Stanley Rice 8 March 2026

My name is Stanley Rice.

I have been asked to speak about the toxicity of Polycyclic Aromatic Hydrocarbons, and how they may be an issue relative to chronic exposure from cruise ship discharges.

Qualifications: (1) Ph. D. in toxicology/comparative physiology, 1971 Kent State University; my dissertation dealt with toxicity issues and physiology of developing trout embryos. (2) 42 years of researching oil effects to marine life, including salmon and herring embryos and juveniles. (3) extensive research prior to and after the Exxon Valdez oil spill; authored/co-authored 150 plus peer reviewed scientific publications, most on oil effects, from salmon embryos to killer whales. (4) I also have direct research experience with Alyeska Terminal treated ballast water discharge in Port Valdez, 1979 Ixtoc blowout in the Gulf of Mexico, , Kuroshima bunker oil release outside of Dutch Harbor, & 2010 Gulf of Mexico blowout; and I testified as an expert witness for the U.S. Dept. of Justice in their prosecution of British Petroleum for the 2010 Gulf Blowout.

Message: Historically, the toxicity "paradigm" for oil spills evolved from a focus on acute toxicity in the aftermath of spills to an increased understanding and concern for the significance and impact of chronic sub-acute exposures on fitness and recruitment of embryos/larvae/juveniles to marine populations. Let me explain.

I was hired in 1971, to initiate an oil effects research program at NOAA's Auke Bay Laboratory, prior to approval of the TransAlaska Pipeline. Oil effects research requires understanding the composition of the exposure (what are the toxic components, and are they available in various solutions to organisms) and the biology of the organism (what are the weak links, like embryos). My course work in physiology and biochemistry set me up to communicate and lead a team of chemists and biologists. We started with current state of knowledge- conducting acute bioassays, and learning what compounds enter the water column under different conditions. Our exposure methods, analytical capabilities, and effects measurements evolved over time, becoming more sophisticated. We were in place when the 1989 Exxon Valdez spilled, becoming the largest spill in U.S. waters at the time. We had a team sampling PAH levels under the oil slick in the first week of the spill and a team evaluating juvenile pink salmon growth using wild and marked fry of known size and release date from hatcheries within a month.

Oil spill damage assessment changed dramatically with the record setting 1989 Exxon Valdez oil spill. Prior to 1989, oil spills were viewed as an "acute toxicity event", and once cleaned up, there was seldom any follow-up to look at long term damage or oil persistence. Damage was assessed primarily by retrieving dead animals such as birds.

Exxon Valdez was the most studied oil spill in world history at the time of the spill, over three decades, and changed the "paradigm" on how we view and assess long term oil persistence and damage. Expected acute mortalities to birds, sea otters, and intertidal organisms were well documented in the first year or two of the spill.

Several Un-expected long term damages were found, and followed for decades.

1. From "acute" exposures in 1989, 40% decrease in two killer whale populations in PWS; resident AB 1 pod (fish prey) and transient AT1 pod (marine mammal prey). AB 1 pod has recovered partially in over 30 years; AT 1 had no females that survived, and the surviving males continues to die as they age, and that pod will become extinct.
2. Pink salmon embryos in oiled streams had decreased survival for about 4 years compared to survival in non-oiled streams (detected by ADFG), apparently from chronic oil leaking out of nearby contaminated sediments. Oil in the stream banks was detected in follow up studies. Controlled laboratory exposure tests by our lab detected pink salmon (and herring) were about 100-1000 times more sensitive to PAH than juveniles and adults. Exposure of pink salmon embryos to about 5 ppb PAH during their development resulted in a 20% lower return of marked adults 1.3 years later. Similar sensitivity to parts per billion PAH was detected in fish embryos in the Gulf of Mexico spill, where heart rates were slowed in developing embryos to part per billion exposures in controlled laboratory tests, thus demonstrating a mechanism for slow growth, and delayed impacts on swimming speed.
3. Sea otters in specific home areas near Knight Island in PWS had poor recovery rates from the spill. Chronic exposure from digging in shallow areas for clams that were in oil contaminated. Recruitment of juveniles to population was lower than other areas of PWS, an effect lasted for about two decades.
4. Oil in intertidal sediments was assessed about 10 years post spill, in response to the poor recovery of sea otters, and a surprising amount of oil persisted in heavily oiled beaches, which had been cleaned vigorously in 1989 and 1990. Oil was primarily in the lower intertidal zones, about 6 inches below the sediment surface, where sea otters, particularly females, forage for clams. The persistent oil was only slightly weathered and still very toxic, and easily transferred to the baby otters when the females returned with contaminated paws and fur.

Bottom line: long term damages and long term oil persistence can affect fitness, and be a limiting factor in recruitment to marine populations. Environment and populations will recover, over time, if they are not repeatedly re-contaminated. However, although the ecosystem will recover, it may not be the same; for example, PWS herring population.

Concern over cruise ship discharges is not likely to be from an "acute" toxicity event. Dead animals will not be likely seen. Discharges containing PAH that are repeated frequently, and in volume by several large vessels could create a chronic exposure situation, possibly resulting in effects to fitness of embryos or larvae that are struggling to acquire energy while avoiding predators. There will be an overlap of timing between cruise ships with developing embryos and larvae of herring, emerging salmon fry and juveniles, crabs/shrimp larvae that go through a series of molts in spring/summer, and also with the embryo/larvae that are prey that come from other species.

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