several others found long-term persistence of oil, and long term impacts from the spill, on the order of decades. Oil is still present in many beaches of Prince William Sound; Elevated mortalities in pink salmon embryos were detected in the four year classes following the oil spill; sea otter population recovery was were impacted for nearly two decades in the hardest hit areas of the spill, and two pods of killer whales (one resident pod- fish predators, and one transient- marine mammal predators) have not recovered from the 40% losses they incurred in the first year of the spill.

These major findings were reviewed in a paper published in *Science* (Peterson et al. 2003), of which I am a co-author. We concluded, based on the suite of studies, that long term impacts were very significant, and that oil spills can no longer be viewed as short term

events. Most importantly, these findings were not predictable from the acute toxicity studies that dominated the literature prior to the Exxon Valdez spill.

The DEIS did not use this available literature that discusses the significance of sublethal effects or embryo sensitivity. Yet, the DEIS did cite Neff (page 4-77), stating that acute toxicity levels were not reached in the days following the *Exxon Valdez* spill. While that is a true statement, it is misleading. By omitting the literature on long-term effects, the DEIS presents a one-sided slant that would suggest that spills do not present a toxicity risk to fauna. That is not the case.

An illustrative example would be the DEIS's omission of substantial literature on Pink Salmon embryos sensitivity to polycyclic aromatic hydrocarbons (PAH) found in crude oil: Pink Salmon embryos were impacted by parts per billion of PAH, much lower concentrations than parts per million (3 orders of magnitude). The impacts at these low doses were for the most part subtle and difficult to see, in contrast to a dead animal in an exposure container; but the impacts were very important. An exposure of 18 ppb PAH during the embryonic stage decreased the adult returns by 40%, and an exposure of 5 ppb PAH reduced the returning adults by 20%. These studies were unique; they combined laboratory exposures and with releases of juveniles to the environment for 18 months; the tests were large and intense experiments, requiring about 70 thousand tagged fry from embryonic exposures per dose group. They were repeated in multiple years. These oil exposures were "sublethal", only normal looking fry were released (including controls), yet the impacts were significant; population level of effect was measured in the returning adults. (Heintz et al. 1999) These embryo studies, along with oil persistence, delayed sea otter recovery for two decades, and

lack of recovery in two pods of killer whales, changed our thinking of how to study spills, and influenced the studies years later of the *Cosco Busan* and *Deepwater Horizon* spills.

Comments on the DEIS by Vancouver Energy (page 4-64) acknowledge that sublethal impacts to fish have been reported in developing embryos and juveniles (Incardona et al. 2015), but do not give these results much significance. Vancouver Energy fails to mention the low exposure levels in the test or the probability that affected embryos will die. The significance of the sublethal effects in embryos is further under-represented by the discussion and reference to the work of Lee and Anderson (2005) suggesting that sublethal effect measurements like the presence of Cytochrome P450, while indicating exposure to PAH, do not necessarily indicate injury. The DEIS and Vancouver Energy's discussion of Cytochrome P450 is misleading. For embryos, where tissues and organs are in a complex continuum process of cell differentiation and development, indices of exposure may well predict damage, as indicated in the embryo exposure tests by Carls et al. (2005); Pink Salmon embryos with elevated P450 response later had decreased growth and poor survival compared to controls with baseline levels of P450 activity.

Q. EACH SPILL IS DIFFERENT, EACH OIL IS DIFFERENT- HOW RELEVANT IS THE LITERATURE FROM OTHER SPILLS TO THE COLUMBIA RIVER OIL TRANSPORT PROJECT?

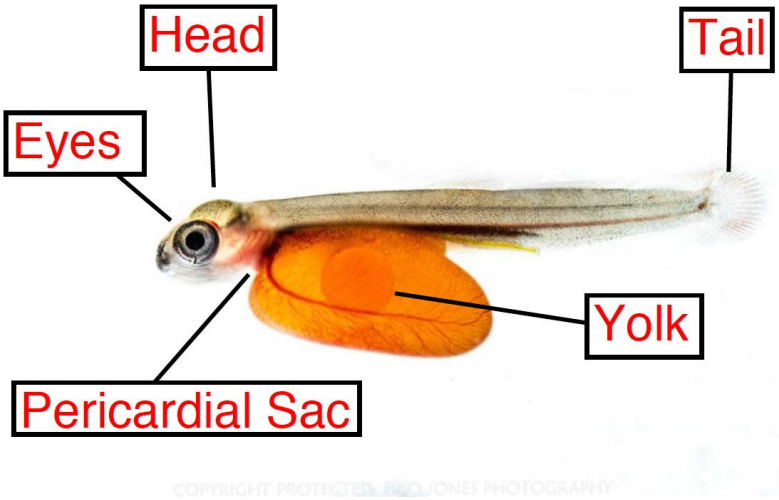
A. Each spill event is different in many ways, from volumes and types of oil spilled to differences in species and habitats that are impacted. *Exxon Valdez* spill in Alaska and the *Deepwater Horizon* spill in the Gulf of Mexico are examples of the diversity and differences between spills, yet these spills and others give us insight on how to deal with other spills,

how to cope with them, and what to expect. Although no two spills are exactly alike, hence they are not replicates, we have learned that when habitats are contaminated, oil can persist for decades; surface species such as birds and marine mammals are vulnerable to acute toxicity (inhalation and direct contact with oil; and heat loss issues from having feathers compromised), subsurface species are threatened by "sublethal" exposures, and embryos are 2-3 orders of magnitude more sensitive than older life stages. While the sensitivity of Tuna embryos from the Gulf of Mexico do not have direct applicability to the Columbia River risks, they are informative of the general sensitivities of the embryos from species specific to the Columbia River.

Q. PLEASE EXPLAIN THE EXTREME SENSITIVITY IN EMBRYOS, AND THE SIGNIFICANCE OF "SUBLETHAL" EXPOSURES TO DEVELOPING EMBRYOS.

A. The impacts of oil on developing embryos, at very low exposure concentrations, is perhaps the best example of the significance of sublethal effects. Let me explain this using the research of John Incardona and his collaborators that has been developed in the last few years since the *Deepwater Horizon* spill

The picture in Figure 1 shows a single coho embryo; note the head, tail, eyes, yolk, pericardial sac.



The images in Figure 2 show embryos of other species; each different to some degree, but they have the same features. Many fish embryos go through similar developmental stages. The sensitivities that we are able to measure may differ between the species, but they are likely to be more similar at the embryonic stage across species compared to their own sensitivities at juvenile and adult life stages within the same species.

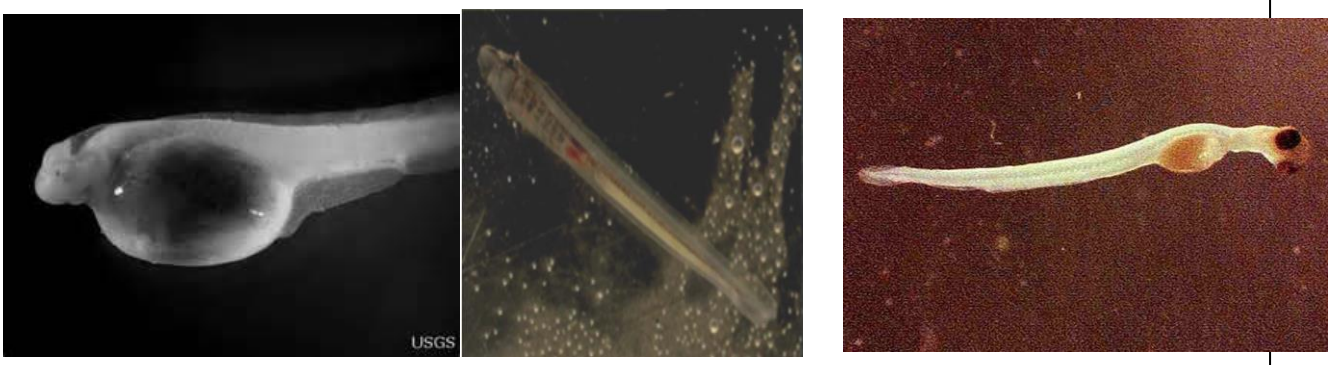


Figure 2. Left to right: Image of larval white sturgeon. Courtesy USGS. Image of a larval lamprey, courtesy of Ralph Lampman, Yakama Nation Fisheries. Image of larval Eulachon courtesy of Canadian Department of Fisheries and Oceans.

In Figure 3, two embryos (yellow fin tuna, from Incardona et al. 2014) are shown, with the one on the right being an exposed and damaged embryo. The damage does not appear catastrophic, note the edema around the pericardial sac of the heart. However, in the video accompanying Figure 3 http://www.critfc.org/blog/advocacy/effects-of-petroleum-spills-on-fish/ note the heart beat rate in the control, which has a good steady rhythm. Now, consider the heart beat of the exposed embryo. The heart rate of the exposed embryo is much slower. Because the heart is affected, there are many down-stream effects. The net result: this animal, if it survives to be a juvenile, is less fit. "Less fit" was demonstrated in the embryonic exposure study of Mahi-Mahi by Mager et al (2014): exposures of 48 hrs to embryos at parts per billion PAH resulted in juveniles that did not swim as well as their control cohorts a month after the exposures. Their chances of surviving the critical juvenile stage were diminished.

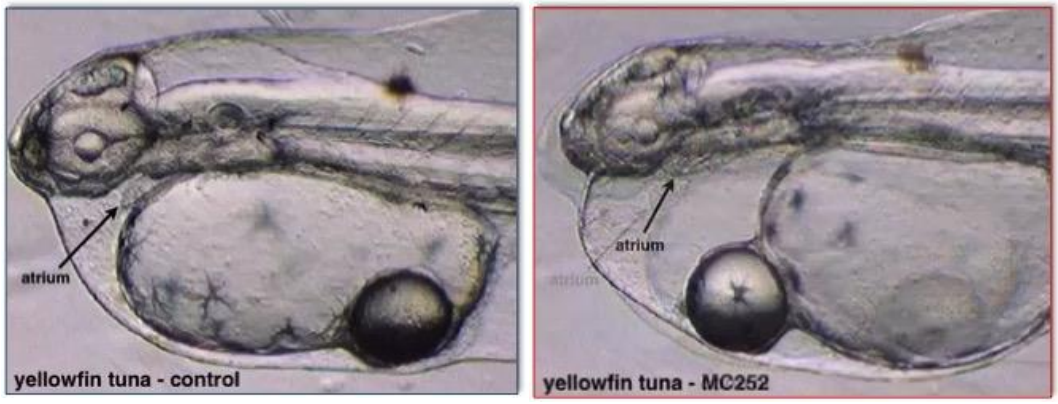


Figure 3. From Incardona et al. 2014. The embryo on the right was exposed to 3.4 PPB polycyclic aromatic hydrocarbons. Video available at http://www.critfc.org/blog/advocacy/effects-of-petroleum-spills-on-fish/

There are similar results for many other species, including pink salmon and herring. Heart damage, function, and decreased swimming performance were found in juveniles up to nine months post exposure of embryos to low parts per billion of PAH (Incardona et al. 2015).

These and other studies demonstrate the significance of a sublethal exposure during the embryo stage. Brief exposures can have long lasting results that require detailed studies and time to detect. Although the exposure doses will seldom reach acute toxicity levels and kill an embryo out right, the sublethal effects will eventually lead to increased predation (less fit), and poorer percentages of survival. Slow swimming performance will make it difficult to be a successful predator, and more difficult to avoid predation. This is an example of how sublethal effects are actually lethal to a population.

Q. PLEASE PROVIDE A SUMMARY OF YOUR CONCLUSIONS.

A. The DEIS paints an optimistic picture. It relies heavily on low odds of an event, but given the length of the project, and the numbers of trains, tank cars, and vessel trips, it is more likely than not there will be an incident. The *Exxon Valdez, Kuroshima, Selendange Ayu, Cosco Busan, Ixtoc, Deepwater Horizon* accidents all had low odds of an event, yet they did happen with severe impacts to the environment. Second, the time scale is important. Time scale of the project is decades long, giving time for a low probability event to happen. The time scale of oil persistence and effects can also be a long term event, as shown in some well-studied spills. Disappearance of oil from the surface signals the end of the response effort, but oil may persist for decades below the surface, particularly wetlands, and effects can last for decades also, particularly for long lived species like white sturgeon and green sturgeon. Oil spills are not a one week or one month event.

The DEIS under-represents effects because of its reliance on acute toxicity concepts and data, and ignores the growing literature on the extreme sensitivity of embryos and the significance of sublethal effects to subsurface organisms. Given the volumes of oil transported over the number of years of the project, there is cause for concern for sensitive life stages.

The DEIS with its reliance on low spill event risk, poor projections of down stream oil movement, reliance on acute toxicity mechanisms as the measure of effects, and the avoidance of important modern literature, paints an overly optimistic picture. Profit is for the oil and transport industry; risk is on the river, organisms living in that habitat, and those who use it.

END OF DIRECT TESTIMONY

I declare under penalty of perjury that the above testimony is true and correct to the best of my knowledge. Executed this 13th day of May, 2016.

Stanley D Rice

Stanley Rice, PhD

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